Yong-Jian Geng, J Clin Exp Cardiolog 2016, 7:6(Suppl)
http://dx.doi.org/10.4172/2155-9880.C1.028

Annual Conference on

Atherosclerosis and Clinical Cardiology

July 11-12, 2016 Philadelphia, USA

Yong-Jian Geng

University of Texas McGovern School of Medicine, USA

Vascular cell apoptosis and calcification in arteries with atherosclerotic hypertension

Cellular accumulation, lipid deposition and connective tissue formation in the arterial intima characterizes the development of atherosclerotic plaques. However, the increased cell mass in the intima does not reflect the rates of cell growth and death. Over the long-lasting process of atherogenesis often for several decades, many plaque cells may undergo apoptosis, a form of programmed cell death. Atherosclerosis and hypertension are closely associated chronic arterial diseases. In many case, they co-exist and interplay, triggering the development of life-threatening complications, such as myocardial and cerebral infarctions. Increased apoptosis and phenotypic alteration of vascular cells occur in the arterial wall with various pathological conditions, including atherosclerosis and hypertension. Several molecular pathways contribute to the changes of cellular components in atherosclerotic lesions. Maintaining normal structure and shape of the blood vessels requires a balance between apoptosis and proliferation. During the pathogenesis of atherosclerosis and hypertension, vascular cells may become malfunctional or injured by various potentially harmful factors. Vascular smooth muscle cells exposed to pro-atherogenic and hypertensive factors are undergoing phenotypic changes, which transform the cells from contractile to synthetic styles. Two prominent pathological alterations are reduction of vascular cell survival and promotion of calcification. Those processes are regulated by an epigenetic process involved in expression of different epigenetic factors. Clarification of these pathways may aid development of novel therapeutic strategies to treat atherosclerotic hypertension, and its complications, including the acute coronary syndromes. The attenuation of apoptosis and calcifiification increases the cellularity and reduces stiffness and vulnerability for rupture in the arterial wall.

Biography

Yong-Jian Geng graduated from Suzhou Medical College, China in 1982. In 1994, he obtained Doctoral degree of Medical Science from Goethenburg University, and then pursued his Cardiology fellowship, and in 1995, was appointed as Instructor of Medicine at Harvard Medical School. In 1999, he joined the Faculty of School of Medicine, University of Texas Health Science Center at Houston. In 2000, he was appointed Director of the Center for Cardiovascular Biology and Atherosclerosis Research, and in 2001, he was named Director of Stem Cells and Heart Failure Research at Texas Heart Institute. He was promoted to Professor of Medicine in 2005.

Yong-Jian.Geng@uth.tmc.edu