Controlling Cardiac Transport and Plaque Formation

ZE'EV ARONIS, SAGI RAZ, ELISHA J.P. MARTINEZ, SHMUEL EINAV

Tel Aviv University, Tel Aviv Israel & NY Stony Brook University , NY ,USA

ABSTRACT: Macro-particles transported in the blood stream, such as LDL particles and macrophages, are considered to be one of the initiating factors of an atherosclerotic plaque development. LDL infiltration from the blood stream into a blood vessel's wall, whether coronary arteries, peripheral arteries or the carotid, are considered nowadays as a major inflammatory factor, recruiting macrophages from the blood flow and leading to the formation of vulnerable atherosclerotic plaques (VP). Infiltration sites are influenced by patterns of blood flow, as regions of lower shear stresses and high oscillations may give rise to higher infiltration rates through the endothelium, exacerbating the growth of a plaque and its tendency to rupture. Previous studies demonstrated a high prevalence of rupture sites proximal to the minimum lumen area, which raised the question as to weather the existence of two distinct adjacent plaques, in which the distal plaque is more severe, can give rise to hemodynamic forces that can push the non-stenotic plaque to rupture.

Models of the coronary arteries with one and two eccentric and concentric stenotic narrowing were built into a closed flow loop. The single stenosis model had a 75% area reduction narrowing (representing the VP) with relevant elastic properties. The double stenosis model included an additional distal 84% area reduction narrowing. The flow in the area between the two stenoses was recorded and analyzed using Continuous Doppler Particle Image Velocimetry (CDPIV), together with the hydrostatic pressure acting on the proximal plaque. Results indicated that the combined shear rates and pressure effects in a model with a significant distal stenosis can contribute to the increase in plaque instability by LDL and enhanced macrophages uptake. The highly oscillatory nature of the disturbed flow near the shoulder of the VP is enriching its lipid soft core, and the high hydrostatic pressures acting on the same lesion in this geometry, induce high internal maximal stresses that can trigger the rupture of the plaque.

KEYWORDS: Vulnerable plaque, atherosclerotic, LDL infiltration, Continuous Doppler Particle Image Velocimetry (CDPIV)

Address for Correspondence: Prof Shmuel Einav, PhD, Dept. of Biomedical Engineering, Tel Aviv University, Tel Aviv 69978, Israel, Fax: +972-3-6409448
e-mail: seinav@sunysb.edu