MCB, vol.16, supplemental 2, pp.32-33, 2019

Three Dimensional Finite Element Simulation of Atherosclerosis via Morphoelasticity

Navid Mohammad Mirzaei[1] and Pak-Wing Fok[1,*]

[1]University of Delaware, Department of Mathematical Sciences, Newark DE 19716, USA.

[*]Corresponding Author: Pak-Wing Fok. Email: pakwing@udel.edu.

Abstract: Atherosclerosis is a disease considered to be one of the leading causes of death. Understanding the behavior and dynamics of the vessel wall before and after atherosclerosis has been a motivation for many studies. We investigate this phenomenon as a combination of mechanical deformation of the vessel wall along with cell and chemical dynamics that occur within.

We consider the vessel wall as a growing hyperelastic material with three layers; intima,media and adventitia. Each of these layers have a different set of mechanical properties [1]. To describe tissue growth, we use morphoelasticity as the mathematical framework. The growth tensor in our study is a function of Platelet Derived Growth Factor (PDGF). To include the stiffening effect of collagen fibers we employ a Holzapfel-Gasser-Ogden anisotropic strain energy function [2]. We construct a functional that accounts for the total energy stored in the artery. This includes both the volumetric and the surface energy. The latter, comes from the effect of the blood pressure on the endothelial wall. Since the blood pressure is a live load exerted on the deformed domain, extra caution is required for its formulation. By minimizing the total energy functional we acquire a displacement field and consequently the stress field for the artery during the growth process. In addition, we explore the distribution of oxidized lipids, macrophages, foam cells, oxygen and necrotic cells in the intima at each growth step via a system of PDEs. All numerical simulations are carried out via the finite element method on the FENICS framework.

Altogether, this allows us to observe intimal thickening as a result of PDGF induced vessel growth along with histological changes within the wall such as the development of necrotic zones. Our simulations show results similar to the images acquired from ultrasounds scans.

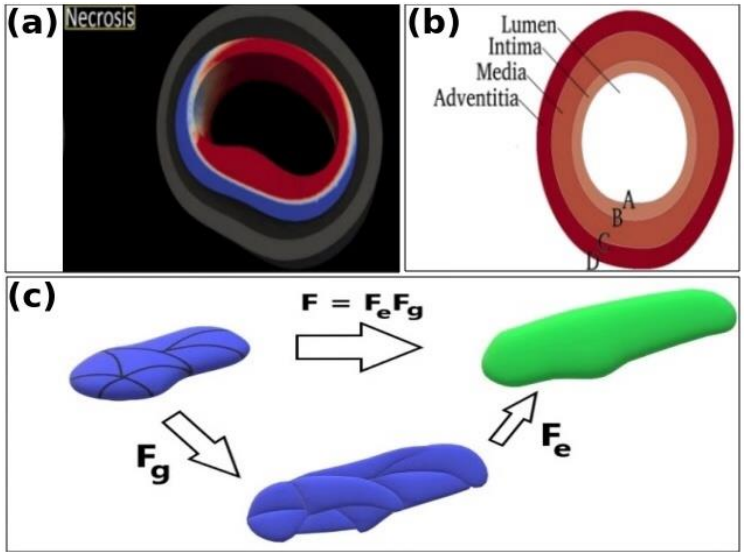

Figure 1: (a) Simulation of necrotic core formation. (b) Three layers of an arterial cross section. (c) Decomposition of deformation gradient into product of growth and elastic tensors.

Keywords: Arterial Mechanics; morphoelasticity; growth; finite element simulation

Acknowledgement: This work was funded by NIGMS IDeA Grant U54-GM104941 through the ACCEL program.

References

1. Holzapfel GA. Nonlinear solid mechanics: a continuum approach for engineering science. *Meccanica* **2002**, 37(4): 489-490.
2. Holzapfel GA, Sommer G, Gasser CT, Regitnig P. Determination of layer-specific mechanical properties of human coronary arteries with nonatherosclerotic intimal thickening and related constitutive modeling. *American Journal of Physiology-Heart and Circulatory Physiology* **2005**, 289(5): H2048- H2058.
3. Mehta SK, McCrary JR, Frutkin AD, Dolla WJS, Marso SP. Intravascular ultrasound radiofrequency analysis of coronary atherosclerosis: an emerging technology for the assessment of vulnerable plaque. *European Heart Journal* **2007**, 28(11): 1283-1288.
4. Gonzalo N, Garcia-Garcia HM, Regar E, Barlis P, Wentzel J et al. *In Vivo* Assessment of High-Risk Coronary Plaques at Bifurcations with Combined Intravascular Ultrasound and Optical Coherence Tomography. *JACC: Cardiovascular Imaging* 2009, 2(4): 473-482.