NUMERICAL PLAQUE PROGRESSION IN CAROTID ARTERY DISEASE: A MULTILEVEL APPROACH

Vassiliki T. Potsika *, Michalis D. Mantzaris *, Panagiotis K. Siogkas *, Ioannis G. Pappas *, Themis P. Exarchos *, ** and Dimitrios I. Fotiadis*, ***

* Unit of Medical Technology and Intelligent Information Systems, Department of Materials Science and Engineering, University of Ioannina, GR 45110 Ioannina, Greece
** Department of Informatics, Ionian University, Plateia Tsirigoti 7, GR49100 Corfu, Greece
*** Institute of Molecular Biology and Biotechnology, Foundation for Research and Technology-Hellas (FORTH)

vpotsika@gmail.com, md.mantzaris@gmail.com, psiogkas4454@gmail.com, giannis.pappas@gmail.com, themis.exarchos@gmail.com, fotiadis@cc.uoi.gr

Introduction

Vascular atherosclerotic plaques in the carotid artery bifurcations lead to progressive narrowing of the vessel lumen, which may erode or rupture, causing thromboembolism and cerebral infarction, manifested as stroke. The aim of the project TAXINOMISIS is to develop a new approach for the stratification of patients suffering from carotid artery disease. TAXINOMISIS focuses on the pathobiology underlying symptomatic plaques, discriminating distinct disease mechanism-driven states and biomarkers. The main outcome will be a software platform which can perform the risk stratification.

Materials and Methods

The TAXINOMISIS risk stratification platform will be composed by: a) a numerical model of plaque progression based on fluid dynamics and finite element analysis of 3D-reconstructed carotid arteries, b) an agent based model of plaque growth, c) the integration of the two models into a hybrid model, and d) the implementation of big data analytics including machine learning and data mining approaches for the integration of model outcomes with available big data in order to stratify patients according to the risk of manifesting cerebrovascular events.

The computational model of plaque progression will be based on a three-level approach. More specifically, the first level comprises advanced 3D-reconstruction of the plaque and carotid artery bifurcation. The second level includes the blood fluid dynamics modeling, together with the transport of lipoproteins into the arterial wall, based on finite element analysis. The Navier Stokes equations are solved in order to estimate blood velocity, pressure and wall shear stress (WSS). The aim of this level is to identify the regions of low WSS and high LDL accumulation, which have been correlated with sites of plaque progression [1]. The third level, models the major mechanisms of atherosclerotic plaque growth (e.g. monocyte infiltration and differentiation into macrophages, oxidation of LDL, foam cell formation).

Results and Discussion

In Fig. 1, the WSS distribution is depicted from a patient enrolled to the observational clinical study. The computational model was established using 3D-reconstruction of Magnetic Resonance Imaging data. The blue areas depict areas of very low WSS in the range 0-0.5 Pa. Areas of low WSS appear at the outer sites of the bifurcation.
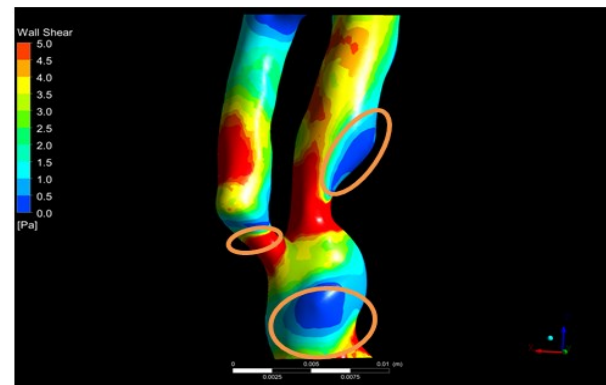

Figure 1. Wall Shear Stress distribution.

The results indicate that the inner bifurcation area presents higher WSS values, whereas at the outer areas of the two side branches, lower values of WSS are identified, constituting them prone to develop atherosclerosis. This is in agreement with the current literature proposing that increased atherosclerosis is prone on developing at the outer side of the bifurcation site [2].

References

1. A. I. Sakellarios, *et al.*, "Natural History of Carotid Atherosclerosis in Relation to the Hemodynamic Environment," Angiology, vol. 68, Feb. 2017, pp. 109-118.
2. N.T.Le, *et al*., "Flow signaling and atherosclerosis," Cell Mol Life Sci, vol. 74, May. 2017, pp. 1835-1858.

Keywords:

Carotid artery disease, computational modeling, wall shear stress.