Computational investigation of wall shear stress-driven in-stent restenosis

Gianluca De Santis

Supervisors: Verhegghe B., Verdonck P.

I. INTRODUCTION

When an artery occludes the insufficient blood supply to downstream districts may lead to myocardial infarction, stroke or necrosis. To reopen the artery a stent is inserted through a catheter into the stenosed location. After some weeks from the intervention the artery can re-occlude again as result of a patho-physiological remodelling process called in-stent restenosis. It is documented that restenosis rate ranges from 20 % to 30 % for bare metal stents, therefore requiring additional surgical interventions [1]. The occurrence of restenosis is partly related to alteration of the flow into the artery produced by the presence of the stent and detected by the endothelial cells. In this study we try to understand the relation between stent design, endothelial Wall Shear Stress and restenosis.

Fig. 1. *The axisymmetric model of stented artery. The quadrilateral mesh is refined near the wall in order to avoid underestimation of the WSS. The different regions of the boundary are shown.*

II. METHODS

PyFormex is an open source software under development at Ghent University (http://pyFormex.berlios.de) dedicated to pre- and post-processing both in FEA and CFD problems. An axisymmetric geometry of stented arterial lumen (a lumen with 2 strut sections) has been designed in PyFormex and meshed with quadrilateral elements (Fig.1). Other vessel/strut dimensions have been also tested by using the parametric design capabilities of the python-based PyFormex scripts. The evolution of the restenosis as local reduction of the lumen has been simulated following the scheme in Fig.2. The quadrilateral mesh (coordinates of the nodes and connectivity) was exported from PyFormex into a commercial software for CFD analysis (Fluent). A journal file set the fluid properties to approximate blood as Newtonian fluid (density 1060 kg/m^3 and viscosity 3.5 mPa•s) and defined the boundary conditions: parabolic velocity profile at the inlet (defined through a user defined function), zero pressure at the outlet and no-slip condition on arterial wall and stent surface. An axisymmetric steady state analysis was performed and WSS values at endothelium nodes were recorded and used to alter the previous lumen geometry: all the nodes experiencing a WSS <0.5 Pa where displaced towords the lumen axis. When no nodes had WSS in the atherogenic range the end of the loop was reached so that the geometry represented the lumen configuration at the end of the restenotic process [2].
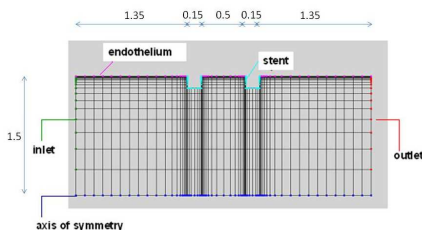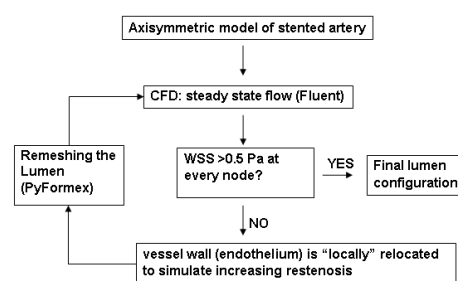
Fig. 2. *The iterative process simulating the in-stent restenosis: endothelial WSS discriminates between active areas (subjected to intimal thickening) and quiescent areas. At the end of each step PyFormex recreates the mesh by locally reducing the lumen for a new CFD analysis.*

III. RESULTS

The simulation of the restenosis has been found to converge, reaching a configuration in which all the nodes have WSS > 0.5 Pa. This confirms the expected negative feed-back between WSS and vessel remodelling: if a low WSS occurs in a large lumen, the vessel narrows, the velocity increases (in order to keep constant the flow even through a smaller section) and the WSS tends to increase. It is evident from Fig.3 that the sites of greatest remodelling have been found on the sides of the stent struts, both proximal and distal, which corresponds to lowest velocity gradient (the density of isolines decreases in this area).

Fig. 3. *Contours of Velocity Magnitude (m/s). The isolines become denser near the wall except at the sides of the struts, where low WSS is found. The top is the initial configuration whereas the bottom is the lumen geometry after 35 iterations.*
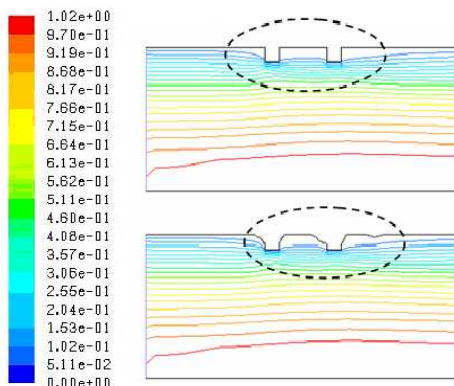
IV. DISCUSSION

The present numerical study describes an active adaptation framework of a living vessel in response to altered fluid-dynamic situation due to the presence of a stent. It aims to explain the in-stent restenosis through a mechano-biological approach. Some authors have already tried to predict the in-stent restenosis but they limited their investigation to the immediate post-expansion situation only, when the natural strut embedding has not occurred yet [3]. The present model is a necessary step towards a future 3D model which would help to compare different stent designs with respect to the amount of restenosis and maybe provide a virtual tool for preclinical testing.

V. REFERENCES

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