Wall Shear Stress Analysis in a Stented Coronary Artery and Numerical Simulation of Restenosis

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Abstract

Restenosis is a frequently encountered complication after angioplasty interventions. This phenomenon often begins and develops mostly at the ends of an implanted stent . Three major factors are believed to contribute mostly to the edge restenosis: (a) mechanical damage of the artery's wall caused by the stent implantation, (b) interaction between the stent and the blood constituents and (c) endothelial growth stimulation by small (lower that 1.5 Pa) and oscillating wall shear stress (WSS) [1]. The aim of the current research is to analyse how geometrical changes inflicted by the stent implantation affect the WSS distribution, and show that dangerous WSS values can be sustained in the vicinity of the stent's edges during large fraction of the cardiac cycle (if not permanently). Then assuming that permanently low WSS is particularly important in cellular proliferation, a numerical model of restenosis basing on wall shear stress distribution in the stented artery was elaborated.

Keywords: coronary artery disease, coronary blood flow,in-stent restenosis

1. Introduction

Blood vessels have living and deforming walls, which internal surfaces are covered by a layer of endothelial cells. The endothelium constitutes an anticoagulation barrier preventing platelets deposition and clotting. The localization of the endothelial cells exposes them to mechanical stimulation from the flow which may cause damage leading to morphological changes. In consequence, functioning of the endothelium can be disturbed and pathology may develop.

In the places of the endothelial cells' damage, a gradual accumulation of platelets deposit occurs, reducing the lumen's diameter and blood delivery to the cardiac muscle. Apart of a pharmacological treatment, non-surgical mechanical widening using inflating balloons and stents are applied. Each year almost one million of angioplasty interventions and stents implantations are made all over the world. Unfortunately, in 20-30% of cases neointimal proliferations leads to restenosis occurring within the following period of 3-6 months.

The factors contributing to restenosis development are:

- mechanical damage of the artery's wall caused by the stent implantation,
- interaction between the stent and the blood constituents
- endothelial growth stimulation by small (lower that 1.5 Pa) and oscillating wall shear stress (WSS).

The last mentioned factor seems to be particularly important. It has been proved that low and oscillating WSS stimulate endothelium in such way that cellular growth is generated which may lead to restenosis[1].

In the clinical practice, it is accepted to implant stents whose diameter exceeds the arteries one - precaution taken, to avoid stent displacement after the intervention. This generates a sudden geometry change and related regions of low wall shear stress may appear. Another important issue is the curvature of the vessel [2]. This is why in the current study, we first made a WSS analysis for three different stent's diameters, to show how this affects low-WSS regions presence. Then we considered a simple mechanical model of the restenosis development.

2. WSS analisys in function for three different stent's diameters

A 3D realistic model of the left main coronary artery, including the LAD and LCX branches, has been made. The presence of the implanted stent is modelled by the artery's wall deformation (Figure 1).

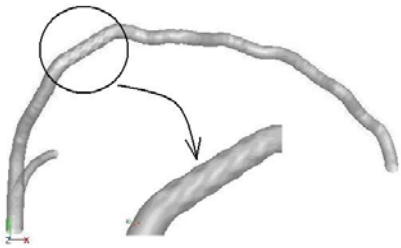

Figure 1: Model of coronary artery with stent

Blood is considered as a newtonian fluid with a viscosity of 3.5×10^{-3} Pa.s, and with a density of 1060 kg/m^3. Blood flow at the inlet has a realistic time variation and a constant split between LAD (70%) and LCX (30%) branches. The diameter at the inlet of the left main coronary artery is 5mm, LAD diameter is 4mm, and LCX – 3mm. The modelled stent is 15mm long and its simulated diameters – 4 mm, 4.1 mm and 4.2 mm. The flow is determined by means of the commercial CFD solver Fluent 12.1, using the grid of about 1 milion tetrahedral finite volumes. The arterie's wall is assumed to be rigid.

During the study regions of low WSS remaining permanently in the dangerous range were sought. Such state appears near the stent edges due to sharp changes of curvature of the artery related to the increased rigidity of the artery segment with implanted stent [2]. This geometric feature, together with the strong flow pulsations, is responsible for existence of large regions of separated flow with permanently low WSS values. As the numerical simulations confirm, the larger is the stent the greater are regions of low WSS persisting during whole cardiac cycle at the ends of the stent (Figures 2).

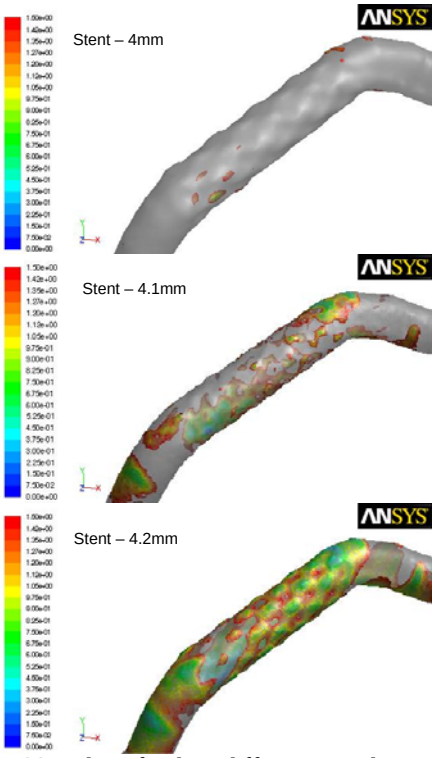

Figure 2: WSS analysis for three different stent diameters.

3. Restenosis simulation

The process of endothelial growth has been assumed to be sensitive only to time-avereged flow characteristics and thus the quasi-static approach to the restenosis development is plausible.

The main idea consists in performing the sequence of flow simulations inside a gradually changing geometric models of the stented artery with progressing stenosis. In each step of this procedure, several cardiac cycles of pulsatile flow in a fixed domain are performed to determine reliably the time-averaged characteristics of the wall stress distribution. Next, the geometric change of the vessel wall is determined and the finite volume grid for the next step is prepared.

The key ingredient of the above procedure is the way how the change of the wall shape is determined. The general assumption in this work is that the absolute value of the endothelial growth is basically not known; the only information available is the relative rate of growth, which is determined by some time-averaged characteristic of the WSS distribution. We will also assume that the growth occurs only in the spatial direction normal to the wall, as well as the maximal displacement p_{max} of the wall surface in per one step of the simulation. Then, the actual radial displacement of the each grid node at the wall has been defined as the averaged-WSS dependent fraction of the maximal displacement. The range of WSS where the relative displacement increases from 0 to 1 has been arbitrarily chosen to be [0.6 Pa, 1.5 Pa]. In other words, it assumed that WSS larger than 1.5 Pa are essentially inactive in the process, while WSS smaller then 0.6 Pa lead to exactly the same growth rate of the endothelial cells in wall-normal direction.

The results presented in Figures 3 and 4, show the comparison between the initial geometry of the stented artery and the artery with developed 50% restenosis. The simulations were performed for two different maximal displacements: 0.0001 m and 0.0002 m.

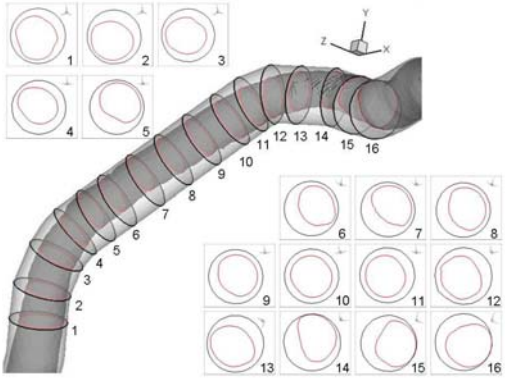

Figure 3: Comparison between the stented artery, and the artery with developed restenosis for $p_{max}= 2 \cdot 10^{-4}$ m

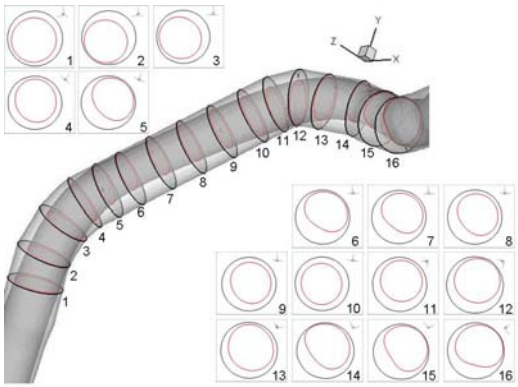

Figure 4: Comparison between the stented artery, and the artery with developed restenosis for $p_{max}= 10^{-4}$ m.

4. Conclusions

Applying CFD methods, the flow simulations in stented LAD have been performed. Regions of permanently low WSS and thus especially prone to intimal proliferation have been localized. According to this results, a simple restonosis development model has been used to obtain the shape of the obstructed artery. This model seems to be quite successful in predicting the sequence of shapes that might be attributed to the consecutive stages of the restenosis. On the other hand, even if predicted forms of restenosis look realistically, the actual dynamics of the process is completely missing. The real and clinically useful prediction (if possible at all) might be provided only by a full model which accounts for cellular physiology and its full (not only mechanical) interaction with the blood flow. To the author's best knowledge we are still quite far way from such model.

References

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