EVALUATION OF STENTING-INDUCED VASA VASORUM COMPRESSION

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Introduction

Percutaneous Transluminal Angioplasty (PTA) is the most efficient therapy for atherosclerosis [1]. However, stenting has still a significant rate of re-stenosis, caused by Neointimal Hyperplasia (NH) formation. Recent studies showed the strong relation between vascular wall hypoxia and in-stent-restenosis [2]. Oxygen deficiency in the artery is likely related to Vasa Vasorum (VV) compression by the stent [3]. Vasa Vasorum are capillary vessels supplying oxygen to the artery. When the stent is over-expanded, it leads to acutely lower oxygen concentrations in the arterial wall and greatly increase the risk of NH formation [4]. Therefore, our study evaluates Vasa Vasorum compression after PTA, simulating three degrees of stent expansion, by means of a Finite Element Analysis (FEA, Abaqus/CAE 6.14-2).

Methods

We created an idealized multi-layered fibroatheroma model in SolidWorks (Dassault Systemes, v. 2016), comprising the Intima, Media, and Adventitia layers as thick walled non-linear elastic cylindrical tubes. The plaque consists of a semi-annular lipid core placed in the intimal layer, causing a 60% stenosis. The three arterial layers are described using Holzapfel anisotropic hyperelastic material model while the lipid core is considered as a nearly incompressible elastic material [5, 6]. Four Vasa Vasorum trees, with three branches of decreasing diameter in the axial and radial direction, penetrate into each vascular layer as empty cavities. The artery is expanded by a Multilink RX Ultra stent made of Stainless Steel 316L. The simulation is based on a dynamic quasi-static implicit analysis of one second time step. The stent is expanded by a linearly increasing internal pressure, reaching a stent/artery luminal diameter ratio of 1.1:1, 1.2:1 and 1.3:1 in different simulations. The two ends of the artery are fixed, reflecting the constraint from the human body environment.

Results

The VV compression was evaluated for the three descending branches (VV1, VV2, VV3) of the four micro vessels. Data were also discerned between top, right, bottom and left cross sectional positions **[Figure 1]**. The cavities beneath the plaque (bottom) experience the smallest degree of compression, while the surrounding regions are highly affected by stent expansion. The greatest decrease in diameter is caused by stent over-expansion and is located in the Media with a maximum compression of nearly 80% **[Figure2]**. Following Poiseulle's law, the fold-increase in flow resistance was calculated **[Table1]**. Here, the blood viscosity and VV length were considered as constant. The results take into consideration the organization "in series" of the branches.

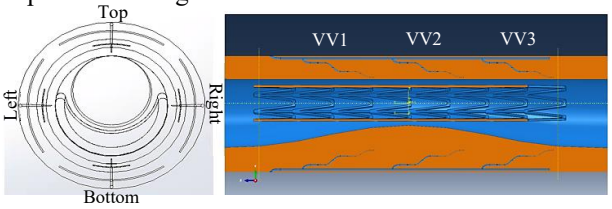

Figure 1: Illustration of the VV layout in cross and lateral sections.

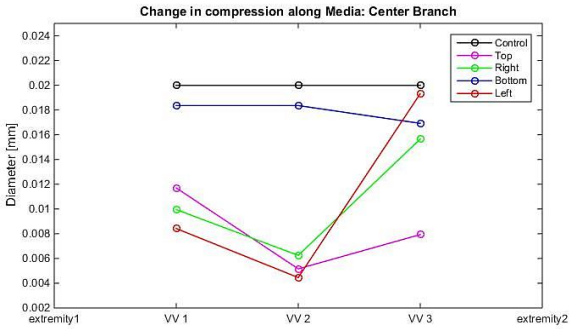

Figure 2: Plot for change in compression along the Media, central branches.

Table 1: Fold-increase in resistance to flow in arterial layers.

Central Branches	Top	Right	Bottom	Left
Intima	**10.8**	**12.2**	**1**	**31.8**
Media	**11.1**	**11.9**	**1.1**	**31.1**
Adventitia	**1.1**	**0.6**	**1**	**0.6**

Discussion

Results clearly show the influence of final stent diameter on the Vasa Vasorum. Stent over-expansion induces a drastic drop in VV diameters, especially on healthy regions around the plaque. The atheroma seems to protect the Vasa Vasorum beneath it. The highest VV compression in the Media agrees with previous findings of Media necrosis and loss in elasticity in case of VV impairment [3, 7]. The resulting resistance to blood flow soars steeply, depicting an acute hypoxic situation. Assuming the pressure gradient at the VV inlet and outlet to remain constant, the blood flow would drop drastically, up to more than 30 times in some regions.

References

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