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CARDIATIS—TEHMED

Finite element analysis in detection of AAA weak spot(s)

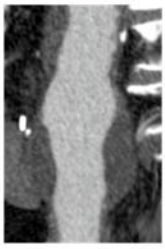
Cardiatis/TehMED

Finite element analysis calculates weak spots of the abdominal aortic aneurysms

As the abdominal aneurysms grow and become larger, the biomechanics of the wall gradually changes, primarily due to the extracellular matrix integrity degradation, which is considered to be spatially variable. Quite a number of clinical articles shows that local factors, like wall shear stress and hypoxia have negative influence on the vessel wall biomechanical strength and as such promote extracellular matrix degradation. Parts of the vessel wall are becoming stiffer, thereby containing less elastin and collagen, as compared to normal aorta. It is widely accepted that the AAA arise and progress, as the stress in the aneurysm exceeds the stress of the normal vessel wall.

Case I

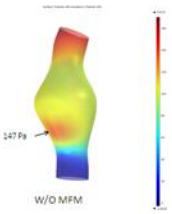
Abdominal aortic aneurysm is presented in the MPR (multiplanar reformat view) view, showing the residual lumen and the aneurysm sack, partially thrombosed.



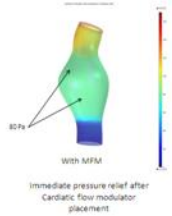
An aneurysm mash is created out of the delivered CT scan (see picture to the right).

3D pressure modeling

A calculation of the pressure distribution within the aneurysm is calculated, based upon the local hemodynamic parameters (see to the right).



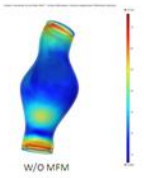
The Cardiatis Flow modulator is virtually positioned and the new situation is calculated. An immediate and substantial pressure release is achieved!



Immediate pressure relief after Cardiatic flow modulator placement

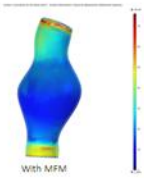
3D stress modeling

The wall shear stress distribution within the aneurysm prior to the stent placement is presented to the right.



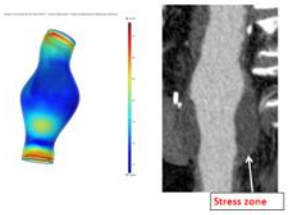
Maximum stress appears at the maximum pressure zone

The wall shear stress distribution after the Cardiatis flow modulator placement (virtual deployment) is presented to the right.

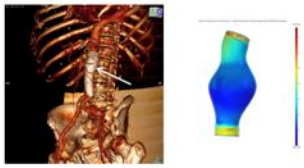


Immediate stress reduction after Cardiatis flow modulator

The picture below shows how the calculated parameters can be converted to the initial CT scan, allowing easy assessment of the weak spot prone to rupture.



The following picture presents the final result, after the Cardiatis flow modulator placement as well as the corresponding computed flow dynamics calculation.



Conclusion:

The pressure on the aneurysm wall dropped significantly after the Cardiatis flow modulator placement. The maximal pressure on the weak spot, determined with the computed flow dynamics, disappeared after the stenting.

The wall shear stress after the Cardiatis flow modulator placement is dramatically lowered, allowing for vessel wall preservation.

Inside this issue:

Special points of interest:

- *Finite element analysis is able to predict weak spots in the aortic aneurysm wall, based on the pressure and wall shear stress calculations, based on the local hemodynamic parameters.*
- *Local extracellular matrix degradation play an important role in the pathophysiology of the aneurysmal disease.*

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TehMED— Who Are We?

TehMED is a young and dynamic company based in Ljubljana - Slovenia. TehMED is the certified distributor of the Cardiatis (Isnes, Belgium) products for the Slovenian market. The company is primarily focused at the distribution of the medical products, used in the interventional radiology and minimally invasive surgery.

Presentation of the Cardiatis product range can be done upon request to the Commercial and Marketing Department of the TehMED company (see the contact data to the left).

Aortic abdominal aneurysm wall shear stress—basics

Abdominal vessel wall configuration and consistency is severely affected by changes in mechanical forces or deformations, caused by the local blood flow hemodynamics. Under the normal physiological conditions, the microstructure of the abdominal vessel wall provides enough strength to preserve the wall consistency. However, when the local hemodynamic is disturbed, elastin concentration will decrease proportional to the increase of the vessel diameter. Aortic wall stress is known to be proportional to its diameter, following the Law of Laplace. Nevertheless, local areas of dilatation occur in response to spatial changes in wall stress. The "blebs" (local saccular outpouchings) within the aneurysm wall are known to be the sites of increased rupture risk, due to the increased stress concentration. Histopathology analysis of the blebs showed decreased collagen and elastin, as opposed to the normal vessel wall tissue.

Increased mechanical forces inside the aorta cause extracellular matrix production by smooth muscular cells, endothelial cells and other cells. A disrupted extracellular matrix homeostasis will lead to weakening of the aortic wall. Degradation of the extracellular matrix through elastolysis and smooth muscular cell loss will cause reduced biomechanical strength, progressive weakening of the vessel wall, generation of aneurysms and dissections and finally highly-fatal aneurysms.

One of the most prominent contributors to extracellular matrix degradation are the matrix metalloproteinase's (MMPs). MMPs are overexpressed in aortic aneurysms (especially in TAAs), where they control degradation of elastin and collagen. The MMPs are elevated in both TAAa and AAAs and are primarily caused by hemodynamic stress, aneurysm size and aneurysm growth rate. It is important to mention that other proteolytic enzymes, apart from MMPs, are elevated as well (e.g. tPA and urokinase plasminogen activator) when exposed to mechanical stresses. Other important factor that causes increased MMPs production are macrophages. They are involved in the inflammatory response that occur with AAA. Macrophages are increased in areas where atherosclerotic plaque is accumulated and an increased MMP production is observed. The local smooth muscular cells, fibroblasts and macrophages somehow "sense" the mechanical forces and start producing proteolytic enzymes. Elevated MMPs degrade afterwards the extracellular matrix and weaken the integrity of the aortic wall., leading to aneurysm formation and risk for rupture.

In conclusion, the local hemodynamic forces cause increased wall shear stress and as such lead to formation of aneurysms. More specifically, local extracellular matrix degeneration coincides with the spots of increased wall stresses. This lead afterwards to compromised integrity of the aortic wall and aneurysm formation. Obviously, there is large range of spatial variations of wall strength that are patient specific and are corresponding to the local hemodynamic forces.