

Numerical Investigation of the Hemodynamic Environment Change in Patient-Specific Intracranial Aneurysm with Progressive Stenosis in Unilateral Internal Carotid Artery

Guangyu Zhu¹, Yuan Wei¹, Qi Yuan^{1*}, Ge Yan² and Jian Yang²

1 Introduction

Intracranial aneurysm (IAs) is a frequently localized cerebral vascular disorder of an overall prevalence of 5-8% [Cebal (2013)]. Subarachnoid hemorrhage (SAH) caused by IAs rupture is one of the major causes of mortality and morbidity in the modern world. Local hemodynamic characteristics played important roles in the rupture of IAs and has been studied extensively from different perspectives [Liu (2015); Brinjikji (2017)]. However, the influence of progressive cerebral stenosis on the hemodynamic of the distal cerebral aneurysm is yet to be further investigated. In this study, a set of patient-specific computational fluid dynamics (CFD) simulations were performed to investigate the impact of internal carotid artery (ICA) stenosis growing on the hemodynamic environments in an anterior communicating artery aneurysm (ACoAA).

2 Methods

CT images of a 66-year-old male patient with an ACoAA were obtained with a 64-detector row spiral CT scanner (Aquilion 64, Toshiba Medical Systems, CA, USA). The entire anterior circulation, including the bilateral ICAs, the bilateral proximal middle cerebral arteries (MCA-M1), the bilateral anterior cerebral arteries (ACAs) and the anterior communicating artery (ACoA) with an ACoAA, were three-dimensionally reconstructed by using commercial software MIMICS (Materialise Inc., Leuven, Belgium). To simulate the stenosis progress of ICA, a series of stenosis degrees (0, 15%, 50% and 75%) were artificially added to the right ICA (R-ICA). The stenosis degrees were defined as instructed in the North American Symptomatic Carotid Endarterectomy Trial (NASCET).

In the CFD simulations, blood was assumed as a Newtonian fluid with a density of a density of 1060 kg/m^3 and a viscosity of $4 \text{ mPa}\cdot\text{s}$. For simplicity and to reduce the computation costs, the elasticity of the arterial wall was neglected as well, a non-slip boundary condition was specified on all the walls and interfaces.

The pulsatile volumetric flow rate of ICA acquired from 4D MRI measurement [Wählin (2013)] were imposed at the proximal ends of the ICAs as inlet boundary condition, and

¹ Xi'an Jiaotong University, Xi'an, 710049, China.

² The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, 710061, China.

* Corresponding Author: Qi Yuan. Email: qyuan@xjtu.edu.cn.

three elements Windkessel models were specified at distal ends of ACA and MCA-M1 as outlet boundaries, respectively.

3 Results

With the increasing of the R-ICA stenosis degree, a drastically change of flow field in the ACoAA can be observed. A jet flow comes from RACA-A1 under no stenosis case gradually shifts toward left with the increased stenosis degree, and finally the blood flow ejected from LACA-A1 becomes the dominate jet. Meanwhile, the impinging area in the ACoAA dome shift from top to the left with the increasing of stenosis degree. Moreover, the low wall shear stress (WSS) area ($WSS \leq 1.5$ Pa) on the ACoAA dome decreased with the increasing of stenosis rate. The high oscillatory shear index (OSI) area ($OSI > 0.2$) changes in the same pattern as well as low WSS. In addition, the overall pressure on the ACoAA dome also decreased with the growing of unilateral ICA stenosis.

4 Conclusions

The results of the current study showed that the hemodynamic environments in the distal IAs might be changed dramatically due to the narrowing of stenosis in proximal cerebral vascular. Contrarily, sudden changes of flow characteristics in IAs can be anticipated as well when the stenosis was relieved after clinical treatments, such as carotid endarterectomy (CEA) or carotid artery stenting (CAS). The findings of this study would enhance the understanding of the impact of stenosis degree of proximal arteries on the flow in distal IAs, and eventually lead to therapeutic and diagnostic applications in the future.

References

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