Hemodynamic Analysis of Intracranial Aneurysms with Atherosclerosis

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Abstract— **Intracranial aneurysms can have atherosclerotic wall properties that may be important in predicting aneurysm history. This study aimed to investigate hemodynamic characteristics of atherosclerotic legions in intracranial aneurysms. We conducted computational fluid dynamic analyses of 30 aneurysms using patient-derived geometries and inlet flow rates. Among 30 aneurysms, seven atherosclerotic lesions with remarkable yellow lipid deposition were identified in five aneurysms. All seven atherosclerotic lesions were spatially agreed with the area exposed to stagnant blood flow. Univariate analysis revealed that male (P = 0.031), cigarette smoking (P = 0.047) and the exposure to stagnant blood flow (P = 0.024) are significantly related to atherosclerotic lesion formation on the aneurysmal wall. Of those variables that influenced atherogenesis, the variable male (P = 0.0046) and the exposure to stagnant flow (P = 0.0037) remained significant in the multivariate regression model. In conclusion, male sex and stagnant blood flow inside aneurysms were independent risk factors for atherosclerosis in intracranial aneurysms.**

Keywords— **cerebral aneurysm, hemodynamics, wall shear stress, stagnation**

I. Introduction

Recently, potentials of computational fluid dynamic (CFD) simulation in predicting natural history of intracranial aneurysms have been reported.[1] However, little work has been done on the atherogenesis in intracranial aneurysms. The purpose of this study was to investigate hemodynamic characteristics of atherosclerotic legions in intracranial aneurysms.

We conducted CFD analysis of 30 unruptured middle cerebral artery (MCA) aneurysms and investigated the relation between the spatial distribution of atherosclerotic lesions on the aneurysm wall and hemodynamic wall parameters including wall shear stress (WSS), oscillatory shear index (OSI), and relative residence time (RRT). WSS is the tangential frictional stress caused by blood flow on the vascular wall. OSI is a dimensionless measure of directional changes in WSS, and used as a marker of oscillatory nature of WSS.[2] RRT demonstrates the residence time of particles near the wall,[3] and was used as a marker of stagnant blood flow in this study.

II. Methods

This study was granted approval from the ethical committee in Kohnan Hospital. We conducted CFD analysis of 30 MCA aneurysms with atherosclerosis. Data sets of 3D rotational angiography were used to reconstruct accurate geometries of the aneurysms and adjacent arteries. Following the conventions of CFD in large vessels, blood flow was treated as an incompressible Newtonian fluid, vessel walls were assumed rigid and no-slip boundary conditions were applied at the walls.[4] A finite-volume package, ANSYS 12.1 (ANSYS Inc.; Lebanon, NH) was used to solve the governing equations: 3D unsteady Navier-Stokes equations and equation of continuity. The patient-specific pulsatile flow condition measured by magnetic resonance velocimetry was prescribed at the inlet boundary.[5] Traction free conditions were applied to outlets. Three pulsatile cycles were simulated to ensure that numeric stability has been reached, and the results from the third cycle were used for analysis.

Wall shear stress refers to the tangential frictional stress caused by the action of blood flow on the vessel wall. For pulsatile flow, the time-averaged wall shear stress was calculated by integrating WSS magnitude over a cardiac cycle for each tetrahedral element:

$$WSS = \frac{1}{T}\int_0^T |\vec{\tau}_w| dt \quad (1)$$

where $\vec{\tau}$ is the instantaneous wall shear stress vector and T is the duration of the cycle.

To describe the temporal disturbance of intra-aneurysm flow, oscillatory shear index (OSI), a dimensionless measure of directional changes in WSS, was calculated using the formula reported by He and Ku: [2]

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$$OSI = \frac{1}{2}\left[1 - \frac{\left|\int_0^T \vec{\tau}_w dt\right|}{\int_0^T |\vec{\tau}_w| dt}\right] \quad (2)$$

Note that $0 \leq OSI < 0.5$, with 0 being completely unidirectional shear and 0.5 being completely oscillatory.

Himburg et al showed that the residence time of particles near the wall is inversely proportional to a combination of WSS and OSI.[3]

$$RRT = \frac{1}{(1 - 2 \times OSI) \times WSS} = \frac{1}{\frac{1}{T}\left|\int_0^T \vec{\tau}_i \, dt\right|} \quad (3)$$

Himburg proposed RRT as a robust marker of disturbed blood flow with low and/or oscillatory WSS.[3] However, RRT may serve as a marker of stagnant blood flow, as the prolongation of RRT means the long residence time of particles near the wall.

In addition to conventional contour maps of WSS, OSI, and RRT, 3D streamlines and WSS vector plots were displayed to examine the temporal variation of wall shear stress vectors.

III. Results

Intra-operative video recordings were examined for all 30 cases. Seven atherosclerotic lesions on five aneurysms (5/30, 16.7%) were distinguished by remarkable yellow lipid deposition. All five patients had several vascular atherosclerosis risk factors such as male sex, old age, obesity, smoking history, hypertension, diabetes mellitus, or dyslipidemia.

Among the three hemodynamic variables examined in the current study, only RRT demonstrated qualitative agreement with the spatial distribution of atherosclerosis in all seven lesions as a single metric. The five lesions on the dome were exposed to low and oscillatory WSS at the center of vortex flow (Figure 1, 2). To evaluate the risk factor of atherosclerotic change of the intracranial aneurysms, statistical analyses were performed . Univariate analysis revealed that male (P = 0.031), cigarette smoking (P = 0.047) and maximum RRT (P = 0.024) are significantly related to atherosclerotic lesion on the intracranial aneurysmal wall. Of those variables that influenced atherosclerotic lesion of the intracranial aneurysmal wall, the variable male (P = 0.0046) and maximum RRT (P = 0.0037) remained significant in the multivariate regression model ($R2$ =0.52).

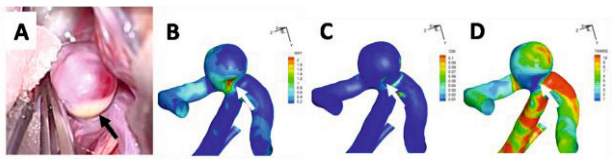

Fig 1. Intraoperative photograph and contour maps of three hemodynamic wall parameters.

A, Intraoperative photograph showed yellowish atherosclerotic legion on the aneurysm wall (black arrow). **B**, Contour maps of relative residence time (RRT) showed qualitative agreement of the prolonged RRT (white arrow) with the spatial distribution of atherosclerosis shown in A.

C, D, Contour maps of oscillatory shear index (OSI, **C**) and time-averaged wall shear stress (WSS, **D**) from the same viewing angle. The area with prolonged RRT had high OSI value and low WSS magnitude (white arrow).

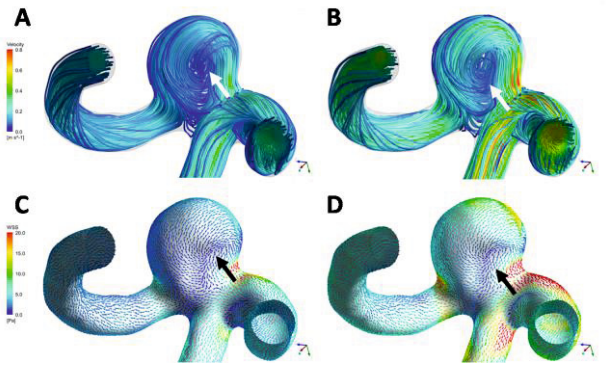

Fig 2. 3D streamlines and wall shear stress vector plots.

A, B, Snapshots of the flow field (3D streamlines) captured at end diastole (**A**) and peak systole (**B**) revealed intra-aneurysm vortex flow moving around over cardiac cycle (white arrow).

C, D, Snapshots of WSS vector plots captured at end diastole (**C**) and peak systole (**D**) showed that WSS vectors were arranged whirl-likely around the vortex center (black arrow) which was correspond to the area with RRT prolongation shown in Figure 1.

IV. Discussion

Many hemodynamic studies of intracranial aneurysms have reported that low WSS is involved in aneurysm rupture,[1,6] and speculated that low WSS can induce degenerative vascular wall remodeling in intracranial aneurysms that may lead to thinning or rupture of the aneurysm wall. However, the results of our study raise the possibility that an aneurysm wall exposed to low WSS can progress to atherosclerotic remodeling. We consider that low WSS is a risk for aneurysm rupture.[6] However, aneurysms with low WSS accompanied by the stagnation of blood flow indicated by prolonged RRT may be stabilized by atherosclerotic remodeling process. Stagnant blood flow prolongs residence time of atherogenic particles in the blood near aneurysmal wall, thus inducing lipid exchange and recruitment of macrophages, and promoting atherosclerosis.

V. Conclusions

The area exposed to stagnant blood flow indicated by prolonged RRT co-localized with atherosclerotic lesions on the aneurysm wall. Male and local stagnant flow were independent risk factors for atherosclerosis in intracranial aneurysms.

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