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Computational Fluid Dynamics Simulation of Aneurysm Initiation in Experimental Animal Models

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***Abstract*— We performed CFD simulations for the vessel of experimental animal models before an aneurysm initiation and observed tissue samples of the model which had the aneurysm by immunostaining. The stretching force on the vessel wall due to the blood flow degenerates both endothelial cells and smooth muscle cells, and the stagnation causes low wall shear stress, which illustrates inflammation, resulting in the initiation of a cerebral aneurysm.**

***Clinical Relevance*— Our results suggest that aneurysm initiation may be predicable by CFD simulations.**

I. INTRODUCTION

It has been reported that hemodynamics factors, as well as pathological factors such as inflammation in a cerebral vessel wall, are involved in the initiation of cerebral aneurysms. To calculate the hemodynamic parameters, computational fluid dynamics (CFD) has been used. However, relationships between pathological factors and hemodynamics effect on blood vessels remain unclear because it is difficult to observe the patient's cerebrovascular tissues directly. In this study, we performed CFD simulations for vessels of experimental animal models and conducted immunostaining of the tissues to investigate the hemodynamic effects on the vascular tissues related to aneurysm initiation.

II. METHODS

We used the experimental model of rats proposed by Shimizu et al. (2021) [1]. In this model, aneurysms were induced at the bifurcation created by end-to-side anastomoses with the bilateral common carotid arteries. Magnetic resonance imaging (MRI) had been conducted for the models weekly after the anastomoses. After three weeks of the observation, the cases which initiated the aneurysm were defined as De Novo cases and the other cases were defined as Stable cases. CFD simulations were performed using the MRI image acquired after a week from the anastomosis. As hemodynamic parameters related to aneurysm initiation [2], dimensionless wall shear stress (*WSS**) and *WSS** divergence (*WSSD**) were calculated. Immunostaining was performed for one case classified as De Novo cases and was observed for four weeks. As an indicator of damage and inflammation on the tissues, we observed endothelial cells (EC), smooth muscle cells (SMC), and macrophages at the aneurysm dome. In addition, these factors were also observed at the parent arterial wall as of the state of normal.

III. RESULTS

Four cases were classified as the De Novo case, and the other four cases were as the Stable case. Three De Novo cases had aneurysms at the apex of their bifurcation. CFD simulations showed that the apex of the bifurcations had lower *WSS** and higher *WSSD** compared to the other area in all eight cases(see Fig.1(A)). The averaged *WSS** of the De Novo cases was lower than that of the Stable cases. The immunostaining images illustrate that EC and SMC degenerated and a lot of macrophages (marked by circles in Fig. 1(B)) were located at the aneurysm dome, where is assumed to be originally the top of the bifurcation before the aneurysm formation, compared to the parent artery.

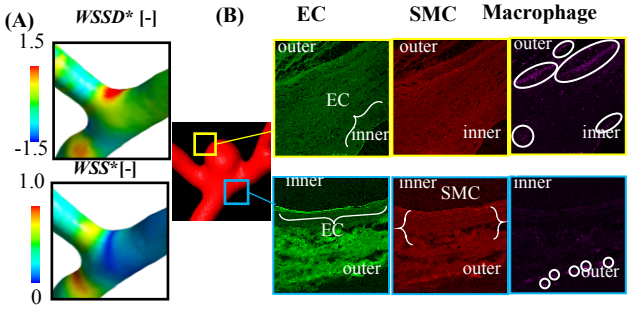

Figure 1. *WSSD** and *WSS**distribution.(A) Immunostaining images for EC, SMC, and macrophage (upper: aneurysm wall, lower: parent artery).(B)

IV. DISCUSSION & CONCLUSION

The CFD simulations and immunostaining results showed that EC and SMC degenerated and a lot of macrophages existed at the top of bifurcation where a low *WSS** and high *WSSD** area existed. High *WSSD**, which indicates the stretching of the vessel wall, may lead to the damage of EC and SMC. Additionally, the low *WSS**, which implies the flow stagnation, may cause inflammation at the top of bifurcation where many macrophages were observed. Although further study is needed, our results indicate that both high *WSSD** and low *WSS** become factors related to the initiation of cerebral aneurysms.

REFERENCES

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