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The Impact of Left Atrial Morphology on Thrombosis Risk in Atrial Fibrillation: revisiting Virchow's Triad

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Extended Abstract

Objectives: Atrial fibrillation (AF) is an arrhythmia disease that can disrupt the movement of the left atrium (LA), which will facilitate thromboembolism in LA and further exacerbate the stroke risk by a five-fold increase. The left atrial appendage (LAA) is a vital site for thrombus formation due to the high blood stasis possibility there. It is reported that about 90 % of the intracardiac thrombi in patients with cardioembolic events is derived from the LAA. The morphology of the LAA is believed to have a profound effect on thrombosis in the LA [1]. However, due to the limitation of observation and monitoring methods, the mechanism of this effect is still unrevealed. Virchow's triad is a theory of thrombogenic mechanisms, stating that endothelial dysfunction, blood hypercoagulability and blood stasis are the three main factors involved in thrombus formation. As Virchow's triad has withstood the test of time, it may be conductive to understand the prothrombotic state in AF.

Methods: Personalized Computational fluid dynamics (CFD) analysis has been conducted for twelve AF patients based on their medical images to get patient-specific hemodynamic parameters information. Among them, LAA thrombus is observed in four patients and none in the others. A consistent flowrate curve is applied at the mitral valve outlet and zero pressure is assumed at all pulmonary vein inlets. Besides, a reduced coagulation cascade model we developed before [2] was loaded to simulate the coagulation response occurring in the LAA. The performance of Virchow's triad is estimated based on the simulation results: endothelial dysfunction is evaluated by the distributions of endothelial cell activation potential (ECAP), blood hypercoagulability is commented based on the concentrations of coagulation factors, whereas the blood stasis is assessed by the distributions of velocity and relative residence time (RRT). As these three factors interact with each other to jointly regulate the formation of thrombus in the LAA, separate and comprehensive comparisons of these three factors is also carried out based on thrombosis potential scores.

Results: The simulation results show that the LAA is more prone to thrombosis than the main region of LA, because of the lower blood flow velocity and significantly higher ECAP values there, which indicates a higher risk of blood stasis and endothelial dysfunction, respectively. The comprehensive scores of thrombosis potential in patients with LAA thrombus are significantly higher than that in patients without LAA thrombus, indicating that the thrombogenic mechanism in LAA can be well analysed and explained by Virchow's triad. Furthermore, the potential of the proposed comprehensive score to improve stroke risk classification in patients with AF is also demonstrated. The morphology of LA has a significant influence on the performance of the three factors in Virchow's triad. However, the correlation between the performance of these three factors and the morphological type of LAA is weak, whether analysed individually or in combination. Therefore, if we want to quickly score LAA thrombosis risk in AF patients from the perspective of Virchow's triad, we need a more refined shape score that can better reflect patient-specific LA (including LAA) morphological features.

References

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