An Image-Based Computational Simulation of Pulmonary Embolism Using Radiological Images

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ABSTRACT: Pulmonary embolism is one of the most prevalent diseases amid hospitalized patients. However, this phenomenon has not been investigated in the field of biomechanics so far and insufficient information is available about hemodynamic factors affecting this phenomenon. In this research, a patient-specific anatomical model of pulmonary arteries has been constructed from computed tomography images. Navier-Stokes equations, as the governing equations, have been solved in an arbitrary Lagrangian-Eulerian formulation, and the fluid-structure interactions method was used. Viscoelastic parameters were adopted in accordance with the red blood clot (stemmed from deep veins) properties for the structure model (emboli). Results revealed that the maximum shear stress magnitude applied on the embolus was about 957 Pa that was occurred when the clot plow into the wall of the artery. In addition, the average shear stress of the arterial wall was reduced about 42 percent due to the presence of the embolus. This reduction may lead to such phenomena as high pulmonary arterial resistance, low pulmonary arterial compliance, endothelial dysfunction, and consequently cause right heart dysfunction and pulmonary arterial hypertension if different clots repeatedly pass through the arteries.

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1. INTRODUCTION

Emboli mostly is a blood clot resulted from three certain conditions, called Virchow’s triad, including abnormal blood flow (stasis, turbulence), hypercoagulability, and endothelial injury or dysfunction [1]. In 2012, Vahidi and Fatoureae [2] investigated the motion of a blood clot through a stenotic common carotid artery so as to analyze effects of embolus size and stenosis severity on arterial hemodynamics, using Fluid-Structure Interactions (FSI) model. In another study, Abolfazli et al. [3] studied the trajectory of emboli in the carotid bifurcation, using the same approach as Vahidi and Fatoureae, and the effects of size and density of emboli on its movements. Furthermore, other simulations have been performed in the carotid artery [4, 5] and cerebral arteries [6] in which thromboembolic phenomenon is simulated. Khodaee et al. [6] simulated the motion of rigid and deformable emboli with different properties and size through circle of Willis to examine hemodynamics changes. The results revealed that clots with higher rigidity tend to enter to the larger arteries. Regarding literature review, what remains unclear, however, is how the blood clot moves through the pulmonary artery interact with the wall vessel, and affect hemodynamic factors. In the present study, we have utilized computed tomography pictures of a male patient to construct an image-based geometry of pulmonary artery, and simulate hitherto not-investigated Pulmonary Embolism (PE) phenomenon. Using fluid-structure interactions computational algorithm, different material properties for the emboli and a realistic model of pulmonary artery enabled us to track emboli movements through the artery and investigate hemodynamic changes in the presence of blood clot, with implication of a large displacement model. Finally, this study was set out to accomplish this process (thrombus formation in deep veins and traveling toward lungs) which only the beginning of it was conceived in previous studies.

2. METHODOLOGY

2.1. Geometry reconstruction.

Mimics Innovation Suite software (Materialise Mimics Innovation Suite Medical v 19.0), a computer image processor, was utilized to convert Digital Imaging and Communications in Medicine (DICOM) images obtained from the radiological department of Sina hospital, Tehran, Iran to a Three-dimensional (3D) model of the pulmonary artery (shown in Fig. 1).

Fig. 1: Protocol for conducting image-based FSI analysis.
2.2. Fluid-structure interaction.

Governing equations of this phenomenon are mass and momentum conservation. Using a FSI module, the coupled fluid and structure model was solved by ADINA software. Blood was assumed as a Newtonian, laminar, and viscous fluid with 3.48 cP viscosity and 1050 kg/m³ density [7]. Regarding Schmitt et al. [8], embolus density was assumed to be 1080 kg/m³, and rubber/foam material model (Mooney-Rivlin) was employed to simulate elastic behavior of clot. viscoelastic coefficients were derived from the characteristics of elastic and viscous components in the generalized Maxwell model to add viscose of the clot [8]. Also, Output pressure pulse wave of the right heart was applied as the entrance boundary condition and zero normal traction condition as the outlet boundary condition.

3. RESULTS AND DISCUSSION

The embolus released at 0.361 s, and finished its pass along the length of the artery for a duration of 0.1664 s while total number of steps was 3793. The diameter of the main pulmonary artery, in this study, was calculated to be 26.67 mm and was compared with previous study [9]. The average flow rate in main pulmonary artery was estimated at about 8.19 L/min, which was dependent on miscellaneous factors and varies from person to person. In this research, the diameter of the right and left branch was calculated about 3.8 and 7.7 mm, respectively. The results also revealed that clots tend to enter a larger diameter vessel [10]. According to Fig. 2, with decreasing diameter, the amount of shear stress on the wall of artery increased. The shear stress also has elevated on both artery wall and clot with getting the clot closer to the artery wall.

To investigate changes caused by the clot, results were compared to another simulation (Computational Fluid Dynamics (CFD) model of the pulmonary artery) performed without the presence of the clot. The comparison between the results of these two models indicated that the distribution of shear stress in them was significantly different, and it was dropped in the FSI model simulated with emboli (shown in Table 1).

Using the basis of mechanobiology, mechanical behavior of blood and clot was investigated for a better understanding of this phenomenon. Once the clot collided with the artery wall, the maximum stress of 608 Pa was applied to the clot which can increase the risk of clot rupture through the artery and lead to pulmonary infarction. In addition, the presence of emboli has altered the normal distribution of shear stress on the wall of the pulmonary artery which can eventuate in pulmonary hypertension and finally right heart malfunction.

REFERENCES


