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Investigating the Effect of Surface Roughness Modeling on Hemodynamics of Blood Flow in Thoracic Aortic Aneurysm

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Abstract

Thoracic aortic aneurysm (TAA) is a life-threatening vascular pathology whose progression is strongly governed by altered hemodynamic environments. While standard computational fluid dynamics (CFD) frameworks frequently assume idealized smooth endoluminal walls, pathological remodeling, calcification, and endothelial degeneration induce non-negligible wall roughness. This study presents a patient-specific numerical investigation evaluating the coupled influences of surface roughness, flow regimes, and turbulence modeling on blood hemodynamics within an anatomically realistic thoracic aorta reconstructed from CT-scan data. Laminar and two-equation turbulence formulations (standard $k-\epsilon$ and $k-\omega$) were solved across smooth and roughened boundaries ($\epsilon = 0, 50$, and $100 \mu\text{m}$). Critical biomechanical indices, including time-averaged wall shear stress (TAWSS), oscillatory shear index (OSI), relative residence time (RRT), pressure drop, and vorticity dynamics, were evaluated over pulsatile cardiac cycles. The results reveal that wall roughness markedly attenuates wall shear stress and contracts areas exposed to elevated TAWSS, while extending zones characterized by high RRT. Elevated OSI remained predominantly localized along the aortic arch curvature and exhibited negligible sensitivity to micro-scale roughness. These quantitative findings demonstrate that omitting luminal roughness in clinical CFD analyses underestimates critical hemodynamic risk factors associated with endothelial dysfunction and thrombotic susceptibility.

Keywords

Thoracic aortic aneurysm, Surface roughness, Hemodynamics, Wall shear stress, Turbulence modeling, Relative residence time.

1. Introduction

Thoracic aortic aneurysms (TAAs) represent abnormal localized expansions of the aorta that pose severe clinical hazards, including vessel dissection and fatal rupture [1-3]. The underlying pathogenic mechanisms driving aneurysm expansion and intramural thrombus formation are intimately linked to altered local hemodynamic stresses, particularly wall shear stress (WSS) and reciprocal flow patterns [4]. In contemporary computational hemodynamic investigations, endoluminal arterial walls are conventionally modeled as rigid and hydraulically smooth boundaries [3]. However, physiological aging, atherogenesis, endoluminal intimal tear, and advanced calcification introduce structural wall irregularities [5]. Surface roughness significantly alters boundary-layer development, momentum transport, near-wall velocity gradients, and turbulent kinetic energy dissipation [6,7]. Although fluid-structure interactions and non-Newtonian blood rheology have been extensively studied, systematic investigations detailing the interactions between surface roughness and turbulence modeling in realistic thoracic aneurysms remain scarce.

The objective of this research is to numerically simulate pulsatile blood flow within a three-dimensional, patient-specific thoracic aortic aneurysm to quantify the direct influences of wall roughness height and flow modeling approaches (laminar, standard $k-\epsilon$, and standard $k-\omega$) on key hemodynamic indicators, providing refined biomechanical criteria for vascular disease assessment.

2. Methodology

The computational domain was derived from computed tomography (CT) angiographic images of a 48-year-old male patient diagnosed with a descending thoracic aortic aneurysm. Image segmentation and anatomical thresholding were conducted using Mimics software, followed by surface smoothing and solid CAD reconstruction in SolidWorks. Straight extension segments were extruded perpendicular to all branch outlets to guarantee fully developed outflow profiles and

eliminate non-physical recirculation at boundary planes. Unstructured tetrahedral meshes with localized prismatic boundary layers were generated in ANSYS ICEM CFD. A grid independence study was performed across five mesh resolutions containing 175,066, 375,330, 519,071, 722,402, and 1,026,030 elements. Comparative analysis of velocity and wall shear stress metrics showed that grid refinement beyond 519,071 elements yielded variations under 1.5%. Consequently, the grid comprising 519,071 elements was selected to ensure high numerical accuracy with balanced computational cost.

Blood was treated as an incompressible, Newtonian fluid with a dynamic viscosity of $\mu=0.0035$ Pa·s and density of $\rho=1050$ kg/m³.

The continuity and momentum transport equations for incompressible flow are formulated as:

$$\nabla \cdot \mathbf{u} = 0 \quad (1)$$

$$\rho(\partial \mathbf{u} / \partial t + \mathbf{u} \cdot \nabla \mathbf{u}) = -\nabla p + \mu \nabla^2 \mathbf{u} + \nabla \cdot \boldsymbol{\tau}^R \quad (2)$$

Where $\mathbf{u}$ represents fluid velocity, p is static pressure, and $\boldsymbol{\tau}^R$ represents Reynolds stress components closure modeled via the standard k - ϵ and standard k - ω frameworks using modified rough-wall functions. Three endoluminal wall conditions were evaluated: smooth ($\epsilon/D=0\%$), moderate roughness ($\epsilon=50$ μm , relative roughness $\epsilon/D=0.25\%$), and severe roughness ($\epsilon=100$ μm , relative roughness $\epsilon/D=0.50\%$), based on the average vessel diameter ($D=21$ mm).

Pulsatile physiological waveforms corresponding to a normal resting heart rate (60 bpm; cardiac cycle $T=1.0$ s) were applied at the ascending aorta inlet, while physiological pressure wave profiles were imposed at the supra-aortic branches and descending aortic outlet. A fixed time step of $\Delta t=0.001$ s was utilized in ANSYS Fluent. Simulations were computed over four consecutive cycles, and data extraction was conducted exclusively from the fourth cardiac cycle after confirming periodicity.

To quantitatively evaluate pathobiological risks, three hemodynamic parameters were computed:

1. Time-Averaged Wall Shear Stress (TAWSS):

$$\text{TAWSS} = \frac{1}{T} \int_0^T |\overrightarrow{\tau_w}| dt \quad (3)$$

2. Oscillatory Shear Index (OSI):

$$\text{OSI} = \frac{1}{2} \left(1 - \frac{\left| \int_0^T \overrightarrow{\tau_w} dt \right|}{\int_0^T |\overrightarrow{\tau_w}| dt} \right) \quad (4)$$

3. Relative Residence Time (RRT):

$$\text{RRT} = \frac{1}{(1 - 2 \times \text{OSI}) \times \text{TAWSS}} \quad (5)$$

3. Results and Discussion

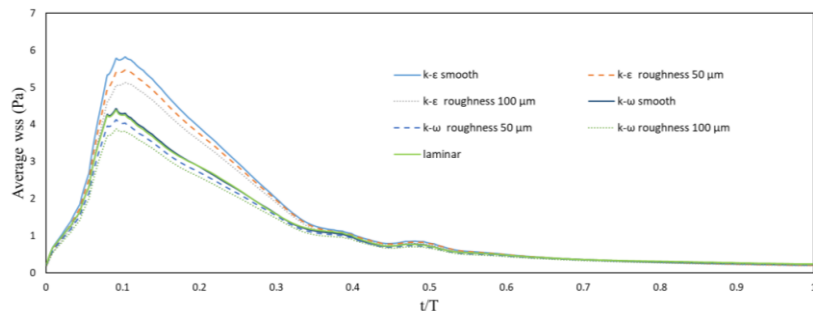


Figure 1. Variations in surface-averaged wall shear stress across a complete cardiac cycle for laminar and turbulent models with different wall roughness heights

3.1. Wall Shear Stress and Turbulence Model Variations

The temporal evolution of the surface-averaged wall shear stress closely tracked the systolic–diastolic cardiac

waveform. At the peak systolic phase ($t/T=0.12$), the standard $k-\epsilon$ model predicted the highest mean wall shear stress, exceeding laminar predictions by 31.3% and $k-\omega$ predictions by 30.0% (Figure 1). Furthermore, incorporating luminal wall roughness led to a noticeable reduction in the mean wall shear stress throughout the cardiac cycle; for turbulent models, the smooth wall exhibited approximately 7% and 14% higher mean wall shear stress compared to the 50 μm and 100 μm roughness cases, respectively.

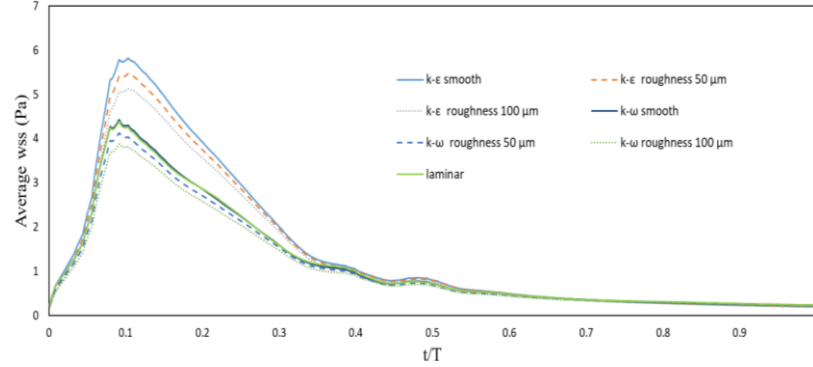


Figure 1. Variations in surface-averaged wall shear stress across a complete cardiac cycle for laminar and turbulent models with different wall roughness heights

3.2. Spatial Distributions of TAWSS, OSI, and RRT

The spatial distribution of TAWSS revealed consistently low magnitudes (<0.4 Pa) inside the aneurysm sac, while elevated values were recorded near branching bifurcations and tortuous segments of the aortic arch (Figure 2). Increasing surface roughness systematically contracted the luminal surface area exposed to high TAWSS.

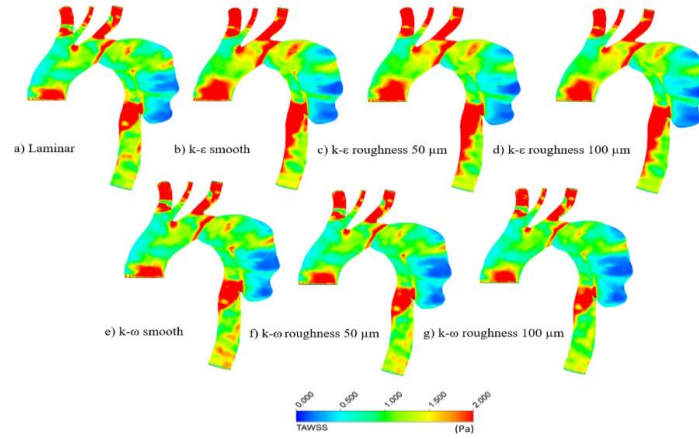


Figure 2. Spatial contours of time-averaged wall shear stress (TAWSS) across a cardiac cycle for smooth and rough vessel models.

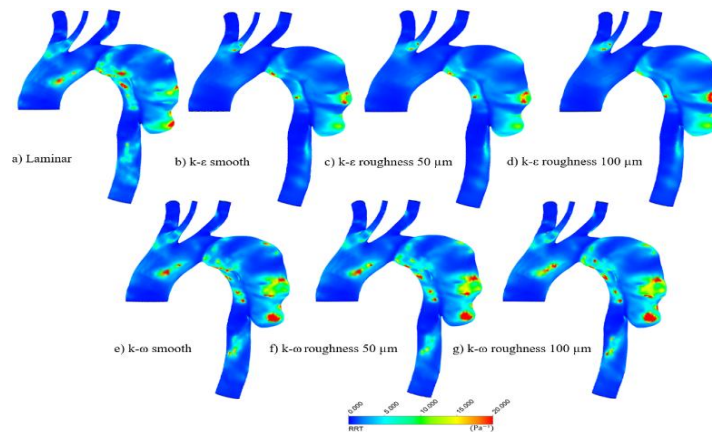


Figure 3. Spatial distributions of relative residence time (RRT) across a cardiac cycle for laminar and turbulent models with varying surface roughness.

Oscillatory patterns, measured via OSI, were elevated (>0.2) along the inner curve of the aortic arch and localized zones within the aneurysm sac. OSI magnitude proved notably insensitive to wall roughness variations, showing minor surface fraction modifications. Conversely, RRT displayed substantial extensions in regions subjected to micro-roughness (Figure 3). High RRT values were concentrated within the posterior and terminal regions of the aneurysm bulge. Because elevated RRT combined with low TAWSS fosters atherogenesis, platelet deposition, and thrombus growth, these distal aneurysm domains were identified as sites of severe pathological vulnerability.

3.3. Intravascular Pressure Drop and Vorticity Dynamics

Evaluation of pressure drops between the inlet and the thoracic outlet indicated that laminar simulations yielded lower overall pressure drops ($\Delta P_{\text{cycle}} = 137$ Pa) than turbulent models (Table 1). For the standard $k-\epsilon$ model, net cycle-averaged pressure drops were 175 Pa, 169 Pa, and 157 Pa for smooth, 50 μm , and 100 μm roughness heights, respectively. Similarly, the $k-\omega$ formulation yielded drops of 143 Pa, 141 Pa, and 140 Pa. This moderate reduction in net pressure loss with increasing roughness is correlated with a noticeable decrease in core bulk vorticity and secondary flow kinetic dissipation, which concurrently elevated the mean intravascular static wall pressure.

Table 1. Intravascular pressure drops between inlet and outlet 1 $t/T=0.08$, $t/T=0.12$ (peak systole), and cycle average.

Time (t/T)	Laminar (Pa)	$k-\epsilon$, smooth (Pa)	$k-\epsilon$, 50 μm roughness (Pa)	$k-\epsilon$, 100 μm roughness (Pa)	$k-\omega$, smooth (Pa)	$k-\omega$, 50 μm roughness (Pa)	$k-\omega$, 100 μm roughness (Pa)
0.08	2880	3022	2982	2944	2882	2854	2830
0.12	375	561	527	492	382	368	354
Cycle average	137	175	169	157	143	141	140

4. Conclusions

This study evaluated the quantitative impact of endoluminal surface roughness coupled with turbulence modeling in a patient-specific thoracic aortic aneurysm. The primary conclusions are summarized as follows:

1. Endoluminal surface roughness significantly alters near-wall hemodynamics by decreasing the vessel surface area exposed to high TAWSS and increasing the surface area subject to elevated RRT.
2. The oscillatory shear index (OSI) is predominantly governed by global macro-geometry and vessel curvature (concentrated at the aortic arch) rather than micro-scale surface roughness.
3. The terminal sac and inner curvature of the aortic arch exhibit simultaneous low TAWSS and high RRT, indicating heightened susceptibility to atherosclerotic plaque aggregation and thrombus formation.
4. Elevated wall roughness dampens internal vorticity intensity, reducing convective pressure drop across the aorta and yielding a slight increase in mean intravascular pressure.
5. Accounting for realistic luminal roughness in computational hemodynamic studies provides a more conservative and clinically accurate appraisal of thrombotic and rupture risks in aortic aneurysms.

5. References

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