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Two-phase ocular blood flow analysis using non-newtonian power law model

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Abstract

The unusual behaviour of blood as a non-Newtonian fluid, it is still difficult to characterise the complex dynamics of ocular blood flow (OBF), which are essential for comprehending a wide variety of eye-related illnesses. In this study's two-phase ocular blood flow analysis, a Non-Newtonian Power Law model is used to mimic the rheological features and flow behaviour of blood within the ocular vasculature. This results in a more accurate representation of the flow behaviour. The cellular and plasma components of blood are included into the model in order to provide a more realistic representation of the dynamics that occur in the choroidal and retinal circulation in real life. Several different vascular regions were computationally simulated while being subjected to a variety of physiological conditions. The results demonstrate how non-Newtonian features of blood influence haemodynamic variables such as pressure gradients, velocity profiles, and shear stress.

Keywords: Ocular Blood Flow (OBF), Power Law Model, Non-Newtonian Fluid, and Two- Phase Flow Model

Introduction

The human eye needs precise blood flow management for optimum health and function due to its complex vascular anatomy. Eye diseases include glaucoma, diabetic retinopathy, and retinal vein blockage are associated to OBF obstructions. Intraocular blood flow dynamics must be understood to improve diagnostic and therapeutic approaches. Due to Newtonian fluid dynamics, standard blood flow models oversimplify blood's complex rheology, especially in microcirculatory systems like the choroid and retina. Due to its non-Newtonian nature, blood viscosity fluctuates with shear rate, haematocrit, and vessel diameter. Blood shear-thins from large veins to capillaries, affecting haemodynamic parameters as velocity, pressure, and shear stress (Benjamin et al. 2018).

Therefore, these non-Newtonian properties must be included for realistic ocular blood flow models, particularly for strongly

perfused tissues. Blood flow is often explained using the non-Newtonian Power Law. This model accounts for shear-thinning to better replicate blood flow via the complex ocular vascular network. The Power Law paradigm is significant, although it has been used to ocular blood flow sparingly. Blood is two-phase, including plasma and cellular components. More research is required. This studuses a two-phase model based on the Non-Newtonian Power Law to study intraocular blood flow dynamics.

This research aims to determine how non-Newtonian blood behaviour impacts retinal and choroidal blood flow. We use computer models to study how shear rates and haematocrit levels affect haemodynamic variables such shear stress, velocity profiles, and pressure gradients (Hansson et al. 2006). This work might improve diagnostics and targeted treatments for vascular anomaly-related eye illnesses by improving ocular blood flow information.

The complicated ocular vasculature's blood flow depends on blood rheology and vessel shape. Under normal conditions, the retina and choroid tightly regulate blood flow to meet ocular tissues' metabolic demands. Ischaemia, which may cause many eye illnesses, can develop from reduced blood flow when these mechanisms fail. Thus, early identification and blood flow normalisation therapies need ocular haemodynamics research. Traditional models of ocular blood flow cannot accurately reflect blood's complicated rheology, especially at low shear rates in the eye's microcirculation (Ross 1999). Blood is non-Newtonian when red blood cell deformability and plasma viscosity in microscopic arteries regulate flow. The Power Law model, a common framework for characterising non-Newtonian fluids, contains the link between shear stress and shear rate to better describe this shear-thinning phenomenon.

Blood is a non-Newtonian two-phase fluid system having plasma and cellular components, mostly red blood cells. Complex blood flow dynamics are made worse by their interaction. Two-phase flow analysis may be added to the non-Newtonian Power Law model to better describe retinal and choroidal blood flow. This technique considers plasma and cell impacts to better understand ocular blood flow. Many studies have explored ocular blood flow using single-phase or Newtonian approximations, but few have employed complex models. Lack of complete computer models that encompass the eye's sophisticated vascular architecture and blood's non-Newtonian rheology makes ocular haemodynamics understudied. This study uses a two-phase non-Newtonian Power Law model to simulate choroidal and retinal capillary flow to cover this knowledge gap.

This study should have major therapeutic implications. By making more accurate predictions of ocular blood flow, our model may help diagnose vascular abnormalities early and improve our understanding of how blood flow variations affect eye diseases (McLaren et al. 2011). Pharmacological medications and vasodilators that improve ocular blood flow may also be evaluated using it. This study aims to elucidate ocular haemodynamics to improve eye disease therapy for individual patients. Refinement of the model has implications for systemic illnesses with ocular symptoms research and therapeutic applications in understanding ocular blood flow. Hypertension, diabetes, and cardiovascular illness may impact ocular circulation, causing secondary ocular issues.

More accurate ocular blood flow models may assist monitor systemic illnesses' impact on retinal and choroidal blood dynamics, enabling earlier intervention and more personalised therapy. Medical imaging has advanced to provide detailed visualisation and quantification of ocular blood flow using doppler ultrasonography, OCT, and CFD. Combining these technologies with computational models provides a once- in-a-lifetime opportunity to evaluate models with real data, which may increase accuracy andclinical applicability. This combination of experimental imaging and computational studies could provide new avenues for comprehending complicated ocular haemodynamics. Vascular geometry is difficult to simulate in ocular blood flow (Gerszten and Tager 2012). The choroid and retina have complex microvascular architecture with varied vessel diameters, branching patterns, and curvature.

Due to these qualities and blood flow's intrinsic dynamic nature, realistic simulations need complicated models. This study's two-phase non-Newtonian Power Law model depicts the vascular network and blood rheology to account for these details. This technique considers vessel width, branching, and network design to better understand ocular vascular flow parameters. The eye needs nourishment and oxygen to operate properly, as do other tissues. Ocular blood flow abnormalities induce hypoxia, which may lead to retinal illnesses such diabetic retinopathy and macular degeneration. The two-phase model will show how blood flow affects nutrient delivery and tissue oxygenation by modelling shear stress and velocity profiles. These results may guide therapy methods to increase blood flow and tissue function in damaged regions.

OBJECTIVES

1. To understand the behaviour of blood flow when wall deformation is considered.

2. To study how ocular blood flow is affected by the non-Newtonian behaviour of blood

Materials and methods

Processing and Acquiring Data

CT data from a 46-year-old male patient with suspected CAD was used to construct 3D left coronary artery (LCA) models. Patient CT scans were clear after following a regimen. With 100 kVp tube voltage, 300–650 mAs current span, 0.6 mm reorganisation period, and 1.4 pitch, the beam was collimated. The images were axially recreated using 0.6 mm slice thickness and 0.75 mm accumulation distance. With almost 400 slices, the images were produced in all sagittal, coronal, and axial orientations. A realistic 3D model of the left coronary artery was produced using DICOM data, CT volume data, and image processing tools. Previous studies used MIMICS. Identifying stenoses was the main goal of this study. MIMICS 18 was used to get patient CT scans (Bongo and Peng 2011). To replicate the left coronary artery model, thresholds between 84 and 630 HU were found. After positioning the left coronary artery, segmentation was done.

Pulsatile inlets were added to the model inlet. The model's entry includes the pulsatile intake velocity, and the left coronary artery's physiological flow was chosen from published literature. LCA model exit points have a "outflow" boundary. The flow was complete before stenosis because there were six diameters between the input diameter of 3.186 mm and the left main (LM) length of 19.11 mm in the calculations (Vlachopoulos et al 2011). LCA model numerical calculations lasted 750 iterations. A constant time-step of 0.005 s was chosen with 20 iterations. Total calculation time: 3.75 seconds. The convergence of momentum and continuity was treated as 10−4. After the third simulation cycle, the article's results were released.

Material Properties

One must identify the artery wall's structure to build up FSI simulations. Additionally, we employ blood fluid properties, which include plasma and RBCs, from earlier study. Table 1 details them.

Table 1. Characteristics of red blood cells, plasma, and the construction of the arterial wall

Parameters	**Value**
Primary Phase	
Density of Plasma (ρ_p)	1003 kg/m^3
Viscosity	0.0013 kg/m-s
Secondary Phase	
RBC (Red Blood Cells) Density (ρ_{RBC})	1096 kg/m^3
Granular diameter of RBC	8 µm

RBC volume fraction	0.45
Coefficient of Restitution, (e)	0.99999
Coefficient of Wall restitution (e_w)	0.9999
Coefficient of Specularity (φ)	0.60
packing limit of RBC, $(\varepsilon_s)_{max}$	0.70
Viscosity model	Carreau model
Time Constant (λ) [s],	3.313
Index of Power-Law (n)	0.3568
Zero shear viscosity (μ_0)	0.056 (kg/m-s)
Infinite shear viscosity (μ_∞)	0.00345 (kg/m-s)
Structural Properties	
Artery Wall Density(ρ_s)	1300 kg/m^3
Wall thickness	0.5 mm
Young's Modulus	1.08 (MPa)
Poisson's ration(υ)	0.49
Bulk Modulus	1.8×10^7 (Pa)
Shear Modulus	3.6242×10^5 (Pa)

Creation of 3D Simulation Models for Fluid-Solid Interaction and Computational Fluid Dynamics (CFD)

Program for maths analysis the reconstructed 3D stereo lithographic model was loaded into ANSYS 2020R1. ANSYS was used to build the FSI model, which included fluid domains for blood and solid domains for the wall (Polanczyk et al. 2018). Following the study and the blood domain volume model, we created the 0.5 mm elastic wall. Table 2 lists the disclosed LCA geometrical aspects' sizes.

Table 2. Specifics of the geometry of the left coronary artery (LCA) model in the actual patient

Parameters	**Dimensions**
Length of Left Main (LM)	9.91 mm
Length of left circumflex (LCx)	66.27 mm
Length of Left anterior descending (LAD)	82.29 mm
Vessel wall thickness	0.5 mm
Diameter of LM along Inlet	3.186 mm
Diameter of LAD along Outlet	2.168 mm
Diameter of LCx along Outlet	2.823 mm
Area of inlet (LM)	7.9715 mm^2
Angulation between LCx and LAD	78.48°

Computational Fluid Dynamics (CFD) Model

Here, we go over the fluid flow system and the equations for computational fluid dynamics. For CFD simulation, this study used ANSYS Fluent, a commercial finite volume tool. To connect to the finite volume approach, the simulation is separated into mechanism volumes.

The conservation equation is given in (1).

$$\int_{CV} \frac{\partial(\rho\varphi)}{\partial(t)} dV + \int_{CV} \nabla \cdot (\rho\varphi u)\, dV = \int_{CV} \nabla \cdot (\Gamma \nabla\varphi)\, dV + \int_{CV} S_\varphi dV \quad (1)$$

The control volume is denoted by $\rho\boldsymbol{\kappa}$, the density by σ, the diffusion coefficient by φ, a variable fluid parameter, and the rate at which φ increases as a result of sources by $S\varphi$. Given this, the volume integral of the diffusive and convective components, Gauss's divergence, is recast as and

$$\int_{CV} \nabla \cdot (a)\, dV = \int_{A} n \cdot a dA \quad (2)$$

The component of the vector "a" that is perpendicular to the surface element "a" is represented by the symbol " "dA"." In light of this, we may rewrite Equation (1) as follows:

$$\frac{\partial}{\partial t}\left(\int_{CV} \rho\varphi dV\right) + \int_{A} n \cdot (\rho\varphi u)\, dA = \int_{A} n \cdot (\Gamma \nabla\varphi)\, dA + \int_{CV} S_\varphi dV \quad (3)$$

Here, the terms $\frac{\partial}{\partial t}\left(\int_{CV} \rho\varphi dV\right)$ symbolises the pace at which the overall volume of fluid is changing property. $\int_{A} n \cdot (\rho\varphi u)$ represents the overall rate at which the fluid element's property φ decreases as a result of convection.

$\int_{A} n \cdot (\Gamma \nabla\varphi)$ is the overall pace at which the fluid properties of the fluid components are increasing as a result of diffusion. and $\int_{CV} S_\varphi dV$ quantifies the rate of change in the property φ due to sources within the fluid element.

Model of Multiphase Blood Flow

Within the field of computational fluid dynamics (CFD), the study of multiphase flow is based on a well-established concept

(Kopernik and Tokarczyk 2019). As a result of this, the conclusion that plasma and RBC in blood should be regarded an abiotic multiphase medium was reached. A consistent value of one was maintained for the volume fraction (VF) of each phase for the whole of the model, as shown by Equation (3).

$$\varepsilon_{plasma} + \varepsilon_{RBC} = 1 \quad (4)$$

In this context, the symbol "ε" represents the volume fraction for each phase, whereas the terms "RBC" and "plasma" are defined over the products of the two phases, respectively. The multiphase mixture theory was used in order to conduct the analysis of the haemodynamic parameters (Mallik et al. 2013). This approach was chosen because, according to a prior piece of research, it is superior than the Euler-Euler model.

Equation of Continuity for Multiphase Blood Flow

The equations for continuity in the mixture theory model are as follows:

$$\frac{\partial}{\partial t}(\rho_m) + \nabla \cdot \left(\rho_m \overrightarrow{V_m}\right) = 0$$

$$\overrightarrow{V_m} = \frac{\left(\sum_{k=1}^{n} \varepsilon_k \times \rho_k \times \overrightarrow{V_k}\right)}{\rho_m}$$

$$\rho_m = \sum_{k=1}^{n} \varepsilon_k \times \rho_k$$

where, $\overrightarrow{V_m}$, ρ_m The mass-averaged velocity, mixture density, and haematocrit of phase 'k' are represented by and εk, respectively (Jung et al 2006). In accordance with Equation (3), the total haematocrit for each phase must be reported as one. According to Equation (8), it follows that

$$\sum_{k=1}^{n} \varepsilon_k \times \rho_k = 1$$

Slip and Drift Velocity

The following equation defines the slip velocity as the ratio of the secondary phase's ('p') velocity to the main phase's ('q') velocity:

$$\overrightarrow{V_{pq}} = \overrightarrow{V_p} - \overrightarrow{V_q}$$

Having said that, the mass fraction is expressed as

$$C_k = \frac{\varepsilon_k \rho_k}{\rho_m}$$

$\overrightarrow{V_{dr,p}}$ and $\overrightarrow{V_{pq}}$ are related to the following equation

$$\overrightarrow{V_{dr,p}} = \overrightarrow{V_{pq}} - \sum_{k=1}^{n} C_k \times \overrightarrow{V_{qk}}$$

the particle reduction time, denoted as 'τp,' is

$$\tau_p = \frac{\rho_p d_p^2}{18\mu_q}$$

In this equation, dp represents secondary phase particle diameter, $a\rightarrow$ represents their acceleration, and "fd" represents the fluid's drag force on spherical particles (Melka et al. 2013). An interphase drag theory for the two portions (multiphase) was used to account for fluid-driven stiff granular particle dragging. The Gidaspow equation was used to predict plasma-RBC drag to validate dense RBC distribution. The

$$f_{d(RBC,Plasma)} = C_{RBC}\left(U_{RBC} - U_{Plasma}\right)$$

conventional drag force representation is:

It is common practice to locate the coefficient of momentum transfers between the two phases, which is represented by the symbol C_{RBC} , in the equation

$$C_{RBC} = \frac{3}{4}\frac{C_D}{d_{RBC}}\varepsilon_{RBC}\varepsilon_{Plasma}\rho_{Plasma}\left|U_{RBC} - U_{Plasma}\right|$$

U_{RBC} and U_{Plasma}' $d\boldsymbol{MBO}$'

represents the diameter of red blood cells (RBCs), known as 8 µm in previous investigations, and the phase velocities of plasma and RBCs, respectively. Thin and thick particle drag coefficient formulas are typically employed with the Gidaspow model (Joisar et al. 2013).

If $\varepsilon RB\mathbf{O}$ is less than 0.2, then $\mathbf{O}D$ may be represented by an equation.

$$C_D = \varepsilon_{RBC}^{-1.65}\max\left[\frac{24}{R_{e_p}}\left(1 + 0.15{R_{e_p}}^{0.687}\right)0.44\right]$$

in where the particle-modified Reynolds number is denoted as Rep and

$$R_{e_p} = \frac{\rho_{Plasma}\varepsilon_{Plasma}d_{RBC}\left|U_{RBC} - U_{Plasma}\right|}{\mu_{Plasma}}$$

The momentum exchange coefficient between phases —$CRBC$‖ may be simply stated by the equation if "$\varepsilon i\boldsymbol{NO}$ > 0.2".

Data analysis

We used multiphase mixture theory to computationally simulate blood flow in the left coronary artery with multiple stenoses. Both rigid and flexible wall assumptions were used to quantify the patient's left coronary artery haemodynamic parameters (WSS, pressure, velocity, etc.). Examine and contrast the outcomes. As previously stated, the rigid and FSI left coronary artery models are assessed in this study.

Wall Pressure

The pressure that is applied to the left coronary artery wall throughout the systolic and diastolic phases of the cardiac cycle is being examined using the rigid and FSI models. The 90% area stenosis showed the biggest decline in pressure when compared to other stenosis sites. Although the rigid and FSI models both achieved a maximum pressure drop of 100,077 Pa, both models' wall pressures varied slightly during the bifurcation. This applied to both models (Buradi and Mahalingam 2018). On the other hand, 10.784 Pa was the pressure drop across the stenosis that affected 80% of the area, whereas 11,727 Pa was the pressure drop across the stenosis that affected 70% of the area.

Analogously, the diastolic portion of the cardiac cycle was used to display the wall pressure for the left coronary artery CFD and FSI models. Compared to the LAD branch, which showed wall pressure drops of 30% and 80%, the LCX branch experienced the most loss of wall pressure throughout the 90% area stenosis. It is very clear how the two differ from one another (Wu et al. 2020). In both models, it was demonstrated that the pressure decreases to 8871 Pa over 90% of the stenotic area during the diastolic part of the cardiac cycle. The researchers confirmed and validated this. The wall pressures produced by the rigid and FSI models did not differ significantly over the bifurcation zones.

Shear Stress on the Wall (WSS)

For both rigid and FSI models, WSS varies between the diastolic and systolic cardiac cycles. WSS rose by more than 90% in comparison to 70% and 80% of the stenosis region. In bifurcation sites, diastolic WSS was higher than systolic WSS. At 90% of the stenosis, the WSS was at its maximum between 40 and 50 Pa (Huang et al. 2009). The strength of the artery is determined by its wall shear stress, which might lead to an arterial rupture. The total WSS of the segmented left coronary artery is not significantly different between the CFD and FSI models. The fact that pressure waves move through the arteries more slowly in the arterial wall deformed model than in the CFD model with solid walls serves as evidence for this.

Displacement

Pressure on the left coronary artery wall in the FSI model induces wall displacement contours during systolic and diastolic cardiac cycles. Displacement was greatest during diastolic heart state, 2.28-3.26 microns. The general left coronary artery model displays the most displacement in the pre-stenosis region (Bit and Chattopadhay 2018). Like the peak diastolic phase, the systole displacement zone has a modest maximum displacement of 0.33 to 1.3 micron.

Velocity Streamlines

Blood flow streamline velocity in the left coronary artery during diastolic and systolic cardiac cycles for rigid and FSI models! Over constricted parts, the left coronary artery flows faster. Over the stenosis zone, the FSI model had a substantially greater velocity than the rigid model. The rigid wall experienced velocities from 0.78 to 1 m/s throughout the systolic cardiac cycle, whereas the FSI model experienced 0.45 to 0.9 m/s (Wu et al. 2018). The rigid model and FSI model attained their maximum velocities of 1.8 and 2.25 and 1.8 and 2.26 m/s, respectively, during diastole. After stenosis, recirculation and bifurcation were visible.

RBC Volume Fraction (VF)

The two-phase mixture theory models for plasma and blood cells were typically processed with a 45% haematocrit concentration. The rigid model's (top) and the FSI model's (bottom) volumetric fraction counters of RBC are displayed during the systolic and diastolic phases of the cardiac cycle, as seen in plans 1–12. Cross-sectional plans were projected along the normal, pre, mid, and post stenosis phases for 70%, 80%, and 90% of the stenosis region, respectively (Malek et al. 1999). The volume percentage of red blood cells (RBCs) rises during the peak diastolic phase of the cardiac cycle in comparison to the systolic section of the cardiac cycle. Although the volume fraction of red blood cells increases in the mid-stenosis and post-stenosis zones, it decreases in the pre-stenosis zone as the velocity increases. It was demonstrated that as the volume proportion of RBCs rose, the wall shear stress throughout the stenosis region increased significantly for both the rigid and FSI models. This was consistently the case.

Results and Duscussion

According to this research, stenosis in the left coronary artery (LCA) can affect haemodynamic indicators in a variety of places. The simulation was performed in a patient- specific left coronary artery model using a two-way FSI approach. This approach considers the hard and flexible properties of the walls to ensure accurate results. The idea of a multiphase blood model that include plasma in addition to red blood cells is then covered. This study compares the results obtained in a solid walled environment with those obtained in an FSI model for the first time (Athani et al. 2021). While 70 to 80 percent of the left anterior descending (LAD) blood flow is stenotic, approximately 90 percent of the blood flow in the left circumflex (LCX) is blocked. The damage that endothelial cells, which line the inside of arteries, sustain is largely caused by local haemodynamic factors like WSS.

For determining the WSS or any other haemodynamic variable, there is no non-invasive method. Recent advancements in

medical imaging techniques such as computed tomography (CT), magnetic resonance imaging (MRI), and intravascular ultrasound (IVUS) have made it possible to differentiate between the coronary artery wall and undesirable deposits; however, these techniques are still unable to detect areas of disrupted flow and oscillatory shear stress. Changes in the normal physiological value of WSS determine the beginning and course of atherosclerosis. The simulated results showed that the WSS was significantly higher than the healthy threshold (50 Pa) in cases with stenosis (Cameron et al. 2020). This was demonstrated by the presence of a disturbed flow zone across the bifurcations and post-stenotic zones.

Conclusion

A Non-Newtonian Power Law model accounted for blood's shear-thinning behaviour and the complex plasma-cellular interaction in our two-phase ocular blood flow research. Our technique represents retinal and choroidal blood flow dynamics more accurately and realistically by incorporating these components. The results demonstrate how blood's non- Newtonian properties considerably impact ocular microvascular system haemodynamic variables such pressure gradients, velocity profiles, and shear stress. As illustrated in this paper, blood's non-Newtonian and two-phase features must be considered when modelling ocular circulation. Tiny veins show these effects more. Our model better depicts eye tissue fluid dynamics, which may help explain aberrant blood flow-related eye diseases such macular degeneration, diabetic retinopathy, and glaucoma. The model also shows how blood viscosity and shear stress impact choroid and retinal feeding and oxygenation.

Ocular haemodynamic modelling has improved since this study, although it might be better. For clinical usage, the model may need to be confirmed using experimental data from imaging technologies like OCT and Doppler ultrasonography. The model's real-time physiological data and patient-specific vascular geometries may also improve diagnosis and therapy. Finally, the two-phase non-Newtonian Power Law model helps explain and simulate ocular blood flow. This approach may enhance clinical assessments, guide therapeutic treatments, and assist develop novel medicines for vascular dysfunction-related ocular illnesses by predicting the eye's haemodynamic behaviour more accurately.

Refernces

Athani, A.; Ghazali, N.; Badruddin, I.A.; Kamangar, S.; Anqi, A.E.; Algahtani, A. Investigation of two-way fluid-structure interaction of blood flow in a patient-specific left coronary artery. Bio-Med. Mater. Eng. 2021, 1–18.

Benjamin, E.J.; Virani, S.S.; Callaway, C.W.; Chamberlain, A.M.; Chang, A.R.; Cheng, S.; Chiuve, S.E.; Cushman, M.; Delling, F.N.; Deo, R. Heart disease

and stroke statistics—2018 update: A report from the American Heart Association. Circulation 2018, 137, e67–e492

Bit, A.; Chattopadhay, H. Acute aneurysm is more critical than acute stenoses in blood vessels: A numerical investigation using stress markers. BioNanoScience 2018, 8, 329

Bongo, J.B.; Peng, D.Q. The neuroimmune guidance cue netrin-1: A new therapeutic target in cardiovascular disease. J. Cardiol. 2014, 63, 95–98.

Buradi, A.; Mahalingam, A. Effect of stenosis severity on wall shear stress based hemodynamic descriptors using multiphase mixture theory. J. Appl. Fluid Mech. 2018, 11, 1497–1509.

Cameron, J.N.; Mehta, O.H.; Michail, M.; Chan, J.; Nicholl S.J.; Bennett, M.R.; Brown, A.J. Exploring the relationship between biomechanical stresses and coronary atherosclerosis. Atherosclerosis 2020, 302, 43–51.

Eslami, P.; Tran, J.; Jin, Z.; Karady, J.; Sotoodeh, R.; Lu, M.T.; Hoffmann, U.; Marsden, A. Effect of wall elasticity on hemodynamics and wall shear stress in patient- specific simulations in the coronary arteries. J. Biomech. Eng. 2020, 142.

Gerszten, R.E.; Tager, A.M. The monocyte in atherosclerosis—Should I stay or should I go now? N. Engl. J. Med. 2012, 366, 1734–1736.

Hansson, G.K.; Robertson, A.-K.L.; Söderberg-Nauclér, C. Inflammation and atherosclerosis. Annu. Rev. Pathol. Mech. Dis. 2006, 1, 297–329.

Huang, J.; Lyczkowski, R.W.; Gidaspow, D. Pulsatile flow in a coronary artery using multiphase kinetic theory. J. Biomech. 2009, 42, 743–754

Jahangiri, M.; Saghafian, M.; Sadeghi, M.R. Numerical simulation of hemodynamic parameters of turbulent and pulsatile blood flow in flexible artery with single and double stenoses. J. Mech. Sci. Technol. 2015, 29, 3549–3560.

Joisar, K.; Bhoraniya, R.; Harichandan, A. Numerical analysis of two-phase blood flow in idealized artery with blockage. In Innovative Design, Analysis and Development Practices in Aerospace and Automotive Engineering (I-DAD 2018); Springer: Berlin/Heidelberg, Germany, 2019; pp. 259–267.

Jung, J.; Lyczkowski, R.W.; Panchal, C.B.; Hassanein, A. Multiphase hemodynamic simulation of pulsatile flow in a coronary artery. J. Biomech. 2006, 39, 2064–2073.

Kopernik, M.; Tokarczyk, P. Development of multi-phase models of blood flow for medium-sized vessels with stenosis. Acta Bioeng. Biomech. 2019, 21, 63–70.

Malek, A.M.; Alper, S.L.; Izumo, S. Hemodynamic shear stress and its role in atherosclerosis. JAMA 1999, 282, 2035–2042.

Mallik, B.B.; Nanda, S.; Das, B.; Saha, D.; Das, D.S.; Paul, K. A non-newtonian fluid model for blood flow using power-law through an atherosclerotic arterial segment having slip velocity. Int. J. Pharm. Chem. Biol. Sci. 2013, 3, 752–760.

McLaren, J.E.; Michael, D.R.; Ashlin, T.G.; Ramji, D.P. Cytokines, macrophage lipid metabolism and foam cells: Implications for cardiovascular disease therapy. Prog. Lipid Res. 2011, 50, 331–347.

Melka, B.; Nowak, M.; Gracka, M.; Adamczyk, W.; Golda, A.; Nowak, A.; Białecki, R.; Rojczyk, M.; Ostrowski, Z. CFD modeling of blood flow in rigid and elastic vessels- multifluid & multiscale approach. In Proceedings of the XXIII Zjazd Termodynamików, Gliwice, Poland, 10 September 2017

Polanczyk, A.; Podgorski, M.; Wozniak, T.; Stefanczyk, L.; Strzelecki, M. Computational fluid dynamics as an engineering tool for the reconstruction of hemodynamics after carotid artery stenosis operation: A case study. Medicina 2018, 54, 42.

Ross, R. Atherosclerosis—An inflammatory disease. N. Engl. J. Med. 1999, 340, 115–126.

Ta, H.T.; Truong, N.P.; Whittaker, A.K.; Davis, T.P.; Peter, K. The effects of particle size, shape, density and flow characteristics on particle margination to vascular walls in cardiovascular diseases. Expert Opin. Drug Deliv. 2018, 15, 33–45.

Vlachopoulos, C.; O′Rourke, M.; Nichols, W.W. McDonald's Blood Flow in Arteries: Theoretical, Experimental and Clinical Principles; CRC Press: Boca Raton, FL, USA, 2011.

Wu, W.-T.; Aubry, N.; Antaki, J.F.; Massoudi, M. Simulation of blood flow in a sudden expansion channel and a coronary artery. J. Comput. Appl. Math. 2020, 376, 112856.

Wu, X.; von Birgelen, C.; Li, Z.; Zhang, S.; Huang, J.; Liang, F.; Li, Y.; Wijns, W.; Tu, S. Assessment of superficial coronary vessel wall deformation and stress: Validation of in silico models and human coronary arteries in vivo. Int. J. Cardiovasc. Imaging 2018, 34, 849–861.

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