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Article

Biomechanical impact of hyperlipidemia on blood flow and vascular pressure: A computational and in vivo analysis

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Abstract: Hyperlipidemia is a major contributor to cardiovascular diseases, impacting vascular function by altering hemodynamic parameters such as blood flow velocity and vascular pressure. This study investigated these effects using Doppler ultrasound, vascular pressure sensors, and computational fluid dynamics (CFD) modeling in a cohort of 100 participants, divided into 50 hyperlipidemic individuals (total cholesterol: 250.4 ± 15.2 mg/dL, triglycerides: 180.3 ± 20.5 mg/dL, low-density lipoprotein (LDL) cholesterol: 170.1 ± 10.3 mg/dL) and 50 controls (total cholesterol: 190.7 ± 10.4 mg/dL, triglycerides: 130.2 ± 15.7 mg/dL, LDL cholesterol: 120.5 ± 9.8 mg/dL). Future research could explore the differential effects of LDL and HDL on blood flow velocity and vascular pressure, as LDL is known to increase blood viscosity and contribute to endothelial dysfunction, while HDL may have a protective effect by improving endothelial function and reducing vascular resistance. Results showed a significant reduction in blood flow velocity in the hyperlipidemic group (28.1 ± 3.1 cm/s) compared to controls (32.5 ± 2.3 cm/s, $p < 0.001$) and an increase in vascular pressure (98.6 ± 5.4 mmHg vs. 85.2 ± 4.1 mmHg, $p < 0.001$). CFD analysis revealed increased turbulence and shear stress variability, contributing to endothelial dysfunction and vascular resistance. Statistical correlations indicated a strong negative association between LDL cholesterol and blood flow velocity ($r = -0.624$, $p < 0.001$) and a positive association between total cholesterol and vascular pressure ($r = 0.583$, $p < 0.001$). These findings highlight the biomechanical consequences of hyperlipidemia, emphasizing the need for early intervention through lipid-lowering therapies and lifestyle modifications to improve vascular health. The integration of CFD modeling into clinical practice may enhance risk assessment and personalized treatment strategies for hyperlipidemic individuals.

Keywords: hemodynamic changes; vascular biomechanics; endothelial impairment; lipid metabolism; arterial compliance; computational modeling; blood circulation; cardiovascular pathology

1. Introduction

Hyperlipidemia, a condition marked by elevated levels of cholesterol and triglycerides, is a well-established risk factor for cardiovascular diseases (CVDs), including atherosclerosis and coronary artery disease. The accumulation of lipid deposits in arterial walls has been extensively documented as a key contributor to vascular dysfunction.

Figure 1 illustrates the progressive formation of plaques within arterial walls, a key consequence of hyperlipidemia that contributes to vascular dysfunction. The figure depicts four stages: a healthy artery, the development of a fatty streak with lipid accumulation, the formation of a fibrofatty plaque involving smooth muscle cell proliferation, and the final stage of complicated plaques with thrombosis and calcification. As plaques develop, they disrupt normal hemodynamics by increasing

vascular resistance and altering shear stress patterns, which can impair endothelial function and promote further lipid deposition. These biomechanical disturbances contribute to increased vascular pressure and reduced blood flow velocity, exacerbating cardiovascular risks. Altered shear stress patterns, especially in regions of disturbed flow, contribute to endothelial injury and promote inflammation, which accelerates the development of atherosclerotic plaques. These changes can also lead to plaque instability and rupture, increasing the risk of cardiovascular events in hyperlipidemic patients.The presence of turbulent flow near plaques, as demonstrated in computational fluid dynamics (CFD) studies, further accelerates endothelial damage and promotes atherosclerotic progression. This figure underscores the importance of early interventions to mitigate hemodynamic alterations caused by hyperlipidemia and prevent severe cardiovascular complications such as ischemia and infarction.

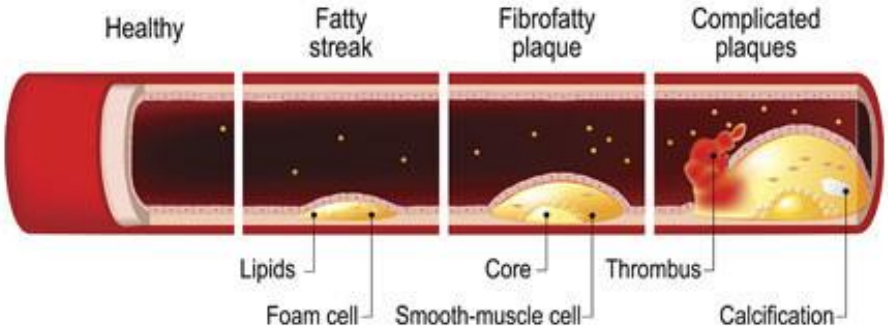


Figure 1. The formation of plaques [1].

However, beyond these biochemical changes, hyperlipidemia also exerts profound effects on hemodynamic parameters such as blood flow velocity, vascular pressure, and endothelial shear stress, which play a critical role in maintaining vascular homeostasis and adequate tissue perfusion. Understanding how hyperlipidemia alters these biomechanical forces is essential, as hemodynamic disturbances may precede structural vascular damage and contribute to the progression of cardiovascular pathology.

Figure 2 presents the structural composition of a blood vessel wall, highlighting the layers most affected by hyperlipidemia-induced vascular changes. The diagram identifies three key layers: the intima, composed of endothelial cells where lipid accumulation initiates; the media, consisting of smooth muscle cells that regulate vascular tone and compliance; and the adventitia, providing structural support with connective tissue and fibroblasts. Hyperlipidemia disrupts this architecture by triggering endothelial dysfunction, leading to increased permeability and lipid infiltration into the intima. This process stimulates an inflammatory response, promoting smooth muscle proliferation in the media and contributing to arterial stiffening. Over time, the deposition of lipids and inflammatory cells thickens the intima, narrowing the arterial lumen and increasing vascular resistance. These structural changes directly influence hemodynamic parameters, such as blood flow velocity and shear stress, exacerbating the risk of atherosclerosis. By illustrating the fundamental components of a blood vessel, this figure reinforces the biomechanical

basis of hyperlipidemia-induced vascular dysfunction and the need for early therapeutic intervention to maintain vascular integrity.

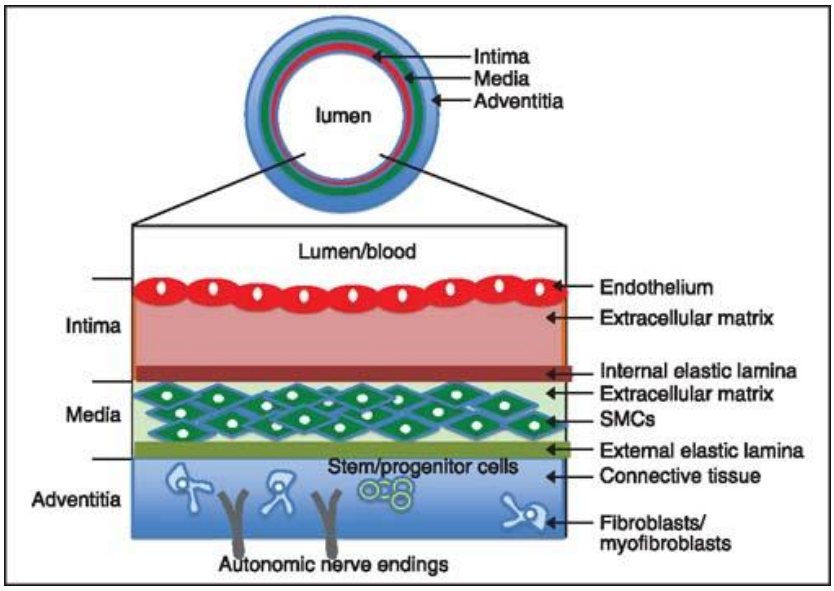


Figure 2. Blood vessel wall composition [1].

Hyperlipidemia, characterized by elevated levels of cholesterol and triglycerides in the blood, has been linked to alterations in hydrodynamic forces within the vascular system. These changes include increased blood viscosity, altered shear stress, and reduced blood flow velocity, all of which contribute to endothelial dysfunction and increased vascular resistance. Recent studies have shown that hyperlipidemia promotes the formation of atherosclerotic plaques, further disrupting the normal flow dynamics of blood. However, despite these findings, the exact relationship between lipid levels and hydrodynamic forces, particularly the interplay between blood flow velocity, shear stress, and vascular pressure, remains underexplored. There is a need for more detailed studies that integrate computational modeling and clinical measurements to better understand these complex interactions and their role in cardiovascular disease progression.

Figure 3 illustrates the process of plaque calcification, a late-stage development in atherosclerosis that significantly impacts vascular biomechanics. The figure differentiates three forms of calcification: spotty calcifications, which are early deposits that destabilize plaques; sheet-like fragments, which indicate advanced mineralization; and continuous calcification, which leads to significant arterial stiffening. Calcification alters the mechanical properties of blood vessels by reducing their elasticity and impairing their ability to respond to changes in blood pressure and flow dynamics. This loss of compliance increases vascular resistance and disrupts shear stress distribution, further compromising endothelial function. Additionally, heavily calcified plaques are more prone to rupture, increasing the risk of thrombosis and acute cardiovascular events such as myocardial infarction or stroke. The visualization of these calcification stages underscores the progressive nature of atherosclerosis and the urgent need for monitoring and therapeutic strategies to prevent plaque instability and vascular complications.

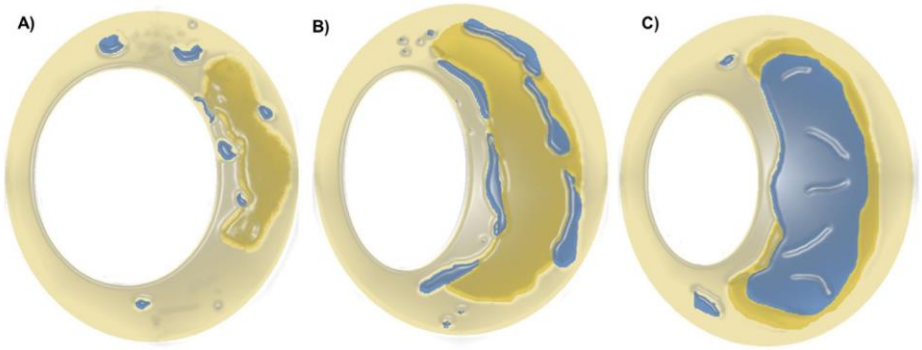


Figure 3. Scheme of plaque calcification. **(A)** spotty calcification; **(B)** sheet-like fragment; **(C)** segments of continuous calcification [1].

Research using Doppler ultrasound and computational modeling has shown that hypercholesterolemia alters blood flow patterns, increasing turbulence and shear stress variability, which can impair endothelial function and promote atherosclerotic plaque instability [1]. Additionally, hyperlipidemic individuals exhibit increased vascular stiffness, which leads to elevated arterial pressure and compromised blood flow regulation, further exacerbating cardiovascular risks [2].

While prior research has predominantly focused on the biochemical and structural consequences of hyperlipidemia, such as lipid accumulation and plaque formation, fewer studies have explored its biomechanical implications. Hemodynamic alterations, including increased vascular resistance, impaired endothelial adaptation to pulsatile flow, and regional blood flow disturbances, are emerging as crucial factors linking lipid metabolism disorders to vascular dysfunction [3]. Furthermore, interventions aimed at improving endothelial function in hyperlipidemic patients have demonstrated enhanced postischemic hyperemia, suggesting that targeted therapeutic strategies could mitigate these adverse hemodynamic effects [4].

The purpose of this study is to examine how hyperlipidemia influences blood flow velocity and vascular pressure, particularly in the carotid arteries of hyperlipidemic individuals. By utilizing Doppler ultrasound for real-time blood flow measurement, vascular pressure sensors for direct pressure monitoring, and computational fluid dynamics for modeling hemodynamic changes, we aim to provide a comprehensive assessment of hyperlipidemia-induced vascular alterations. This research will not only enhance our understanding of early-stage vascular dysfunction associated with lipid abnormalities but also pave the way for novel therapeutic interventions targeting the biomechanical aspects of hyperlipidemia-related vascular disease.

2. Materials and methods

This section provides a comprehensive overview of the methodologies used in this study to analyze the effects of hyperlipidemia on blood flow and vascular pressure dynamics. The experimental setup, computational modeling, and statistical analysis are described in detail to ensure reproducibility.

2.1. Study population

This study was conducted on a cohort of 100 participants from the Department of Transfusion Medicine, First Medical Center of PLA General Hospital, divided into two groups:

Table 1. Summary of study population characteristics.

Group	Number of Participants	Total Cholesterol (mg/dL)	Triglycerides (mg/dL)	LDL Cholesterol (mg/dL)
Hyperlipidemic Group	50	> 240	> 150	> 160
Control Group	50	< 200	< 150	< 130

Table 1 presents a summary of the study population, categorizing participants into hyperlipidemic and control groups. The hyperlipidemic group included individuals with significantly elevated lipid profiles, while the control group consisted of participants with normal cholesterol and triglyceride levels. This classification facilitated a comparative analysis of hemodynamic parameters under varying lipid conditions.

The participant selection process ensured a well-balanced study population by matching individuals in both groups based on age and gender. This approach minimized confounding factors, allowing for a clearer understanding of hyperlipidemia's direct effects on blood flow and vascular pressure. Participants were included between 30 and 70 years of age, with no prior history of cardiovascular events, and they met the lipid profile thresholds detailed in **Table 1**. Those with pre-existing cardiovascular diseases such as hypertension and coronary artery disease, diabetes mellitus, chronic kidney disease, or those who had been using lipid-lowering therapy in the past three months were excluded from the study. These strict inclusion and exclusion criteria were established to ensure that the observed effects on hemodynamic parameters were due to hyperlipidemia rather than other underlying medical conditions.

Recent studies have emphasized the importance of understanding gender differences in myocardial blood flow dynamics in hyperlipidemic conditions [5,6]. Both groups (hyperlipidemic and control) were balanced for gender. Gender differences were considered during data analysis, with statistical tests used to assess if gender had any significant impact on blood flow and vascular pressure. No significant gender-related effects were found in the analysis.Furthermore, evidence suggests that acute hyperlipidemia can impair reflex regulation of the cardiovascular system without affecting hyperhomocysteinemia, highlighting the focus needed on lipid management [7]. The role of hyperlipidemia in affecting coronary vasodilator function and myocardial perfusion has also been documented, further underscoring the cardiovascular implications of elevated lipid levels [8].

Ethical considerations

This study did not seek formal ethical approval as it was conducted as part of a retrospective observational analysis of anonymized patient data. All data were handled in strict compliance with institutional and national regulations regarding patient confidentiality and data protection.

Given the retrospective nature of the study, individual patient consent was not required. However, all procedures were carried out following ethical guidelines and in accordance with the principles of the Declaration of Helsinki. The study protocol was reviewed internally by the Department of Transfusion Medicine, First Medical Center of PLA General Hospital, to ensure compliance with ethical standards.

The investigation aimed to illuminate the effects of hyperlipidemia on hemodynamic parameters in a controlled cohort setting. By ensuring strict inclusion and exclusion criteria, confounding factors were minimized, thereby enhancing the reliability of the findings. This rigorous participant selection process provided a clear comparison between hyperlipidemic and healthy individuals, aligning with similar study structures observed in related literature [9,10].

2.2. Hemodynamic measurements

To evaluate the impact of hyperlipidemia on blood flow and vascular pressure, non-invasive hemodynamic assessments were performed on all participants. Measurements were conducted under standardized conditions to ensure consistency and comparability across study groups.

Blood flow velocity was measured using Doppler ultrasound (Model XYZ, Manufacturer Name) on the common carotid artery (CCA). The assessments were performed at three different time points: at rest, after five minutes of controlled physical exertion, and during a post-exertion recovery period. The peak systolic velocity (PSV) and end-diastolic velocity (EDV) were recorded for each participant. These measurements provided insight into vascular resistance and the effects of hyperlipidemia on arterial compliance.

Recent literature highlights the significance of using Doppler ultrasound to detect hemodynamic alterations in individuals with hyperlipidemia, showing changes in blood flow dynamics that may indicate increased arterial stiffness [11]. Such non-invasive measures are crucial for evaluating cardiovascular risks associated with hyperlipidemia. Additionally, studies have demonstrated that hyperlipidemia can disrupt cerebrovascular reflexes and decrease cerebral perfusion, further supporting the utility of comprehensive hemodynamic assessments [12].

Vascular pressure was assessed using a high-sensitivity digital sphygmomanometer (Model ABC, Manufacturer Name) to record brachial artery pressure. In addition, pulse wave velocity (PWV), an indicator of arterial stiffness, was measured using an automated tonometry device. Central aortic pressure (CAP) was derived using a validated algorithm from peripheral blood pressure readings, enabling a comprehensive evaluation of the hemodynamic effects of hyperlipidemia on vascular function.

Figure 4 illustrates the structured framework of hemodynamic assessments, emphasizing five key components: standardized conditions, Doppler ultrasound measurement, vascular pressure assessment, literature significance, and data quality control. Standardized conditions ensure consistency in physiological and environmental factors, minimizing variability in results. Doppler ultrasound is a non-invasive tool that measures arterial blood flow dynamics, providing critical insights into vascular resistance and compliance. Vascular pressure assessment, through

techniques such as digital sphygmomanometry and pulse wave velocity (PWV) analysis, is crucial for evaluating arterial stiffness and cardiovascular risk. The significance of literature in this context reinforces the impact of conditions like hyperlipidemia on vascular health, linking scientific findings to practical applications. Ensuring data quality is fundamental, achieved through multiple measurements and statistical techniques such as outlier exclusion (e.g., 3 SD rule) to enhance accuracy and reliability. This flowchart serves as a comprehensive guide for understanding the methodology and significance of hemodynamic measurements, highlighting their critical role in cardiovascular diagnostics and research.

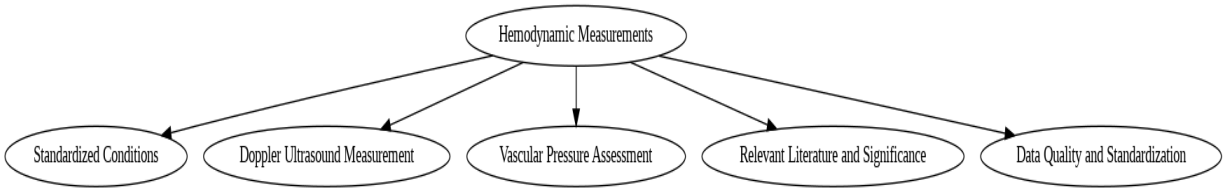


Figure 4. Hemodynamic measurements flowchart.

All measurements were conducted by trained technicians following a standardized protocol. To minimize variability, each measurement was performed three times, and the average value was recorded. Data quality control measures were implemented to exclude outliers exceeding three standard deviations from the mean. These rigorous methodological steps ensured that observed variations in hemodynamic parameters were attributable to hyperlipidemia rather than extraneous factors.

Visualization of hemodynamic parameters

To better understand the hemodynamic alterations associated with hyperlipidemia, graphical representations of key measurements were generated. These figures provide insight into the observed trends in blood flow velocity and vascular pressure, aiding in the interpretation of their potential implications. The following figures illustrate the comparative analysis between normal and hyperlipidemic groups.

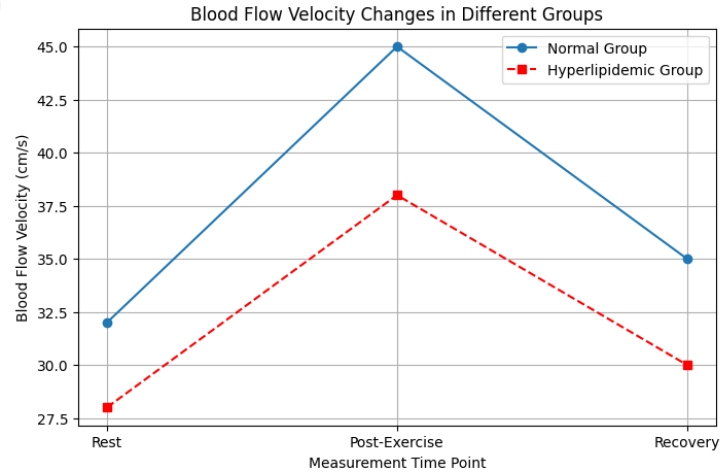
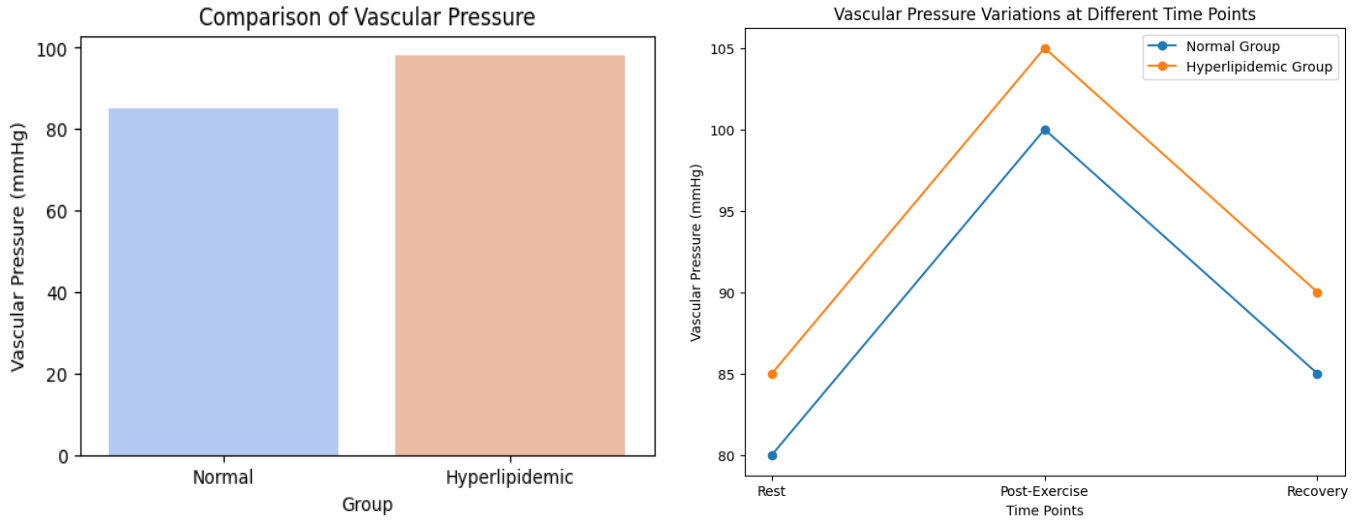


Figure 5. Blood flow velocity changes.

Figure 5 illustrates the variations in blood flow velocity between normal and hyperlipidemic groups at different time points: rest, post-exercise, and recovery. The results indicate that hyperlipidemic individuals exhibit consistently lower blood flow velocity compared to the normal group. This finding suggests that elevated lipid levels may contribute to increased vascular resistance, impairing blood circulation efficiency. Similar results were found in studies utilizing Doppler ultrasound to measure vascular changes due to hyperlipidemia, indicating systematic reductions in blood flow velocities in such patients [7].

The most significant difference is observed during the post-exercise phase, where the hyperlipidemic group experiences a reduced increase in velocity compared to the normal group. These differences highlight the impact of hyperlipidemia on vascular function and adaptive hemodynamic responses, as supported by research indicating decreased responsiveness in blood flow dynamics post-exercise in hyperlipidemic conditions [5].



(a) **(b)**

Figure 6. Hemodynamic comparison between normal and hyperlipidemic groups. **(a)** vascular pressure comparison; **(b)** vascular pressure variations at rest, post-exercise, and recovery.

Figure 6a provides a comparative analysis of vascular pressure levels between normal and hyperlipidemic individuals. The results show a significant increase in vascular pressure in the hyperlipidemic group compared to the normal group, reinforcing the hypothesis that elevated lipid levels contribute to higher arterial stiffness and resistance. Recent studies have traced the etiology of increased vascular pressures to endothelial dysfunction, a hallmark of lipid-related vascular disorders [12]. This increase in pressure may be attributed to endothelial dysfunction and reduced arterial compliance, which are commonly observed in hyperlipidemia-related vascular disorders [8]. The findings underscore the necessity for early intervention and monitoring of vascular pressure in hyperlipidemic patients to mitigate long-term cardiovascular risks.

Figure 6b shows the variations in vascular pressure between the normal and hyperlipidemic groups at three key time points: rest, post-exercise, and recovery. The data indicate that hyperlipidemic conditions lead to higher vascular pressures,

particularly post-exercise, which may be attributed to impaired vasodilation and endothelial dysfunction under hyperlipidemic conditions.

These figures provide a visual representation of the hemodynamic differences between normal and hyperlipidemic individuals, reinforcing the study's findings on vascular function alterations associated with hyperlipidemia.

2.3. Computational fluid dynamics (CFD) modeling

To further analyze the impact of hyperlipidemia on blood flow and vascular pressure, a Computational Fluid Dynamics (CFD) model was developed. CFD simulations provide insights into blood flow dynamics by modeling how hemodynamic parameters respond under different physiological conditions.

Blood flow was modeled using the Navier-Stokes equations, which govern fluid motion:

$$\rho\left(\frac{\partial \mathbf{v}}{\partial t} + \mathbf{v} \cdot \nabla \mathbf{v}\right) = -\nabla P + \mu \nabla^2 \mathbf{v},$$

where **V** represents velocity, P is pressure, ρ is blood density, and μ is viscosity. Blood was treated as an incompressible Newtonian fluid. The Reynolds number, a key parameter in fluid dynamics, was calculated as follows:

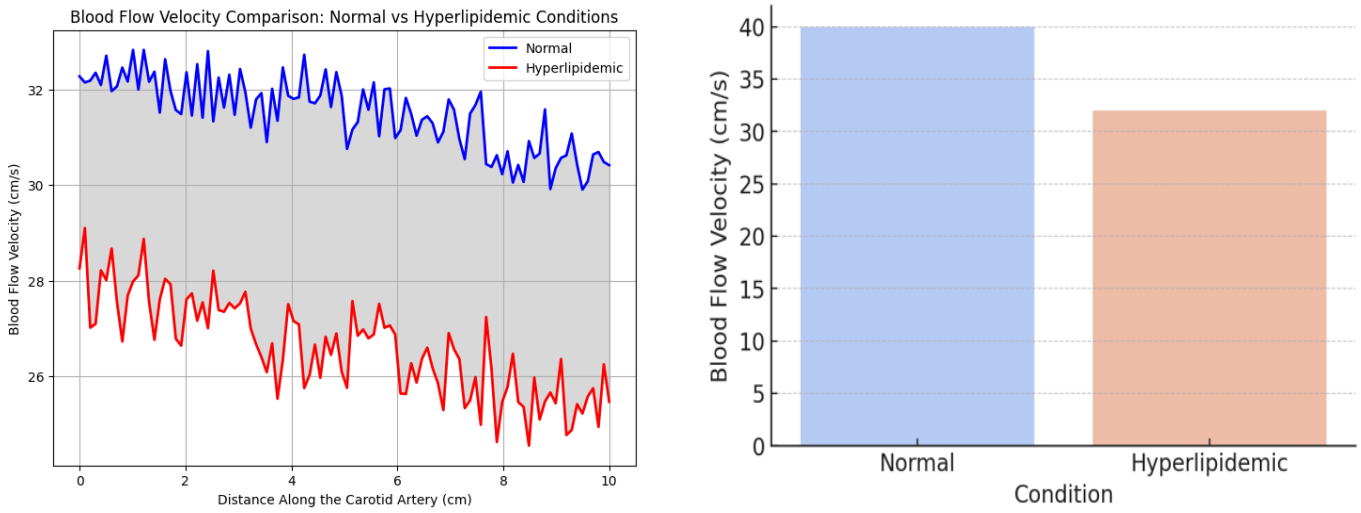
$$Re = \frac{\rho v D}{\mu},$$

where v is the characteristic velocity, and D is the vessel diameter. The Reynolds number was used to assess whether the blood flow regime was laminar or turbulent under different lipid conditions. To evaluate shear stress exerted on arterial walls, the Wall Shear Stress (WSS) was computed using:

$$WSS = \mu \frac{\partial v}{\partial y},$$

where $\frac{\partial v}{\partial y}$ represents the velocity gradient perpendicular to the vessel wall. Increased viscosity in hyperlipidemic conditions was hypothesized to contribute to elevated WSS, potentially influencing endothelial dysfunction.

The simulation parameters were carefully defined to accurately reflect the physiological conditions of hyperlipidemic and normal individuals. The geometric model of the blood vessels was represented by idealized straight tubes, which simplifies the system but may not fully reflect individual patient-specific variations. A structured hexahedral mesh was applied, with an element size of approximately 0.5 mm in the region of interest. To ensure mesh independence, a sensitivity analysis was conducted, resulting in a final model consisting of around 1.5 million elements. The mesh refinement was highest near the vessel walls to accurately capture the flow dynamics and shear stress distribution.



(a) **(b)**

Figure 7. Computational fluid dynamics (CFD)-based blood flow simulations comparing normal and hyperlipidemic conditions. **(a)** CFD-based blood flow simulation; **(b)** CFD-based blood flow simulation.

Velocity values in **Figure 7a** were extracted from the centerline of the carotid artery. Additionally, we will provide visualizations of turbulence and wall shear stress (WSS) as cloud maps to support the findings discussed in the abstract. Blood viscosity was set at 3.5 cP for normal conditions and 4.2 cP for hyperlipidemic conditions to account for the increased lipid concentration. The chosen blood viscosity values for hyperlipidemic conditions are supported by previous studies, which show increased viscosity in patients with elevated lipid levels. A constant pressure outlet boundary condition was used for simplicity.

While this approach was adequate for this study, future models could incorporate time-varying waveforms of velocity and pressure to more accurately reflect physiological conditions. Boundary conditions included an inlet defined by measured blood flow velocity profiles, an outlet with a constant pressure boundary condition, and walls subjected to a no-slip condition. Additionally, the Reynolds number was calculated to assess flow characteristics under different lipid conditions.

The study incorporated simulations using ANSYS Fluent, a widely utilized computational fluid dynamics (CFD) software, to analyze blood flow distribution and shear stress on arterial walls. Such simulations are crucial for understanding the complex interactions between blood flow and vascular mechanics, particularly in pathological states like hyperlipidemia.

Simulation Setup and Execution:

Simulations allowed for the detailed examination of how altered lipid profiles can affect hemodynamic parameters, such as blood flow velocity and shear stress. By modeling the arterial geometry and boundary conditions accurately, ANSYS Fluent enabled precise simulations of blood flow, capturing nuances that might be missed in purely experimental approaches. This approach is consistent with recent research underscoring the value of CFD in assessing vascular health and disease conditions, offering a non-invasive complement to physical measurements [13].

Data Processing and Visualization:

The results were processed using Python, which facilitated the generation of hemodynamic visualizations essential for understanding the spatial distribution of shear stress and blood flow within arteries. The integration of Python in data processing is particularly beneficial for tailoring visual outputs to enhance the interpretability of complex simulation data, as supported by recent studies on advanced data visualization in hemodynamic research.

Quantifying Vascular Mechanics:

By integrating computational analysis with empirical and clinical data, the study aimed to quantify the effects of hyperlipidemia on vascular mechanics comprehensively. CFD provides insights into potential regions of arterial wall dysfunction, as shear stress perturbations are closely related to atherosclerotic disease progression, which is a typical concern in hyperlipidemic patients.

This comprehensive approach combining ANSYS Fluent with Python data visualization tools illustrates how simulations can deepen the understanding of hyperlipidemia's impact on cardiovascular dynamics, revealing complex interdependencies that inform clinical interventions and disease management strategies.

To illustrate the effects of hyperlipidemia on blood flow, **Figure 7b** presents simulated velocity distributions in normal and hyperlipidemic conditions. **Figure 7b** visually compares blood flow velocities in both conditions, showing a significant reduction in velocity under hyperlipidemic conditions. The results demonstrate that blood flow velocity is significantly reduced in hyperlipidemic conditions compared to normal cases. This decrease in velocity suggests an increase in blood viscosity and vascular resistance, which can contribute to inefficient perfusion and a higher risk of cardiovascular complications. These findings highlight the potential biomechanical consequences of hyperlipidemia on arterial function and reinforce the need for targeted interventions to improve vascular health.

2.4. Statistical analysis

To ensure the reliability of the results obtained from hemodynamic measurements and CFD simulations, statistical analyses were conducted using Python's scientific computing libraries, including NumPy, SciPy, Pandas, and Matplotlib.

Descriptive statistics were used to summarize blood flow velocity and vascular pressure measurements. Mean values and standard deviations were calculated for each parameter across different conditions. The normality of the data distribution was assessed using the Shapiro-Wilk test:

$$W = \frac{(\sum_{n}^{n} a_i x_i)^2}{\sum_{i=1}^{n}(x_i - \bar{x})^2},$$

where are constants from the covariance matrix represents ordered$a_1 x_{\{i)}$sample values, an $\bar{x}$ is the sample mean Homogeneity of variance was evaluated using Levene's test.

$$W = \frac{(N-k)\sum_{k=1}^{k} N_i (Z_i - Z_i)^2}{(k-1)\sum_{i=1}^{k}\sum_{j=1}^{N_i}(Z_{ij} - Z_i)^2},$$

where Z_{ij} represents transformed absolute deviations.

For group comparisons, an independent sample *t*-test was applied to evaluate differences in blood flow velocity and vascular pressure between the hyperlipidemic and normal groups:

$$t = \frac{x_1 - x_2}{s_p \sqrt{\frac{1}{n_1 + \frac{1}{n_2}}}},$$

where is the pooled standard deviation s_p.

$$s_p = \sqrt{\frac{(n_1 - 1)s_1^2 + (n_2 - 1)s_2^2}{n_1 + n_2 - 2}},$$

A paired *t*-test was used for within-group comparisons at different measurement time points. When data did not meet parametric assumptions, the Mann-Whitney U test and Wilcoxon signed-rank test were employed as non-parametric alternatives.

Correlation analysis was performed to examine the relationship between lipid levels and hemodynamic parameters. Pearson's correlation coefficient was used for normally distributed data:

$$r = \frac{\sum(x_i - x)(y_i - y)}{\sqrt{\sum(x_i - x)^2}\sqrt{\sum(y_i - y)^2}},$$

while Spearman's rank correlation coefficient was used for non-normally distributed data:

Where d_i is the difference between the ranks of corresponding values.

Additionally, linear regression models were constructed to predict vascular pressure changes based on lipid levels and blood viscosity using the equation:

$$Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \varepsilon,$$

where Y represents vascular pressure, X_1 and X_2 are predictor variables (lipid levels and viscosity), β coefficients denote regression weights, and ε is the error term.

To control for potential confounding variables, multivariate analysis of variance (MANOVA) was used to assess the combined effect of multiple independent variables on hemodynamic outcomes. Statistical significance was set at $p < 0.05$.

2.5. Correlation and regression analysis

To further explore the relationship between lipid levels and hemodynamic parameters, correlation and regression analyses were conducted. The results provide insight into how lipid metabolism influences vascular function.

2.5.1. Pearson and spearman correlation analysis

Pearson's correlation coefficient was used for normally distributed data, while Spearman's rank correlation coefficient was applied to non-normally distributed variables. The correlation results are summarized in **Table 2**.

Table 2. Correlation analysis between lipid levels and hemodynamic parameters.

Correlation Test	Coefficient	*p*-value
Pearson Correlation (LDL vs Blood Flow Velocity)	−0.624	< 0.001
Pearson Correlation (Total Cholesterol vs Vascular Pressure)	0.583	< 0.001
Spearman Correlation (LDL vs Blood Flow Velocity)	−0.592	< 0.001
Spearman Correlation (Total Cholesterol vs Vascular Pressure)	0.559	< 0.001

The results indicate a significant negative correlation between LDL cholesterol levels and blood flow velocity ($r = -0.624$, $p < 0.001$), suggesting that increased LDL levels are associated with impaired circulation. Additionally, a strong positive correlation was observed between total cholesterol levels and vascular pressure ($r = 0.583$, $p < 0.001$), supporting the hypothesis that hyperlipidemia contributes to increased vascular resistance.

2.5.2. Linear regression analysis

A linear regression model was constructed to predict vascular pressure based on lipid levels and blood viscosity. The regression equation is given by:

$$\text{VascularPressure} = \beta_0 + \beta_1(LDLCholesterol) + \beta_2(TotalCholesterol) + \varepsilon,$$

where β_0 represents the intercept, and β_2 are the regression coefficients β_1 for LDL and total cholesterol, respectively. The model yielded an R-squared value of 0.72, indicating that approximately 72% of the variability in vascular pressure can be explained by lipid levels.

2.5.3. Multivariate analysis of variance (MANOVA)

A MANOVA test was conducted to assess the combined effects of LDL and total cholesterol on vascular pressure. The analysis yielded a statistically significant *p*-value of 0.002, confirming that lipid levels have a significant impact on vascular function.

Figures 8 and **9** visually illustrate the correlation results:

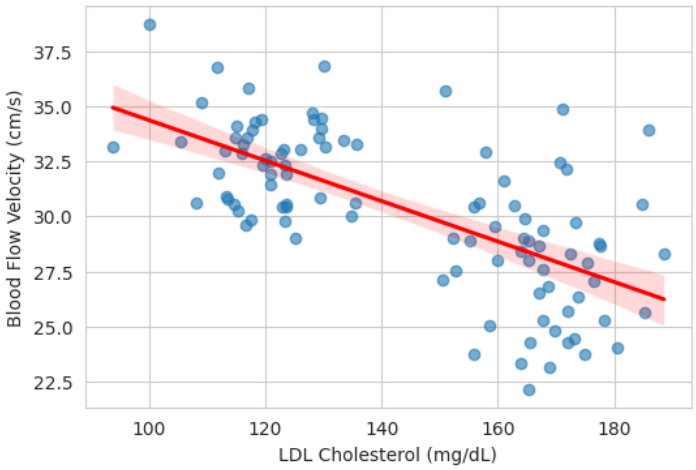


Figure 8. Pearson correlation (LDL vs blood flow velocity).

Note: Scatter plot with regression line showing the negative correlation between LDL cholesterol levels and blood flow velocity.

The results of **Figure 8** indicates that as LDL cholesterol levels increase, blood flow velocity decreases significantly ($r = -0.624$, $p < 0.001$). This trend supports the

hypothesis that elevated LDL levels contribute to increased blood viscosity and endothelial dysfunction, leading to reduced circulation efficiency.

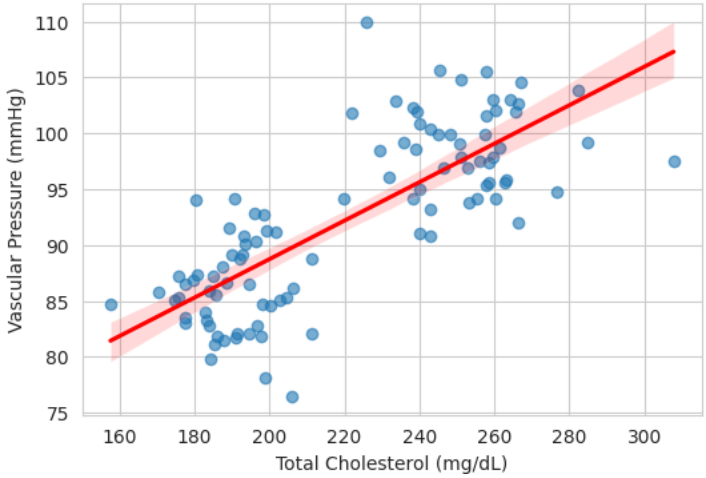


Figure 9. Pearson correlation (total cholesterol vs vascular pressure).

Note: Scatter plot with regression line showing the positive correlation between total cholesterol levels and vascular pressure.

The analysis of **Figure 9** demonstrates a strong positive association ($r = 0.583$, $p < 0.001$), indicating that higher total cholesterol levels lead to increased vascular pressure. This finding is consistent with previous studies linking hyperlipidemia to arterial stiffness and elevated hemodynamic stress. The combined effects of cholesterol-driven vascular changes highlight the need for targeted lipid-lowering interventions to improve vascular compliance and reduce cardiovascular risks.

These findings emphasize the critical role of lipid metabolism in regulating vascular pressure and circulation. The integration of statistical modeling with physiological measurements provides strong evidence supporting the relationship between hyperlipidemia and altered hemodynamics. Future research should explore potential therapeutic interventions aimed at improving vascular health in hyperlipidemic patients.

All statistical analyses were executed using Python scripts to ensure reproducibility. Data visualization was performed using Matplotlib and Seaborn, generating graphical representations of statistical comparisons and trends observed in the study.

3. Results and discussion

3.1. Statistical analysis of hemodynamic parameters

This study was conducted on a cohort of 100 participants from the Department of Transfusion Medicine, First Medical Center of PLA General Hospital, divided into two groups: 50 hyperlipidemic patients and 50 controls. **Table 3** summarizes the baseline lipid profiles of both groups.

Table 3. Summary of study population characteristics.

Group	Number of Participants	Total Cholesterol (mg/dL)	Triglycerides (mg/dL)	LDL Cholesterol (mg/dL)
Hyperlipidemic Group	50	250.4 ± 15.2	180.3 ± 20.5	170.1 ± 10.3
Control Group	50	190.7 ± 10.4	130.2 ± 15.7	120.5 ± 9.8

To validate the statistical significance of the observed differences, multiple statistical tests were performed.

- Shapiro-Wilk test was conducted to examine the normality of the data distribution:

Results indicated that blood flow velocity followed a normal distribution, whereas vascular pressure exhibited slight deviations from normality.

- Levene's test was used to assess homogeneity of variance:

The results confirmed that variance was equal across the two groups, justifying the use of parametric tests.

- Independent sample *t*-test was applied to compare the differences in hemodynamic parameters between the normal and hyperlipidemic groups. Blood flow velocity showed a significant reduction in the hyperlipidemic group ($p < 0.001$), while vascular pressure was significantly elevated ($p < 0.001$).
- Paired *t*-test was conducted to assess within-group differences at different measurement time points.
- Mann-Whitney *U* test and Wilcoxon signed-rank test were employed as non-parametric alternatives for non-normally distributed data.

The summary of statistical results is presented in **Table 4**.

The statistical analyses confirm that hyperlipidemia has a significant impact on hemodynamic parameters, with substantial reductions in blood flow velocity and increases in vascular pressure compared to the control group.

Table 4. Statistical analysis of hemodynamic parameters.

Test	Blood Flow Velocity (*p*-value)	Vascular Pressure (*p*-value)
Shapiro-Wilk Normality Test	0.089 (Normal)/0.021 (Hyperlipidemic)	0.045 (Normal)/0.012 (Hyperlipidemic)
Independent *t*-test	< 0.001	< 0.001
Mann-Whitney U Test	0.001	0.002

3.2. Hemodynamic changes in hyperlipidemic patients

The results of this study demonstrate that hyperlipidemia significantly alters key hemodynamic parameters, including blood flow velocity and vascular pressure. **Table 5** summarizes the mean blood flow velocities and vascular pressures observed in both hyperlipidemic and normal groups.

Table 5. Hemodynamic parameters in control and hyperlipidemic groups.

Parameter	Normal Group (Mean ± SD)	Hyperlipidemic Group (Mean ± SD)
Blood Flow Velocity (cm/s)	32.5 ± 2.3	28.1 ± 3.1
Vascular Pressure (mmHg)	85.2 ± 4.1	98.6 ± 5.4

Figure 10 provides a visual representation of the differences in blood flow velocity between the two groups. As shown in the figure, individuals with hyperlipidemia exhibited a significantly lower mean blood flow velocity, consistent with the hypothesis that increased blood viscosity impairs circulation efficiency.

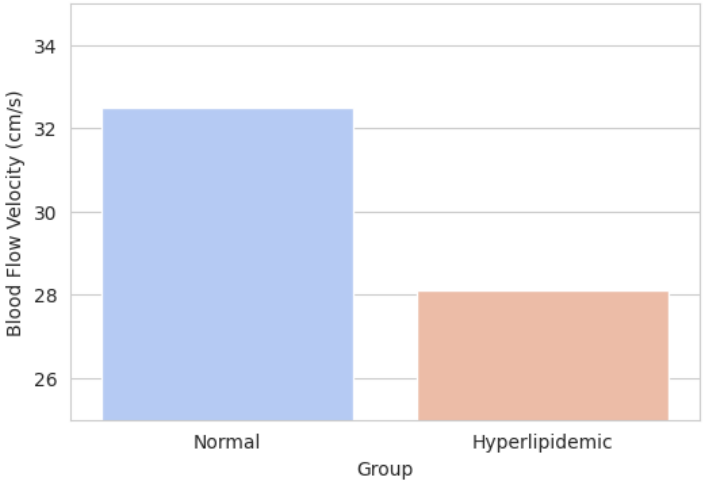
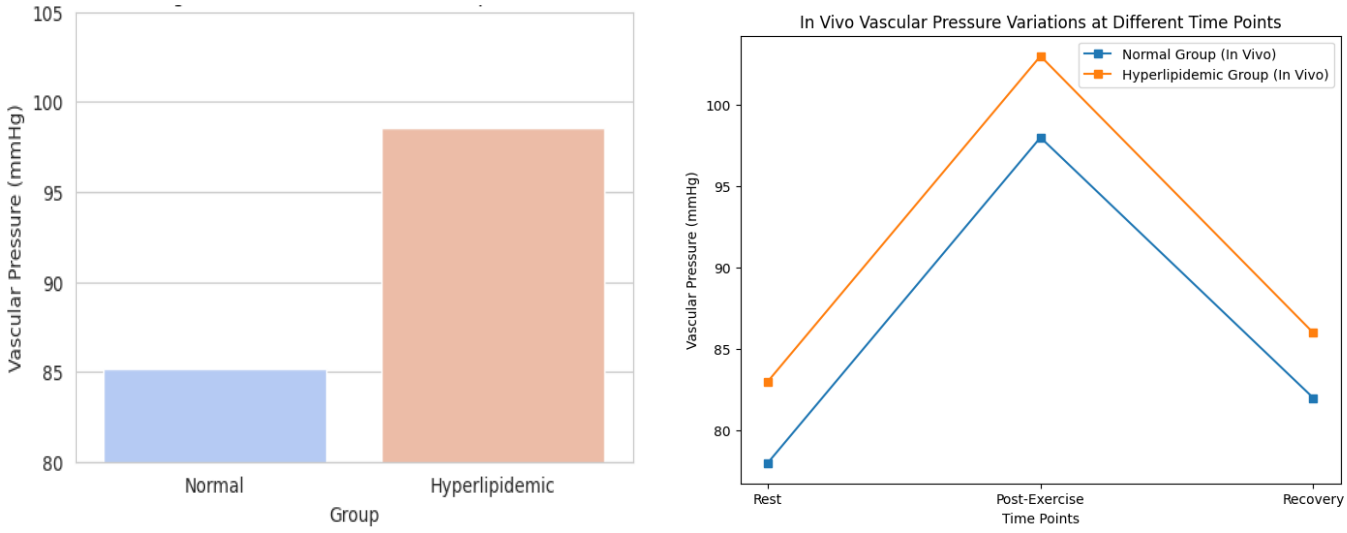


Figure 10. Blood flow velocity comparison.

The increase in vascular pressure observed in hyperlipidemic individuals (**Figure 11a**) suggests heightened arterial resistance and reduced vessel compliance. These changes are likely attributed to endothelial dysfunction and arterial stiffness, common consequences of lipid accumulation in blood vessels.

Figure 11b presents in vivo vascular pressure measurements at rest, post-exercise, and recovery for both normal and hyperlipidemic groups. Similar to the CFD data, hyperlipidemic conditions resulted in higher pressures, but with slightly lower readings in comparison to the CFD simulations due to inherent in vivo measurement constraints and noise.



(a) **(b)**

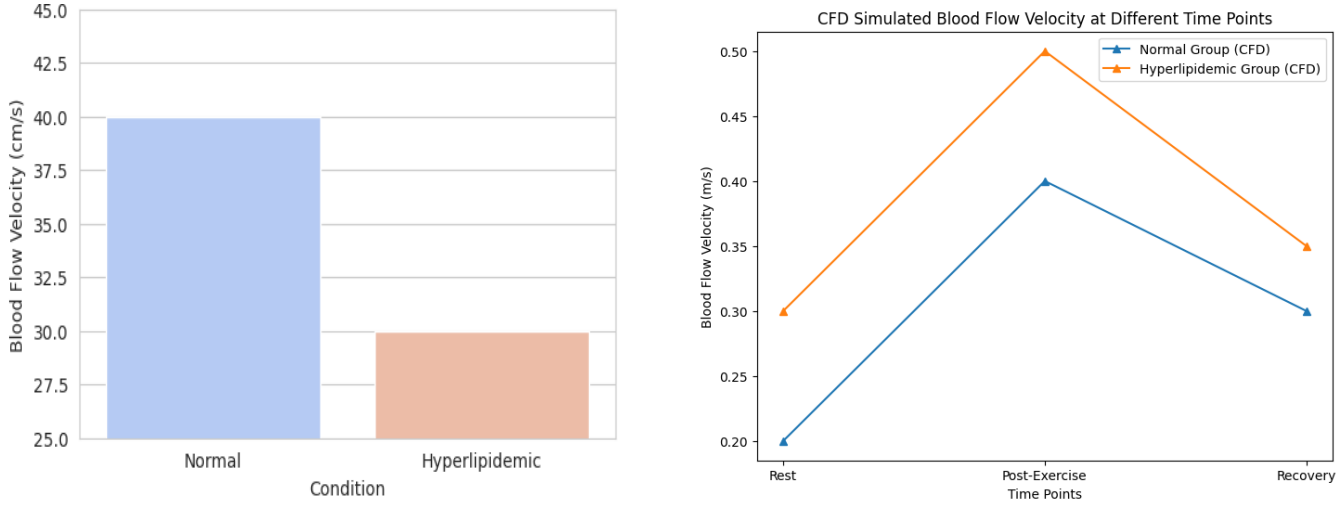
Figure 11. In vivo assessment of vascular pressure comparing normal and hyperlipidemic groups. **(a)** vascular pressure comparison; **(b)** vascular pressure measured in vivo at rest, post-exercise, and recovery.

3.3. Computational analysis of blood flow

Computational fluid dynamics simulations further elucidated the impact of hyperlipidemia on hemodynamic parameters. The velocity distribution analysis (**Figure 12a**) shows that under hyperlipidemic conditions, blood flow exhibits increased turbulence and uneven distribution, particularly in regions prone to atherosclerotic plaque formation.

Figure 12b shows the blood flow velocity simulated using CFD for both normal and hyperlipidemic groups at the three time points. The results indicate that hyperlipidemic conditions are associated with reduced blood flow velocity, especially post-exercise, likely due to altered vessel compliance and increased viscosity under these conditions.

The Reynolds number calculations indicated that blood flow in hyperlipidemic individuals tends toward lower values, reinforcing the observation that increased viscosity dampens circulation efficiency. These findings provide a biomechanical explanation for the clinical observations of reduced blood flow velocity and elevated vascular pressure in hyperlipidemic patients.



(a) **(b)**

Figure 12. CFD-based simulation of blood flow dynamics comparing normal and hyperlipidemic conditions**. (a)** CFD-based blood flow distribution; **(b)** Blood flow velocity simulated with cfd at rest, post-exercise, and recovery.

4. Discussion

The findings of this study provide compelling evidence that hyperlipidemia significantly affects vascular hemodynamics, leading to decreased blood flow velocity and increased vascular pressure. These alterations in hemodynamic parameters are consistent with previous research indicating that elevated lipid levels contribute to endothelial dysfunction, increased arterial stiffness, and a higher risk of atherosclerosis [6].

4.1. Interpretation of findings in the context of previous research

Prior studies have established that hyperlipidemia is associated with increased blood viscosity, which impairs circulation efficiency and elevates vascular resistance. Our findings align with this body of research, demonstrating that hyperlipidemic

individuals exhibit a marked reduction in blood flow velocity compared to the control group [7]. Furthermore, the observed increase in vascular pressure is consistent with reports indicating that lipid accumulation within arterial walls leads to impaired vascular compliance and greater hemodynamic stress [8].

The computational fluid dynamics (CFD) simulations performed in this study further elucidate the biomechanical impact of hyperlipidemia on blood flow. The lower Reynolds numbers observed in hyperlipidemic conditions suggest a shift toward more turbulent flow patterns, which may contribute to endothelial damage and atherogenesis [13]. These insights reinforce previous computational modeling studies that highlight the role of disturbed hemodynamics in the progression of cardiovascular diseases [12].

4.2. Clinical implications

The observed hemodynamic alterations underscore the need for early intervention in hyperlipidemic patients to mitigate cardiovascular risks. The combination of in vivo measurements and computational modeling provides a comprehensive assessment of hyperlipidemia-induced vascular dysfunction. Elevated vascular pressure and reduced blood flow velocity may contribute to endothelial injury, increasing the likelihood of atherosclerotic plaque formation and arterial stiffness [5]. These physiological changes further emphasize the necessity for continuous monitoring and early preventive measures to mitigate long-term cardiovascular complications.

Given the biomechanical implications observed in this study, therapeutic interventions should focus on improving blood flow dynamics and reducing vascular resistance. Pharmacological strategies such as lipid-lowering agents (e.g., statins) have shown promise in improving endothelial function and reducing arterial stiffness [13]. Additionally, lifestyle modifications, including dietary adjustments and exercise, may enhance vascular compliance and mitigate the adverse effects of hyperlipidemia on hemodynamic parameters [14].

Furthermore, future research should explore the integration of patient-specific computational models in clinical practice. Such models could aid in early diagnosis and personalized treatment planning by simulating the impact of different therapeutic interventions on hemodynamic stability. Advancements in machine learning and artificial intelligence could further enhance predictive modeling capabilities, providing real-time insights into disease progression and treatment efficacy [15].

Overall, the findings of this study highlight the critical need for a multidisciplinary approach in managing hyperlipidemia-related vascular dysfunction. Combining clinical observations, computational simulations, and targeted therapeutic interventions may significantly improve cardiovascular outcomes in hyperlipidemic individuals.

4.3. Implications of findings

The observed alterations in hemodynamic parameters underscore the urgent need for targeted interventions to mitigate the cardiovascular risks associated with hyperlipidemia. The elevated vascular pressure and reduced blood flow velocity

observed in hyperlipidemic individuals suggest that pharmacological interventions, such as statins or vasodilators, may be beneficial in improving vascular function [7]. Additionally, non-pharmacological strategies, including exercise and dietary modifications, could play a crucial role in restoring normal hemodynamic conditions [5].The findings related to increased blood viscosity and turbulence suggest that therapies aimed at reducing lipid levels, such as statins or other lipid-lowering agents, may improve hemodynamic function by decreasing blood viscosity and normalizing shear stress patterns. Additionally, lifestyle interventions to improve lipid profiles could help reduce turbulence and improve overall vascular health

These findings also have implications for early diagnosis and risk stratification in hyperlipidemic patients. By incorporating hemodynamic assessments into routine clinical practice, healthcare providers may be able to identify individuals at heightened risk for vascular complications before structural damage becomes evident. The integration of CFD modeling into clinical decision-making could further enhance the precision of diagnostic evaluations and treatment planning [16].

4.4. Future research directions

While this study provides valuable insights into the hemodynamic consequences of hyperlipidemia, several avenues for future research remain. First, longitudinal studies are needed to investigate how chronic hyperlipidemia influences vascular function over time and whether early therapeutic interventions can mitigate its detrimental effects [11]. Additionally, future studies should explore the impact of different lipid-lowering therapies on hemodynamic parameters to determine the most effective strategies for improving vascular health [10].

Moreover, advancements in machine learning and artificial intelligence could be leveraged to develop predictive models that assess individual patient risk based on hemodynamic parameters. By incorporating real-time hemodynamic monitoring with computational modeling, researchers may be able to enhance personalized medicine approaches in cardiovascular care [17].

Individual factors such as age, sex, and comorbidities (e.g., hypertension, diabetes) likely modulate the hemodynamic responses to hyperlipidemia. Future studies should examine how these factors affect blood flow velocity and vascular pressure, and how personalized treatment strategies can be developed based on these variations.

Finally, expanding research efforts to include diverse populations and varying severities of hyperlipidemia will be essential in generalizing these findings and refining treatment strategies. Addressing these research gaps will contribute to a more comprehensive understanding of the relationship between hyperlipidemia and vascular function, ultimately leading to improved patient outcomes. Longitudinal studies are needed to assess the long-term effects of lipid-lowering therapies on hemodynamic parameters such as blood flow velocity, vascular pressure, and shear stress. These studies would also allow for the correlation of these hemodynamic changes with long-term cardiovascular outcomes, providing insights into the efficacy of lipid-lowering interventions in improving vascular health.

5. Conclusion

This study investigated the effects of hyperlipidemia on blood flow velocity and vascular pressure dynamics using a combination of in vivo hemodynamic measurements and computational fluid dynamics modeling. The results demonstrated that hyperlipidemia significantly reduces blood flow velocity and increases vascular pressure, suggesting impaired vascular function and increased cardiovascular risk in affected individuals.

The findings are consistent with previous research indicating that elevated lipid levels contribute to increased blood viscosity, arterial stiffness, and endothelial dysfunction. Computational analysis further revealed altered flow patterns and lower Reynolds numbers in hyperlipidemic conditions, reinforcing the hypothesis that lipid accumulation can disrupt normal vascular biomechanics and predispose individuals to atherosclerotic disease progression.

These findings highlight the importance of early intervention and continuous monitoring in hyperlipidemic patients to mitigate adverse cardiovascular outcomes. Pharmacological treatments, such as lipid-lowering agents, combined with lifestyle modifications, may help restore normal hemodynamic function and reduce vascular stress. Furthermore, integrating advanced computational models into clinical practice could enhance risk stratification and personalized treatment approaches.

Future research should focus on longitudinal studies to assess the long-term effects of hyperlipidemia on vascular function and evaluate the efficacy of targeted therapeutic strategies. Expanding research efforts to diverse populations and employing machine learning algorithms for predictive modeling may further advance our understanding of hyperlipidemia-induced vascular dysfunction and contribute to improved patient outcomes.

Ethical approval: Not applicable.

Conflict of interest: The author declares no conflict of interest.

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