Cardiovascular Topics

The relationship between radial artery stenosis and whole blood viscosity after transradial coronary angiography

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Abstract

Background: The transradial approach (TRA) is preferred for coronary procedures due to improved outcomes and lower complication rates. However, complications such as radial artery stenosis (RAS) and occlusion (RAO) post TRA require investigation. This study aimed to explore the link between whole blood viscosity (WBV) and RAS/RAO after TRA coronary angiography.

Methods: A retrospective analysis of 215 TRA coronary angiography patients was conducted. Doppler ultrasonography assessed RAS/RAO one month post procedure. WBV, calculated from haematocrit and total plasma protein (TP), was evaluated at low (LSR) and high shear rates (HSR).

Results: RAS/RAO incidence was 15.3%, with 7.4% of patients experiencing RAO. Patients with RAS/RAO showed significantly elevated HSR, LSR and TP levels, with lower blood urea nitrogen levels. Multivariable analysis identified body mass index, HSR and LSR as independent RAS/RAO predictors.

Conclusion: This study established WBV association with RAS/RAO after TRA, suggesting WBV as a potential predictor and aiding pre-TRA risk assessment for alternative angiography routes.

Keywords: blood viscosity, coronary angiography, radial artery

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The transradial (TRA) method in coronary and peripheral procedures has become standard practice because of its numerous advantages, including a lower incidence of vascular complications at the intervention site, decreased mortality rates in patients with acute coronary syndrome, enhanced patient comfort and reduced cost.[1] However, with the increasing prevalence of TRA procedures, a better understanding of associated complications has emerged. Complications include radial artery stenosis (RAS) and occlusion (RAO).

The insertion of a sheath and catheterisation through the radial artery results in endothelial damage, and subsequent post-procedure radial compression leads to blood stasis, potentially causing thrombus formation and RAO.[2] The occurrence of RAO varies widely, ranging from 0.8 to 38% in the published data. RAO hampers re-access through the radial artery, its utilisation as a conduit for coronary bypass grafting, and fistula formation in donors and haemodialysis patients.[3]

Whole blood viscosity (WBV) refers to the inherent resistance to flow within the bloodstream, resulting from frictional interactions among key blood constituents such as plasma, plasma proteins and red blood cells as they traverse blood vessels.[4] WBV is determined for both low (LSR) and high shear rates (HSR) (shear stress), derived from haematocrit (HCT) and total plasma protein (TP) levels.[5]

WBV has been proposed to serve as an independent risk factor for cardiovascular and peripheral vascular diseases.[6] Recent studies have highlighted the significant role of WBV levels in the development of atherosclerosis. Estimated WBV has been linked to carotid thickness, which is believed to correlate with subclinical atherosclerosis.[7] Some studies have suggested that an increase in viscosity heightens the propensity for thrombosis by causing both disruption of blood flow and endothelial damage owing to elevated shear stress.[8]

In this study, we aimed to elucidate the correlation between RAS/RAO and WBV following transradial approach (TRA) angiography.

Methods

This retrospective study enrolled 215 patients who underwent coronary angiography (CAG) using the TRA approach between 1 June 2023 and 1 January 2024. Exclusion criteria were applied to patients with a history of prior radial artery catheterisation, multiple punctures with a radial needle, undergoing radial percutaneous coronary intervention (PCI) either during the same

session or within one month, use of oral calcium channel blockers or oral nitrates before the procedure, pre-procedural systolic blood pressure < 90 mmHg, known RAS before the procedure, history of malignancy, end-stage kidney or liver disease, atrial fibrillation, or receiving anticoagulant therapy for any other indication. Additionally, patients for whom postprocedural data were unavailable were excluded.

The study complied with the guidelines for human studies and was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all the patients. This study was approved by the Sivas Cumhuriyet University Non-Interventional Clinical Research Ethics Committee on 21 September 2023, under protocol number 2023-09/49.

Patients who underwent CAG using the TRA approach without subsequent PCI were examined. Individuals who developed postprocedural RAO or notable RAS were identified. Doppler ultrasonography of the radial artery was conducted during out-patient clinic follow ups one month after the procedure. Patients with > 50% stenosis or complete occlusion of the radial artery were categorised into the RAS/RAO group. Subsequently, the patients were segregated into two groups: those exhibiting significant RAS and those without.

For the CAG procedure, the primary choice was to utilise 6 French (F) Judkins 4.0 for the right and 6F Judkins 3.5 diagnostic catheters. The catheters were advanced using a 0.035 normal guidewire.

After completing CAG, patients were classified as normal if no coronary artery stenosis was detected, as having minimal coronary artery disease (CAD) if stenosis was less than 50%, and as having significant CAD if stenosis was greater than 50%.

All transradial procedures were conducted by experienced interventional cardiologists who had performed more than 500 TRA CAGs annually at a single centre. To support the patient's arm, a wooden board was placed underneath and the arm was positioned in hyperextension at the wrist, directed towards the patient's side. Before radial puncture, 2 ml of 2% prilocaine was subcutaneously injected using an insulin syringe at the procedure site in all patients. Subsequently, a 20-gauge radial needle was inserted 1–1.5 cm proximal to the radial styloid process, followed by the insertion of a 0.021-inch, 45-cm guidewire. A 6F 7-cm-long radial artery sheath (REPA brand) was then placed over the wire.

Subsequently, all patients received 5 000 U of heparin through the sheath. Additionally, patients were administered 200 µg nitroglycerin and 10 ml of normal saline through the sheath. Standard techniques using 0.035-inch guidewires and left and right Judkins diagnostic catheters were employed for the CAG procedures.

After the completion of CAG, the radial sheath was removed, and to ensure radial artery haemostasis, a compression band (TR band, Terumo, Japan) was secured around the patient's wrist. Subsequently, the air chamber was inflated to achieve effective haemostasis. In our investigation, compression times were standardised at 60 minutes in both groups. In instances where bleeding persisted beyond this duration, an additional 30 minutes of compression was administered.

The patients underwent ultrasound examination one month post procedure. The examination was performed by a skilled vascular sonographer using a GE Logiq S7 ultrasound machine (General Electric Company, Milwaukee, Wisconsin, USA). Radial artery diameter measurements were performed using two-dimensional grey-scale longitudinal images.

Radial artery lumen diameter measurements were synchronised with the R-wave peak on the electrocardiogram. Following ultrasound assessment, the patients were divided into two groups: those exhibiting > 50% stenosis in the radial artery were categorised as RAS/RAO, while those with < 50% stenosis were classified as non-RAS/RAO.

For extrapolation of WBV, both low- and high-shear stress models were calculated separately for each patient, resulting in the creation of two models per patient. WBV was calculated based on the HCT and TP concentration for both LSR ($0.5 s^{-1}$) and HSR ($208 s^{-1}$) using the established formulation developed by Simone *et al.*:[9,10]

For HSR: WBV ($208\ s^{-1}$) = (0.12 × HCT) + 0.17 (TP 2.07)

For LSR: WBV ($0.5\ s^{-1}$) = (1.89 × HCT) + 3.76 (TP 78.42)

where HCT represents haematocrit (%), TP denotes total protein concentration in g/l, WBV indicates whole blood viscosity in centipoise (cP).

Statistical analysis

Data were analysed using Windows Statistical Package For Social Sciences (SPSS) 23.0. Continuous variables are expressed as mean ± standard deviation or median (minimum–maximum) and categorical variables are expressed as number (percentage). After determining the normality of the distribution with the help of the Kolmogorov–Smirnov and Shapiro–Wilk tests, continuous variables were compared with the Mann–Whitney *U*-test or Student's *t*-test.

Categorical variables were compared with the Chi-squared test or Fisher's exact test. Independent predictors of significant RAS were found using multivariate stepwise logistic regression analysis. The multivariable model included all variables found to be significantly associated in the univariate regression analysis. Receiver operating characteristic (ROC) analysis was performed for HSR and LSR as independent predictors of significant RAS and RAO. A p-value < 0.05 was considered statistically significant.

Results

The mean age of the 215 patients included in the study sample, among whom 36.2% were female, was 61.1 ± 10.5 years years. The overall rate of patients who developed RAS/RAO was 15.3% ($n = 33$). Specifically, the rate of patients developing RAO alone was 7.4% ($n = 16$). The distribution of the demographic, clinical and angiographic characteristics of the patients by group is presented in Table 1.

Analysis of demographic data, revealed that the body mass index (BMI) was significantly lower in the group that developed RAS/RAO (25.4 vs 26.8%; $p = 0.024$). No significant differences were observed between the two groups in terms of other demographic parameters.

The distribution of the laboratory test results by group is shown in Table 2. HSR, LSR and total protein levels were notably higher in patients with RAS/RAO than in those without RAS/RAO, whereas blood urea nitrogen was significantly lower

Table 1. Demographic and clinical characteristics of patients

Variables	*RAS/RAO (+)* (n = *33*)	*RAS/RAO (–)* (n = *182*)	p-*value*
Age (years), mean ± SD	59.1 ± 11.3	61.4 ± 10.3	0.257
Females, *n* (%)	13 (39.4)	65 (35.7)	0.686
Tobacco, *n* (%)	16 (48.5)	82 (45.1)	0.716
BMI (kg/m^2), median (min–max)	25.4 (19.6–37.1)	26.8 (20.2–44.8)	0.024
Diabetes mellitus, *n* (%)	10 (30.3)	61 (33.5)	0.718
Hypertension, *n* (%)	17 (51.5)	93 (51.1)	0.965
Processed radial artery, *n* (%)			1.000
Right radial artery	33 (100.0)	177 (97.3)	
Left radial artery	0 (0.0)	5 (2.7)	
Processing time (min), median (min–max)	7.0 (3.0–15.0)	7.0 (2.0–28.0)	0.312
CAG result, *n* (%)			0.610
Normal	7 (21.2)	54 (29.7)	
Minimum CAD	12 (36.4)	58 (31.9)	
Severe CAD	14 (42.4)	70 (38.5)	
PPA use, *n* (%)	24 (72.7)	130 (71.4)	0.879
PPS use, *n* (%)	23 (69.7)	127 (69.8)	0.992

BMI, body mass index; CAG, coronary angiography; CAD, coronary artery disease; PPA, post-procedure anti-aggregant; PPS, post-procedure statin.
$p < 0.05$ shows statistical significance.

Table 2. Laboratory findings of patients

Variables	*RAS/RAO (+)* (n = *33*)	*RAS/RAO (–)* (n = *182*)	p-*value*
White blood cell count (×10^3 cells/mm^3), median (min–max)	8.4 (4.6–14.0)	7.8 (4.1–16.0)	0.071
Platelet count (×10^3 cells/mm^3), median (min–max)	256.0 (156.0–400.0)	255.0 (107.0–470.0)	0.526
Hemoglobin (g/dl), median (min–max)	14.0 (12.2–17.6)	14.0 (9.2–18.0)	0.387
Haematocrit (%), median (min–max)	41.0 (36.0–52.7)	41.0 (31.0–52.0)	0.598
Total protein (g/l), median (min–max)	69.0 (60.0–77.5)	66.0 (60.0–77.0)	< 0.001
Fasting blood glucose (mg/dl), median (min–max)	103.0 (72.0–325.0)	100.0 (76.0–319.0)	0.458
Blood urea nitrogen (mg/dl), median (min–max)	19.0 (9.6–48.0)	23.5 (8.0–89.0)	0.041
Creatinine (mg/dl), median (min–max)	0.8 (0.5–1.3)	0.8 (0.5–1.9)	0.840
HSR (208 s^{-1}), mean ± SD	16.4 ± 0.7	15.8 ± 0.6	< 0.001
LSR (0.5 s^{-1}), median (min–max)	42.8 (13.8–75.4)	31.5 (4.4–68.4)	< 0.001
AST (U/l), median (min–max)	17.0 (9.0–50.0)	19.0 (7.0–75.0)	0.503
ALT (U/l), median (min–max)	19.0 (6.0–64.0)	18.0 (5.0–72.0)	0.663
HDL-C (mg/dl), median (min–max)	43.0 (29.0–83.0)	43.0 (21.0–208.0)	0.941
LDL-C (mg/dl), mean ± SD	111.2 ± 40.8	109.6 ± 33.8	0.802
Triglycerides (mg/dl), median (min–max)	129.0 (55.0–586.0)	140.5 (49.0–590.0)	0.588
Total cholesterol (mg/dl), mean ± SD	184.1 ± 49.9	184.0 ± 41.9	0.992

AST, aspartate transaminase; ALT, alanine transaminase; HSR, high shear rate; LSR, low shear rate; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol. $p < 0.05$ shows statistical significance.

in the RAS/RAO group ($p < 0.05$). No significant differences were observed between the groups in terms of other blood parameters.

The univariate logistic regression analysis demonstrated that HSR, LSR and BMI were significantly associated with RAS/RAO (Table 3). Subsequent analysis of these variables using multivariable logistic regression indicated that BMI, HSR and LSR were independent predictors of RAS/RAO ($p < 0.05$).

The area under the ROC curve (AUC), constructed for HSR and LSR, as these were independent predictors of significant RAS and RAO, were respectively: 0.717 (0.617–0.817), $p < 0.001$; 0.732 (0.631–0.832), $p < 0.001$. HSR predicted significant stenosis and occlusion in the radial artery with a cut-off point of 16.11, 76% sensitivity and 65% specificity, while LSR predicted significant stenosis and occlusion in the radial artery with a cut-off point of 38.09, 76% sensitivity and 66% specificity (Fig. 1).

Discussion

This study has elucidated that WBV at both LSR and HSR is significantly independently linked with RAS/RAO, with markedly elevated WBV values. To our knowledge, this is the first study to explore the relationship between WBV and RAS/RAO.

WBV is a constituent of Virchow's triad and contributes to cardiovascular complications. WBV is associated with atherothrombosis, endothelial dysfunction and stasis.[11] Key factors influencing blood viscosity include plasma viscosity, HCT value, red blood cell deformability and aggregation properties.

Any modifications in these factors affect plasma viscosity, with increased viscosity exacerbating endothelial disruption at sites of mechanical stress.[12] Studies have demonstrated an association between viscosity and the risk of cardiovascular disease and mortality.[13,15]

In studies involving transradial interventions, a significant benefit has consistently been demonstrated owing to the generally high procedural success rates, lower potential for access site bleeding and increased patient comfort.[16,17] However, complications such as RAS and RAO can still occur after TRA intervention. RAO is the most prevalent postprocedural complication of TRA. While most patients remain asymptomatic with regard to ischaemia, ipsilateral TRA for future procedures is hindered. In particular, utilisation of the radial artery may be limited for coronary artery bypass grafting surgery and dialysis fistula usage.[18]

Upon reviewing the literature, the incidence of RAO development post TRA intervention was found to be

Table 3. Stepwise logistic regression models for independent predictors of significant RAS

	Univariate analysis			*Multivariate analysis*		
Variables	*OR*	*95% CI*	p-*value*	*OR*	*95% CI*	p-*value*
Model 1						
BMI	0.854	0.737–0.990	0.037	0.803	0.678–0.950	0.010
BUN	0.968	0.936–1.001	0.057			
HSR	3.445	1.925–6.165	< 0.001	3.872	2.118–7.079	< 0.001
Model 2						
BMI	0.854	0.737–0.990	0.037	0.815	0.691–0.963	0.016
BUN	0.968	0.936–1.001	0.057			
LSR	1.068	1.038–1.100	< 0.001	1.072	1.040–1.104	< 0.001

BMI, body mass index; BUN, blood urea nitrogen; HSR, high shear rate; LSR, low shear rate; OR, odds ratio; CI, confidence interval.
$p < 0.05$ shows statistical significance.

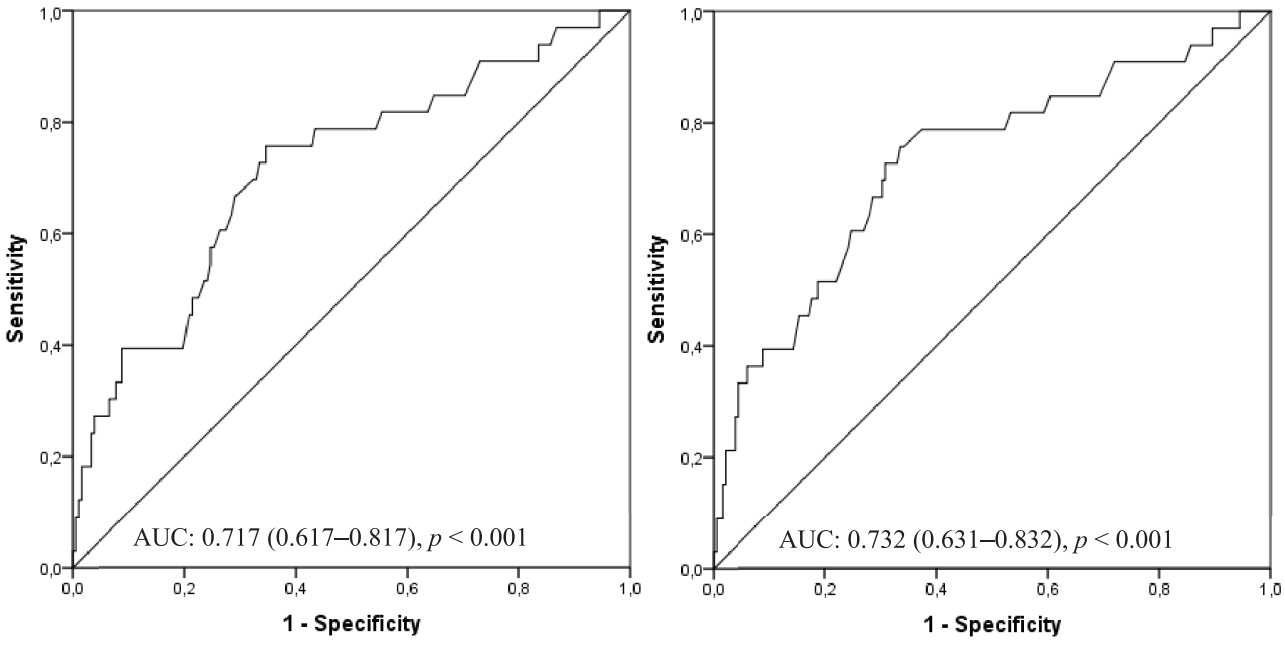

Fig. 1. Receiver operating characteristics curves for HSR and LSR for significant stenosis and occlusion in the radial artery. HSR, high shear rate; LSR, low shear rate; AUC, area under the curve.

approximately 10%;[19-21] however, this rate varies widely, with some studies reporting rates as high as 0.8 to 30%.[22] In our study, this rate was found to be approximately 7.4%, which is consistent with findings from the existing literature.

RAO pathophysiology is believed to be associated with spasm or thrombus formation. The key determinants of blood viscosity include plasma viscosity, HCT, red cell deformability and aggregation.[23] Furthermore, endothelial dysfunction in the radial artery has been documented following TRA intervention.[24]

Endothelial shear stress is defined as the stress exerted per unit area on the endothelial surface by blood flow. This force is influenced by blood viscosity. Shear stress is one of the most significant physiological stimuli for endothelial cells. Under abnormal shear stress conditions, there is an increase in the secretion of various growth factors, which can lead to arterial diameter reduction and intimal hyperplasia.[25] High shear stress primarily results in erythrocyte deformation and disaggregation, whereas low shear stress promotes erythrocyte aggregation and rouleaux formation.[26] In our study, elevated WBV values in both the LSR and HSR were identified in associated with RAS/RAO.

In a study conducted by Güneş *et al.*, WBV at both LSR and HSR was elevated in patients with deep-vein thrombosis compared to the control group. Furthermore, for the first time, WBV in both LSR and HSR was identified as an independent determinant of deep-vein thrombosis.[8] In another study involving patients with systemic lupus erythematosus (SLE), heightened levels of WBV were deemed a non-traditional risk factor for arterial events in SLE patients.[27]

In addition, Ekizler *et al.* demonstrated a significant and independent relationship between stent thrombosis and WBV. This investigation focused on the association between high WBV and the risk of stent thrombosis. Closer monitoring and stronger and/or longer-term antiplatelet and anticoagulant therapy may offer additional benefits in patients with high WBV following stent implantation.[28] Furthermore, in another study, substantial elevations in WBV levels at both HSR and LSR were found to be linked to the presence of thrombus in the left atrium and left atrial appendage in patients undergoing transoesophageal echocardiography.[29]

Limitations

First, evaluation of platelet and erythrocyte aggregability and rigidity, which could impact on blood viscosity, was not conducted. Second, we calculated WBV values only before the immediate procedure and did not incorporate subsequent WBV data.

Oxidative stress and inflammatory cytokines are important factors that contribute to endothelial dysfunction and blood viscosity. Assessing the factors influencing oxidative stress and inflammatory molecules could enhance our understanding of the results. Finally, direct measurement of blood viscosity was not performed in this study. Investigating the correlation between the calculated WBV and blood viscosity measured directly using a viscometer or haemodynamic parameters associated with endothelial shear stress would bolster the robustness of the findings.

Conclusion

WBV is a straightforward, cost-effective and easily accessible predictor of RAS/RAO, which can be determined using a basic blood test. Our study revealed a notable independent correlation between WBV and RAS/RAO. The use of a simple

formula to compute WBV from HCT and TP levels may indicate the need for alternative coronary angiography routes in these patients, facilitating the assessment of the risk of RAS/RAO development.

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