

Can Routine Blood Tests Predict Response to Drug-Coated Balloon Angioplasty? A Real-World Retrospective Analysis

Behice Hande Şişman Uzunoğlu

handesisman@gmail.com

Kırklareli University

Sezgin Uzunoğlu

Kırklareli University

Oğuzhan Abanoz

Bezmialem Foundation University Medical Faculty Hospital

Esra Danişman

Bezmialem Foundation University Medical Faculty Hospital

Mahsa Khanmohammadi

Bezmialem Foundation University Medical Faculty Hospital

Lina Boukhemis

Bezmialem Foundation University Medical Faculty Hospital

Kamran Kerimzade

Bezmialem Foundation University Medical Faculty Hospital

Mahmut Uluganyan

Bezmialem Foundation University Medical Faculty Hospital

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Abstract

Background

As a strong alternative to drug eluting stents, drug-coated balloons (DCB) are increasingly used in the treatment of coronary artery disease. However, data regarding biomarkers that predict the response to DCB therapy are lacking.

Methods

We retrospectively analyzed 95 patients with chronic coronary syndrome who underwent DCB angioplasty and follow-up angiography. Baseline laboratory parameters, including hemogram-derived inflammatory indices as well as metabolic parameters were recorded prior to the procedure. Restenosis was defined as binary restenosis ($\geq 50\%$ diameter stenosis) on follow-up angiography by using quantitative coronary angiography (QCA).

Results

A total of 95 patients treated with drug-coated balloon (DCB) angioplasty were included. Restenosis was observed in 29 patients (30.5%). In univariate analysis, hypertension, hemoglobin, RDW, and MLR were associated with restenosis. In multivariate analysis, hypertension (OR: 5.71, $p = 0.008$), RDW (OR: 1.68, $p = 0.005$), and $MLR \geq 0.24$ (OR: 8.99, $p = 0.002$) remained independently associated with restenosis.

Conclusion

Our findings suggest that MLR and RDW may be associated with restenosis after DCB angioplasty. These results support a potential role of inflammation-related pathways in vascular healing after DCB treatment, although further prospective studies are required.

Introduction

Coronary artery disease remains the leading cause of mortality worldwide despite significant advances in revascularization strategies (1). Percutaneous coronary intervention (PCI) has become the cornerstone of treatment; however, in-stent restenosis (ISR) continues to limit long-term success. However, ISR continues to limit long-term efficacy, occurring in 5–10% of cases reaching up to 10–15% in complex lesions such as chronic total occlusions (2).

Percutaneous coronary intervention triggers pathological processes that eventually lead to ISR. The major mechanisms involved in ISR include vascular inflammation due to vascular damage and metal struts, neointimal hyperplasia, and neo-atherosclerosis (3, 4). Patient-related factors such as chronic kidney

disease, diabetes mellitus, female sex, higher body mass index and older age are also predictors of ISR (5). Accordingly, increasing evidence has focused on the prognostic value of systemic inflammatory and metabolic markers. Hemogram-derived indices and lipid-related parameters - Low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), monocyte-to-lymphocyte ratio (MLR), mean platelet volume (MPV), neutrophil-to-lymphocyte ratio (NLR), platelet distribution width (PDW), platelet-to-lymphocyte ratio (PLR), red cell distribution width (RDW), systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), triglycerides (TG), triglyceride-glucose index (TyG)- have emerged as practical and cost-effective laboratory results for risk stratification in ISR (6–8).

Due to the persistent risk of in-stent restenosis (ISR), drug-coated balloons (DCB) have emerged as a promising therapeutic strategy for reducing restenosis rates (9). Drug-coated balloons (DCBs) allow effective local delivery of antiproliferative agents while avoiding permanent metallic scaffold and have recently gained increasing attention as a revascularization strategy (10). By preserving vascular integrity and minimizing chronic inflammatory stimuli, DCBs may offer advantages, especially in small vessels, bifurcation lesions, ISR, and, CTO.

Despite these promising results, data regarding biomarkers that can predict the response to DCB therapy are lacking. Identifying readily available and clinically applicable predictors may help improve patient selection and optimize treatment outcomes.

Therefore, present study aimed to evaluate predictive value of hemogram-derived indices and metabolic parameters such as NLR, PLR, SIRI, SII, MPV, RDW, TyG index and AIP in patients treated with DCB angioplasty.

Methods

Study design and population

This retrospective, single-center study included patients with chronic coronary syndrome who underwent DCB angioplasty at our institution between September 2023 and January 2025. A total of 108 consecutive patients were evaluated. Mean follow-up angiography duration was 178.3 ± 114.3 days. Most procedures were performed for de novo coronary lesions, whereas a smaller proportion were conducted for in-stent restenosis. Patients with missing laboratory data, active infection, known inflammatory or hematological disorders, malignancy, or severe hepatic dysfunction were excluded. In addition, patients without follow-up angiographic data were not included in the analysis. After exclusion 95 patients were involved in final analysis.

The study protocol was approved by the local ethics committee (decision no: 2026/203, dated 04.05.2026) and conducted in accordance with the Declaration of Helsinki.

Data collection

Baseline clinical characteristics, including age, sex, cardiovascular risk factors, and medical history, were obtained from hospital records. Laboratory parameters measured prior to the procedure were recorded.

Hemogram-derived indices were calculated from routine complete blood count parameters, including NLR, PLR, MLR, MPV, and RDW. In addition, SIRI and SII were calculated using standard formulas. Metabolic parameters, including TyG, and AIP were also assessed.

Procedure and angiographic evaluation

All procedures were performed according to current clinical practice. Lesion characteristics, target vessels, and procedural strategies were recorded. Follow-up coronary angiography findings were derived from available records. QCA measurements were obtained at baseline and during follow-up from available records to assess lesion severity using Philips Syncvision. Binary restenosis was defined as $\geq 50\%$ diameter stenosis in the target lesion on follow-up angiography.

Statistical analysis

Statistical analyses were performed using [SPSS version 27, IBM Corp., Armonk, NY, USA]. Continuous variables were expressed as mean $\pm$ standard deviation or median (interquartile range), depending on data distribution. Categorical variables were presented as counts and percentages.

Comparisons between groups (restenosis vs. no restenosis) were performed using the independent samples t-test or Mann–Whitney U test for continuous variables, and the chi-square test for categorical variables.

Univariate logistic regression analysis was used to identify variables associated with restenosis. Variables with a p-value < 0.10 in univariate analysis were included in multivariate logistic regression models to determine independent predictors. Results were presented as odds ratios (OR) with 95% confidence intervals (CI).

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the discriminative ability of significant parameters and to determine optimal cut-off values. A two-sided p-value < 0.05 was considered statistically significant.

Results

A total of 95 patients who underwent DCB angioplasty were included in the study. During follow-up, restenosis ($\geq 50\%$ diameter stenosis) was observed in 29 patients (30.5%).

Baseline clinical, biochemical, and hematological characteristics of the study population according to restenosis status are presented in Table 1. There were no significant differences between the groups in terms of age, sex, body mass index, or most cardiovascular risk factors. However, hypertension was

more common in patients who developed restenosis (82.8% vs. 75.8%, $p = 0.025$). Diabetes mellitus was also more frequent in the restenosis group, although it did not reach statistical significance (65.5% vs. 45.5%, $p = 0.072$).

Table 1
Baseline characteristics of the study population according to binary restenosis status

Parameters	Binary Stenosis			
	<%50	>%50	Total	p-value
Age	59.41 ± 11.98	60.62 ± 9.287	59.78 ± 10.836	0.618
Sex (Male), n (%)	50 (75.8)	20 (69.0)	70	0.489
BMI	28.27 ± 3.94	29.62 ± 3.75	28.68 ± 3.91	0.631
HT, n (%)	39 (59.1)	24 (82.8)	63 (66.3)	0.025
DM, n (%)	30 (45.5)	19 (65.5)	49 (51.6)	0.072
PCI, n (%)	27 (40.9)	15 (51.7)	42 (44.2)	0.328
CABG, n (%)	2 (3.0)	3 (10.3)	5 (5.3)	0.141
ASA, n (%)	45 (68.2)	23 (79.3)	68 (71.6)	0.268
P2Y12, n (%)	34 (51.5)	14 (48.3)	48 (50.5)	0.771
Statin, n (%)	47 (71.2)	21 (72.4)	68 (71.6)	0.905
OAD, n (%)	23 (34.8)	14 (48.3)	37 (38.9)	0.216
Insulin, n (%)	2 (3.0)	2 (6.9)	4 (4.2)	0.388
Smoking, n (%)				0.339
Never	22 (36.1)	14 (53.8)	36 (41.3)	
Current	29 (47.5)	7 (26.9)	36 (41.3)	
Ex	10 (16.4)	5 (19.2)	15 (17.4)	
Family history, n (%)	35 (53.0)	17 (60.7)	52 (55.3)	0.726
Creatinine	0.97 ± 0.21	0.95 ± 0.31	0.96 ± 0.25	0.174
Glucose	140.04 ± 69.73	128.43 ± 57.89	136.38 ± 66.05	0.285
LDL-C	118.3 ± 37.6	107.9 ± 34.2	115.2 ± 36.8	0.084
Triglyceride	148.45 ± 67.82	165.16 ± 113.83	154.02 ± 85.24	0.490
HDL-C	46.2 ± 10.2	50.6 ± 12.1	47.6 ± 10.9	0.170
Troponin	1072.08 ± 2682.33	535.68 ± 846.43	908.34 ± 2291.35	0.477
Neutrophil	5.29 ± 1.82	4.94 ± 1.30	5.18 ± 1.68	0.370
Lymphocyte	2.46 ± 0.94	2.12 ± 0.79	2.35 ± 0.91	0.097
Hemoglobin	13.5 ± 1.4	12.8 ± 1.4	13.3 ± 1.4	0.030

Parameters	Binary Stenosis			
	<%50	>%50	Total	p-value
RDW	13.7 ± 1.2	14.6 ± 2.0	13.9 ± 1.5	0.026
Platelet	272.7 ± 71.6	277.5 ± 97.0	274.2 ± 79.7	0.734
MPV	10.4±.8	10.5 ± 1.1	10.4±.9	0.599
PDW	11.50 ± 1.26	11.98 ± 1.98	11.65 ± 1.53	0.242
Monocyte	0.62 ± 0.20	0.71 ± 0.23	0.65 ± 0.21	0.048
NLR	2.49 ± 1.56	2.67 ± 1.34	2.55 ± 1.49	0.190
PLR	123.36 ± 46.87	139.14 ± 6.68	128.18 ± 50.29	0.090
TyG	8.97 ± 0.95	9.08 ± 0.80	9.01 ± 0.90	0.813
AIP	0.48 ± 0.23	0.42 ± 0.20	0.46 ± 0.22	0.351
SII	664.01 ± 403.27	689.53 ± 364.82	671.61 ± 390.44	0.774
MLR	0.28 ± 0.13	0.36 ± 0.14	0.31 ± 0.14	0.001
SIRI	1.58 ± 1.24	1.79 ± 0.79	1.64 ± 1.13	0.037
EF	57.32 ± 6.04	55.39 ± 7.13	56.70 ± 6.43	0.196

AIP, Atherogenic Index of Plasma; BMI, Body Mass Index; CABG, Coronary Artery Bypass Grafting; CVE, Cerebrovascular Event; DM, Diabetes Mellitus; EF, Ejection Fraction; HDL-C, High-Density Lipoprotein Cholesterol; HT, Hypertension; LDL-C, Low-Density Lipoprotein Cholesterol; MLR, Monocyte-to-Lymphocyte Ratio; MPV, Mean Platelet Volume; NLR, Neutrophil-to-Lymphocyte Ratio; OAD, Oral Antidiabetic Drugs; PCI, Percutaneous Coronary Intervention; PDW, Platelet Distribution Width; PLR, Platelet-to-Lymphocyte Ratio; RDW, Red Cell Distribution Width; SII, Systemic Immune-Inflammation Index; SIRI, Systemic Inflammation Response Index; TG, Triglycerides; TyG, Triglyceride-Glucose Index.

Among laboratory parameters, hemoglobin levels were lower in patients with restenosis (12.8 ± 1.4 vs. 13.5 ± 1.4 g/dL, $p = 0.030$), whereas RDW values were higher (14.6 ± 2.0 vs. 13.7 ± 1.2 , $p = 0.026$). Monocyte count was also higher in the restenosis group (0.71 ± 0.23 vs. 0.62 ± 0.20 , $p = 0.048$). In terms of inflammatory indices, MLR (0.36 ± 0.14 vs. 0.28 ± 0.13 , $p = 0.001$) and SIRI (1.79 ± 0.79 vs. 1.58 ± 1.24 , $p = 0.037$) were significantly higher in patients with restenosis. Other hematological and biochemical parameters, including MPV, NLR, PLR, TyG, AIP, and SII, were comparable between the groups.

Receiver operating characteristic (ROC) curve analysis demonstrated that MLR had the highest discriminative ability among the evaluated markers (AUC: 0.709), followed by SIRI (AUC: 0.637), RDW (AUC: 0.626), and PLR (AUC: 0.609) (Fig. 1, Table 2). The optimal cut-off value for MLR was 0.24, with a sensitivity of 96.6% and specificity of 36.9%.

Table 2
Optimal Cut-off Values and Diagnostic Performance of Inflammatory Markers for
Predicting Binary Restenosis

Variable	AUC	Cut-off	Sensitivity (%)	Specificity (%)	Youden Index
RDW	0.626	13.0	82.1	27.7	0.098
PLR	0.609	90.4	86.2	21.2	0.074
MLR	0.709	0.24	96.6	36.9	0.335
SIRI	0.637	0.81	96.4	20.0	0.164

MLR, Monocyte-to-Lymphocyte Ratio; RDW, Red Cell Distribution Width, PLR, Platelet-to-Lymphocyte Ratio; SIRI, Systemic Inflammation Response Index.

Univariate logistic regression analysis identified hypertension (OR: 3.32, 95% CI: 1.13–9.80, $p = 0.029$), hemoglobin (OR: 0.70, 95% CI: 0.50–0.97, $p = 0.033$), RDW (OR: 1.50, 95% CI: 1.10–2.04, $p = 0.010$), and MLR (OR: 56.53, 95% CI: 2.01–1591.26, $p = 0.018$) are associated with restenosis (Table 3). When analyzed as categorical variable, $MLR \geq 0.24$ was also strongly associated with restenosis (OR: 7.90, 95% CI: 2.17–28.72, $p = 0.002$). Monocyte count showed borderline significance ($p = 0.054$).

Table 3
Univariate logistic regression analysis for predictors of binary restenosis

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
YAS	1.010	0.970–1.052	0.614
HT	3.323	1.127–9.798	0.029
Glucose	0.997	0.989–1.005	0.486
LDL-C	0.992	0.980–1.004	0.205
Triglyceride	1.002	0.996–1.009	0.488
HDL-C	1.037	0.987–1.090	0.152
Neutrophil	0.873	0.655–1.163	0.353
Lymphocyte	0.627	0.362–1.087	0.096
Hemoglobin	0.698	0.501–0.972	0.033
RDW	1.496	1.100–2.035	0.010
Platelet	1.001	0.995–1.006	0.786
MPV	1.134	0.713–1.806	0.595
PDW	1.226	0.918–1.636	0.168
Monocyte	7.946	0.969–65.140	0.054
NLR	1.081	0.812–1.439	0.593
PLR	1.006	0.998–1.015	0.164
TyG	1.160	0.604–2.228	0.656
AIP	0.280	0.020–3.941	0.345
SII	1.000	0.999–1.001	0.771
MLR	56.525	2.008–1591.256	0.018
MLR $\geq$ 0.24	7.902	2.174–28.718	0.002
SIRI	1.171	0.803–1.707	0.413

AIP, Atherogenic Index of Plasma; HDL-C, High-Density Lipoprotein Cholesterol; HT, Hypertension; LDL-C, Low-Density Lipoprotein Cholesterol; MLR, Monocyte-to-Lymphocyte Ratio; MPV, Mean Platelet Volume; NLR, Neutrophil-to-Lymphocyte Ratio; PDW, Platelet Distribution Width; PLR, Platelet-to-Lymphocyte Ratio; RDW, Red Cell Distribution Width; SII, Systemic Immune-Inflammation Index; SIRI, Systemic Inflammation Response Index; TG, Triglycerides; TyG, Triglyceride-Glucose Index.

To further evaluate independent predictors, two multivariate logistic regression models were constructed (Table 4). In the first model, where MLR was included as a continuous variable, hypertension (OR: 5.69, $p = 0.007$) and RDW (OR: 1.59, $p = 0.010$) remained independently associated with restenosis, while MLR showed borderline significance ($p = 0.053$). In the second model, using a categorical definition of MLR based on ROC analysis, $MLR \geq 0.24$ emerged as an independent predictor of restenosis (OR: 8.99, $p = 0.002$), together with hypertension (OR: 5.71, $p = 0.008$) and RDW (OR: 1.68, $p = 0.005$).

Table 4
Multivariate Logistic Regression Analysis

Model 1 (continuoss MLR)	Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
	Hypertension	5.694	1.603–20.225	0.007
	RDW	1.594	1.119–2.269	0.009
	MLR	38.557	0.953–1560.436	0.053
Model 2 (categorical MLR)	Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
	Hypertension	5.71	1.59–20.55	0.008
	RDW	1.68	1.17–2.42	0.005
	$MLR \geq 0.24$	8.99	2.21–36.57	0.002

MLR, Monocyte-to-Lymphocyte Ratio; RDW, Red Cell Distribution Width

Discussion

In this study, we evaluated the association between routinely available hematological and metabolic parameters and restenosis following DCB angioplasty. The main findings can be summarized as follows: (i) MLR, particularly when analyzed using a cut-off value (≥ 0.24), was strongly associated with restenosis and emerged as an independent predictor in multivariate analysis; (ii) RDW also showed an independent association supporting the role of chronic inflammatory burden; and (iii) commonly used inflammatory markers such as NLR, PLR, SIRI, and metabolic indices such as TyG and AIP were not associated with restenosis in this cohort. These findings suggest that monocyte-centered inflammatory pathways may play a more dominant role in restenosis after DCB therapy. To the best of our knowledge, data regarding the predictive value of these biomarkers in patients treated with DCB is limited. Hypertension also retained its significance as a clinical determinant.

Inflammation plays a central role in the pathophysiology of restenosis. Many studies showed that inflammatory markers such as CRP, tumor necrosis factor- α , were elevated in patients with ISR (11). Vascular injury induced by PCI initiates a cascade involving endothelial dysfunction, cytokine release, and recruitment of smooth muscle cells, T lymphocytes, and macrophages, ultimately leading to

neointimal proliferation and restenosis (12). Among circulating cells, monocytes are particularly important, as they contribute to myocardial repair, myofibroblast proliferation, and angiogenesis (13). MLR reflects not only inflammatory activation but also immune regulation, making it a more integrative marker compared to isolated inflammatory indices. Sani Namik Murat et al. demonstrated a direct association between the elevated MLR values and the risk of ISR in patients with stable coronary artery disease treated with bare-metal stents (14). Our findings extend this evidence by demonstrating that this relationship is also valid in patients with chronic coronary syndrome treated with DCB.

The present study demonstrated that MLR is significantly associated with restenosis following DCB angioplasty. This finding is consistent with previous reports in stented populations, where inflammation-related markers have been shown to predict neointimal proliferation and adverse vascular remodeling. However, evidence regarding patients treated with DCB remains limited. Unlike stent-based therapies, DCB angioplasty does not involve permanent metallic implantation and may therefore attenuate chronic inflammatory stimulation. Nevertheless, vascular injury induced during the procedure and the subsequent healing response remain closely linked to the systemic inflammatory environment (15). Persistent inflammatory activation may promote smooth muscle cell proliferation and extracellular matrix deposition, ultimately contributing to restenosis even in the absence of a permanent scaffold. These mechanisms may explain why inflammation-related markers, particularly MLR, retain their predictive value in patients undergoing DCB treatment. The high sensitivity of MLR suggests that it may be useful as a screening marker rather than a definitive diagnostic tool.

Another important finding of our study was the independent association between RDW and restenosis. Inflammatory processes can impair erythropoiesis and shorten erythrocyte lifespan, leading to increased heterogeneity in red blood cell size. Consequently, RDW is recognized as a readily available marker of chronic inflammation (16). Therefore, elevated RDW may reflect an unfavorable biological environment that predisposes to restenosis. Consistent with our findings in DCB treated patients, Zhao et al RDW was independent predictor of ISR in stable angina pectoris patients with coronary DES implantation, suggesting that a chronic inflammatory response might be involved in DES restenosis pathogenesis (7).

Interestingly, commonly used inflammatory markers such as NLR, PLR, and SIRI were not associated with restenosis in our study. This finding suggests that these indices may primarily reflect acute or nonspecific inflammatory responses rather than sustained, low-grade inflammation. In contrast, MLR and RDW may better capture chronic inflammatory activity and immune dysregulation, which are more relevant to vascular healing and neointimal formation (17, 18). These results indicate that not all inflammatory pathways contribute equally to restenosis and highlight the importance of pathway-specific biomarkers.

Previous studies have reported an association between various blood cell-derived parameters and restenosis. In a study by Zhichao Wang et al., multiple hemogram-derived markers were found to be associated with in-stent restenosis, supporting the role of systemic inflammation in vascular outcomes (19). Similarly, Chenguang Li et al. demonstrated that preprocedural inflammatory and metabolic

parameters were predictive of restenosis in patients with chronic total occlusion lesions treated with drug-eluting stents (20). However, stent-based interventions are associated with persistent local inflammatory stimuli due to permanent metallic scaffolds and polymer coatings. In contrast, DCB therapy eliminates these factors, potentially altering the relative contribution of systemic versus local inflammation. This difference may explain why broader inflammatory indices were found to be significant in stent-based studies, whereas more specific markers such as MLR and RDW emerged as predictors in our DCB cohort.

In the present study, hypertension emerged as an independent predictor of restenosis, whereas other common comorbidities such as diabetes mellitus and chronic kidney disease were not significantly associated with the outcome. The association between hypertension and restenosis is well established and may be explained by increased vascular wall stress, endothelial dysfunction, and enhanced smooth muscle cell proliferation, all of which contribute to neointimal hyperplasia (21). Consistent with this, prior studies have demonstrated that hypertension promotes adverse vascular remodeling and impairs endothelial healing following percutaneous coronary interventions.

Although diabetes mellitus and chronic kidney disease are recognized risk factors for atherosclerosis and adverse cardiovascular outcomes, their relationship with restenosis has been inconsistent across studies, particularly in cohorts with limited sample size or specific lesion and treatment characteristics (22, 23). This variability may be related to differences in metabolic control, disease severity, and concomitant therapies. Therefore, the lack of a significant association in our study may reflect population-specific factors and limited statistical power rather than a true absence of effect.

In conclusion, our study suggests that MLR and RDW are associated with restenosis after DCB angioplasty, highlighting the potential role of inflammation-related pathways in vascular healing. Further large-scale, prospective studies are needed to better define the clinical utility of these markers.

Limitations

This study has several limitations. First, its retrospective and single-center design limits the generalizability of the findings. Second, the relatively small sample size may have affected statistical power and the stability of the regression models. Third, only baseline laboratory parameters were evaluated, and changes over time were not assessed. Finally, the follow-up period was limited to approximately 6 months, and longer-term clinical outcomes were not available.

The binary restenosis rate observed in this study (30.5%) was higher than several contemporary DCB studies. However, this study was not designed to evaluate the incidence of restenosis among all patients treated with DCB in our clinic. Of 296 patients who underwent DCB treatment during the study period, only 95 patients with available follow-up coronary angiography were included. Therefore, the study population represents a selected angiographic follow-up cohort. This selection process may have

enriched the cohort with patients at higher risk for restenosis and should be considered when interpreting the observed restenosis rate.

Conclusion

In this study, monocyte-to-lymphocyte ratio and red cell distribution width were found to be associated with restenosis following drug-coated balloon angioplasty. Unlike stent-based studies, our findings specifically reflect DCB-treated populations, where the absence of permanent metallic scaffolds may alter the underlying biology of restenosis. These findings suggest that inflammation-related mechanisms may play an important role in vascular healing after DCB treatment. Although the predictive performance of these markers is modest, they may contribute to risk stratification in daily clinical practice. Larger prospective studies are needed to confirm these results.

Declarations

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Due to retrospective nature of the study, requirement for informed consent was waived by the Ethics Committee.

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****** Declaration of originality, authorship and competing interest on behalf of all authors of the manuscript.

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Persons appointed as authors meet all the criteria of authorship.

Author Contribution

Behice Hande Şişman Uzunoğlu wrote the Introduction and Discussion sections. Sezgin Uzunoğlu performed the statistical analyses and wrote the Materials and Methods and Results sections of the manuscript. Oğuzhan Abanoz prepared the tables and figures. Esra Danışman and Mahsa Khanmohammadi performed the angiographic measurements and data verification. Lina Boukhemis and Kamran Kerimzade collected and curated the demographic and clinical data. Mahmut Uluganyan conceived and supervised the study, contributed to the literature review, critically revised the manuscript for important intellectual content, and approved the final version. All authors reviewed and approved the final manuscript.

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Figures

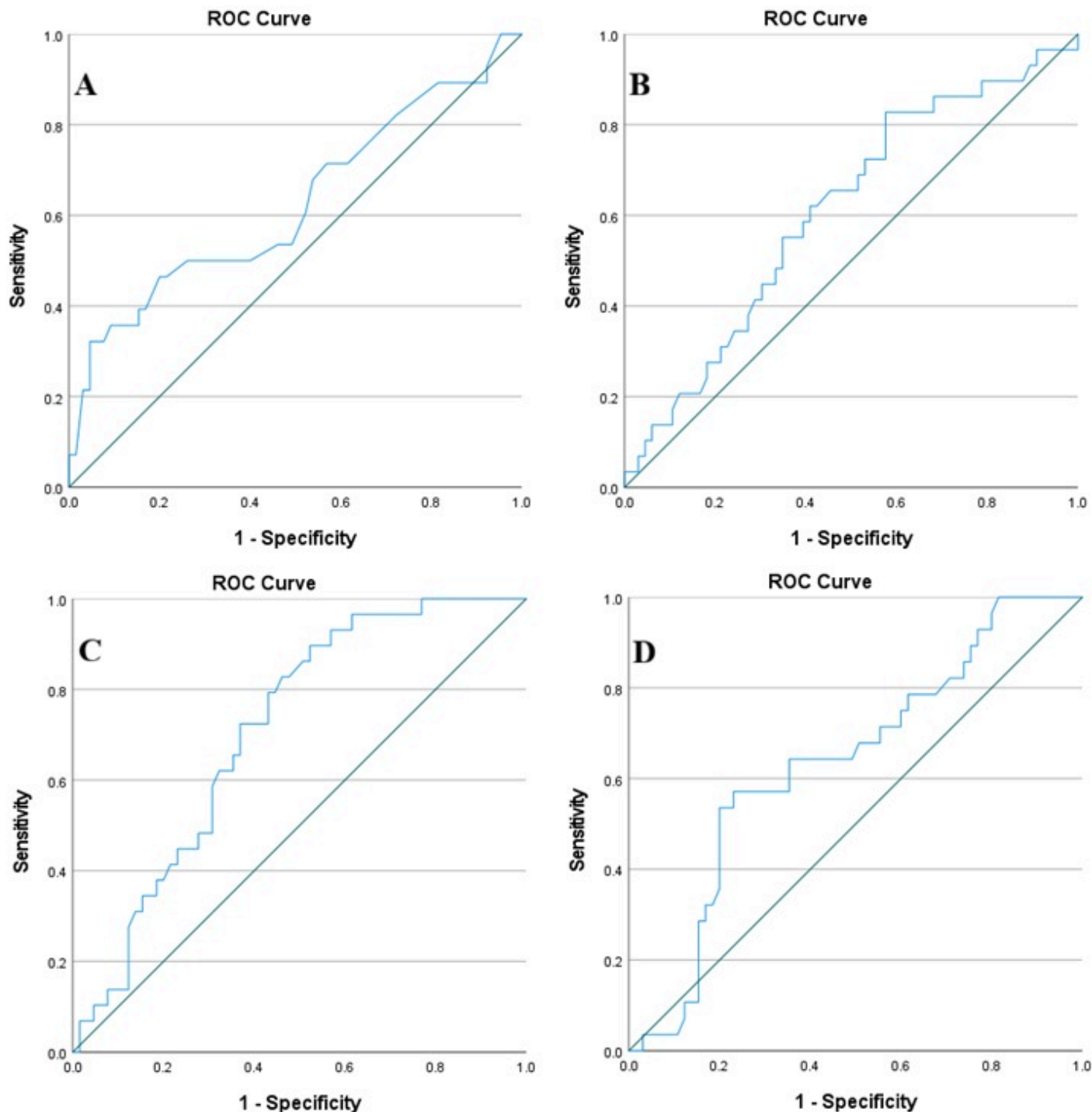


Figure 1

ROC Curve analysis of A: RDW, AUC: 0.626; B: PLR, AUC: 0.609; C: MLR, AUC: 0.709; D: SIRI AUC: 0.637.

Supplementary Files

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