Mustafa Kaplangoray[1], Kenan Toprak[2], Cihan Aydın[3],
Yusuf Cekici[4], Arafat Yildirim[4], Ozge Ozcan Abacioglu[4]

[1] Bilecik Şeyh Edebali University Faculty of Medicine Department of Cardiology, Bilecik, Turkey

[2] Republic of Turkey Ministry of Health Siverek State Hospital, Cardiology Department, Sanlıurfa, Turkey

[3] Tekirdag Namık Kemal University, Department of Cardiology, Tekirdağ, Turkey

[4] University of Health Sciences, Adana Health Practice and Research Center, Department of Cardiology, Adana, Turkey

The MAPH Score Predicts Coronary Slow Flow. A Retrospective Case-Controlled Study

Aim	The MAPH score is a new score that combines mean platelet volume (MPV), hematocrit, and total protein, which are markers of whole blood viscosity (WBV). We aimed to investigate the relationship between the MAPH score and the coronary slow flow phenomenon (CSF).
Material and methods	A total of 201 patients were included in the study. 105 had CSF and 96 had normal coronary flow (NCF). Coronary flow was measured by the Thrombolysis in Myocardial Infarction frame count (TFC) method. The patients' MPV, age, hematocrit, and total protein were recorded. High (HSR) and low shear rates (LSR) were calculated, based on total protein and hematocrit values. Cut-off values for CSF were determined using the Youden's index, and the score was determined as 0 or 1 according to the cut-off values. The sum of these scores was the MAPH score.
Results	The mean age of the patients included in the study was 51.1±7.9 (n=201, 54.2% male). Hyperlipidemia, DM, and HT rates of both groups were similar, but the mean age of the CSF group was higher (p=0.773; p=0.549; p=0.848; p <0.001, respectively). Total protein, MPV,hematocrit, HSR and LSR were higher in the CSF group (p< 0.001, for all values). Comparative receiver operating characteristic (ROC) curve analysis showed that the performance of the MAPH score in predicting CSF is better than the performance of these parameters separately.
Conclusion	A new score, the MAPH score, may be used to identify the presence of CSF
Keywords	Coronary slow flow; MAPH score; shear rate; TIMI frame count; whole blood viscosity
For citations	Mustafa Kaplangoray, Kenan Toprak, Cihan Aydın, Yusuf Cekici, Arafat Yildirim, Ozge Ozcan Abacioglu. The MAPH Score Predicts Coronary Slow Flow. A Retrospective Case-Controlled Study. Kardiologiia. 2024;64(2):67–72. [Russian: Мустафа Каплангорай, Кенан Топрак, Джихан Айдын, Юсуф Чекиджи, Арафат Йылдырым, Озге Озджан Абаджиоглу. Оценка по шкале MAPH предсказывает замедление коронарного кровотока. Ретроспективное исследование случай-контроль. Кардиология. 2024;64(2):67–72].
Corresponding author	Mustafa Kaplangoray. Email: mkaplangoray@gmail.com

Introduction

The incidence of coronary slow flow phenomenon (CSF) is 1–7% in patients undergoing coronary angiography for anginal complaints [1, 2]. CSF is characterized by delayed, opaque flow into the distal portion of normal epicardial vessels in the absence of heart failure, connective tissue disease, valvular heart disease, coronary spasm, and coronary ectasia [3, 4]. Despite numerous studies in this field, the underlying mechanism of CSF remains unclear. The main factors suggested in the pathogenesis of CSF are endothelial dysfunction [5], microvascular abnormalities [6], occult atherosclerosis, and inflammation. Previous studies have demonstrated a relationship of SCF with whole blood viscosity, as calculated from total protein and hematocrit values [7], and with mean platelet volume (MPV) [8] and with age [9].

Abacioğlu et al [10] demonstrated that the MAPH score, computed with the combination of total protein, hematocrit, MPV, and age values, performed better than these parameters independently in identifying a high thrombus burden in patients with ST elevation myocardial infarction (STEMI). To our knowledge, no study has investigated the relationship between the MAPH score and CSF. The MAPH score includes those parameters that are considered as factors responsible for CSF pathogenesis. Thus, the aim of this study was to investigate the relationship between the MAPH score and CSF.

Material and methods

Patient population

Approximately 2500 patients who underwent diagnostic coronary angiography at our institution were retrospectively screened to identify CSF patients for this observational, case-controlled, and comparative study. The indication for coronary angiography was the presence of typical angina pectoris and equivalent symptoms or positive

results of noninvasive stress tests to investigate myocardial ischemia. For the study, 105 CSF patients and 96 control patients with angiographically proven normal coronary artery with normal coronary flow (NCF) were enrolled. Patients were excluded if any of the following conditions were present: CSF secondary to percutaneous coronary angioplasty after myocardial infarction or coronary bypass surgery, significant organic valvular heart disease, congestive heart failure, congenital heart disease, atrial fibrillation, hypo/hyperthyroidism, any collagen vascular disease, any hematologic disease, any autoimmune and neoplastic disease, chronic renal or hepatic insufficiency (aspartate transaminase or alanine transaminase values more than three times higher than normal), active infection, use of anticoagulant drug.

Patients receiving antihypertensive treatment were considered as hypertensive (HT), and the diagnosis of hyperlipidemia was defined according to the criteria of the European Society of Cardiology guidelines [11]. Diabetes mellitus (DM) was identified if the patient had been diagnosed with diabetes and was taking antidiabetic medications or if the patient, who was unaware of their diabetes status, had high blood glucose according to the American Diabetes Association's criteria [12]. Left ventricular ejection fraction was obtained from echocardiographic recordings performed before coronary angiography. The study was conducted in accordance with the principles of the Helsinki Declaration, and the study protocol was approved by the Ethics Committee of Harran University Faculty of Medicine.

Cardiac catheterization and participant data

The coronary angiography procedure was performed by the Judkins technique via the femoral or radial route. Angiographic procedures were performed by experienced cardiologists blinded to the study data and design. The coronary frame count of all participants was performed according to the Thrombolysis in Myocardial Infarction (TIMI) frame count (TFC) calculation described by Aciksari G et al. [13] The number of frames obtained for each vessel was multiplied by two since angiographic recordings are performed at 15 frames/second in our clinic. Since the time taken for opacification was longer due to the length of the LAD, the TFC value calculated for the LAD was divided by 1.7 to obtain the correct TFC (c-TFC) value. As previously reported, c-TFC threshold values were accepted as 36.2±2.6 frames for the LAD artery, 22.2±4.1 frames for the Cx artery, and 20.4±3.0 frames for the RCA artery [13]. CSF was diagnosed in patients with a TFC greater than two standard deviations (SD) above the specified thresholds in any of the three vessels. The mean c-TFC value for the patient and control groups was obtained by dividing the sum of the c-TFC values counted for the LAD, Cx, and RCA arteries by three.

Analysis of hematologic and biochemical parameters

Routine biochemical and hematological data were obtained from the records of the examinations performed before coronary angiography. Hematocrit, total protein, MPV, CRP, serum albumin, lipid parameters, liver and kidney function tests were analyzed using the appropriate kit. Whole blood viscosity (WBV) was calculated from hematocrit and total protein concentration using the validated formula [14] for both low shear rate (LSR) (0.5 s-1) and high shear rate (HSR) (208 s-1). Cut-off values for MPV, total protein, age, and hematocrit parameters for high TCF were determined using the Youden index [15, 16]. Values higher than the cut-off value were accepted as 1 point, and the MAPH score was calculated by summing the scores between 0 and 1 that were obtained according to the cut-off values determined for each variable.

Statistical Analysis

The Kolmogorov-Smirnov test was used to determine the normality of data distributions. Normally distributed, continuous variables are shown as mean±SD. For normally distributed data, the significance of differences between group means was determined by Student's t-tests. For non-normally distributed data, the Kruskal-Wallis test was used. Categorical data are presented as numbers and percentages. Chi-square tests were used to compare categorical data. Correlations between variables were examined with Spearman's correlation. Receiver operating characteristic (ROC) curve analysis and the Youden index were used to determine the sensitivity and specificity values for MPV, age, total protein, and hematocrit values. Pairwise comparison of ROC curve analysis was used to evaluate the discriminative power of variables for slow coronary flow with two models, first the MAPH score and the consisting variables and second the MAPH score and the most frequently used blood viscosity markers.

Results

The mean age of all patients was 51.1±7.9 yrs, and 109 (54.2%) were male. 105 patients had CSF (mean age 52.7±8.5 yrs. 57.1% male). The NCF group consisted of 96 patients (mean age 49.2±6.7 yrs, 51% male)]. The baseline characteristics and laboratory data of the groups are summarized in Table 1. The patients of both groups were similar in terms of gender and the frequency of HT, hyperlipidemia, and DM (p=0.848; p=0.773; p=0.549, respectively), but the patients with CSF were older and ratio of smokers were higher

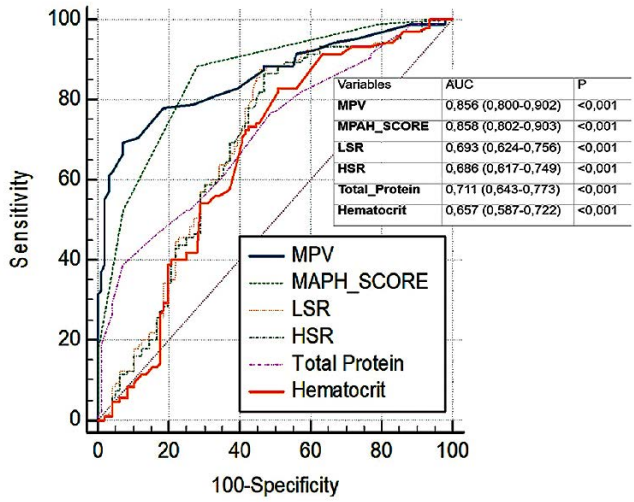
ISSN 0022-9040. Кардиология. 2024;64(2). DOI: 10.18087/cardio.2024.2.n2322

Table 1. Characteristics and laboratory values of the CSF and NCF groups

Variable	CSF (n=105)	NCF (n=96)	p
Demographics			
Age, yrs	52.7±8.5	49.2±6.7	<0.001
Gender, male	60 (57.1)	49 (51)	0.386
Comorbidities			
HT	38 (36.2)	36 (37.5)	0.848
DM	33 (31.4)	34 (35.4)	0.549
Hyperlipidemia	43 (41)	37 (38.9)	0.773
Smoking	59 (56.2)	33 (34.4)	0.002
Medications			
Aspirin	36 (34.3)	22 (22.9)	0.076
B-Blocker	24 (22.9)	12 (12.5)	0.056
ACEİ/ARB	34 (32.4)	26 (27.1)	0.412
CCB	16 (15.2)	11 (11.4)	0.432
Statins	18 (17.1)	16 (16.7)	0.928
Laboratory measurements			
Glucose, mg/dl	136.3±83	124.5±60.9	0.258
Urea, mg/dl	31.5±11.2	30.3±10.4	0.453
Creatinine, mg/dl	0.87±0.15	0.91±0.57	0.511
LDL, mg/dl	126.5±32.9	117.4±34.2	0.056
HDL, mg/dl	47.6±6.7	45±11.8	0.059
Total cholesterol, mm/dl	208.2±35.7	193.8±45.5	0.013
Triglyceride, mg/dl	209.3±88.8	186.4±113.2	0.111
HGB, gr/dl	13.9±1.8	13.3±1.7	0.07
WBC, 10^3/ml	9.6±9.4	8.3±1.8	0.201
Platelets, 10^3/ml	179.2±42.3	177.6±40.3	0.148
Hematocrit, %	42.1±3	40.1±4.4	<0.001
MPV, fl	10.9±1	9.5±0.6	<0.001
Total protein, g/l	7.4±0.3	7.1±0.3	<0.001
Albumin, g/l	4.3±0.3	4.2±0.6	0.341
MAPH score	2.57±0.9	1.1±0.8	<0 .001
HSR	4.2±0.3	3.9±0.5	<0 .001
LSR	29.2±6.1	24.2±8.7	<0 .001
Angiographic measurements			
LAD c-TFC	39.3±11.4	18.9±5.5	<0 .001
LCX c-TFC	28.4±8.0	17.5±11	<0 .001
RCA c-TFC	32.7±10.3	18.1±6.7	<0 .001
Mean c-TFC	33.5±6.6	18.2±5.5	<0 .001

Data are mean±SD or number (percentage). HT, hypertension; DM, diabetes mellitus; LDL, low density lipoprotein cholesterol; HDL, high density lipoprotein cholesterol; HGB, hemoglobin; WBC, white blood cell count; MPV, mean platelet volume; MAPH, MPV+age+total protein+hematocrit; HSR, high shear rate; LSR, low shear rate; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blockers; LAD, left anterior descending coronary artery; LCX, circumflex coronary artery; RCA, right coronary artery; c-TFC, corrected TIMI frame count.

Figure 1. ROC curve analysis of variables for coronary slow flow. MPV, mean protein volume; LSR, low shear rate; HSR, high shear rate



(p<0.001; p=0.002, respectively). Total protein, MPV, and hematocrit were significantly higher in the CSF group (p <0.001; p<0.001; p <0001, respectively). HSR, LSR, and MAPH scores were significantly higher in the CSF group.

The most significant correlation with CSF was the MAPH score (Table 2) (r=0.583, p<0.001). MPV, total protein, hematocrit, and age cut-off values that predicted slow coronary slow were calculated according to ROC analysis and Youden index: for age >52 yrs – AUC 0.626, sensitivity 52.3%, specificity 70,8%; for hematocrit >39.4% – AUC 0.657, sensitivity 82.8%, specificity 48.9%; for MPV >10.3 fl – AUC 0.856, sensitivity 69.5%, specificity 92.7%; for total protein >7.5 g/l – AUC 0.711, sensitivity 39%, specificity 92.7%; for MAPH score >1 – AUC 0.858, sensitivity 88.5%, specificity 71.8% (Figures 1). Pairwise comparison of ROC curve analysis demonstrated that the MAPH score had better performance compared to the self containing parameters of MAPH score and to the HSR and LSR values for predicting CSF (Table 3, Figure 2).

Table 2. Results of correlation analysis of mean c-TFC with relevant variables

Correlation	Rho	p
Mean c-TFC – total protein	0.394	<0.001
Mean c-TFC – hematocrit	0.321	<0.001
Mean c-TFC–MPV	0.521	<0.001
Mean c-TFC–MAPH score	0.583	<0.001
Mean c-TFC – age	0.179	0.011
Mean c-TFC – HSR	0.362	<0.001
Mean c-TFC–LSR	0.375	<0.001

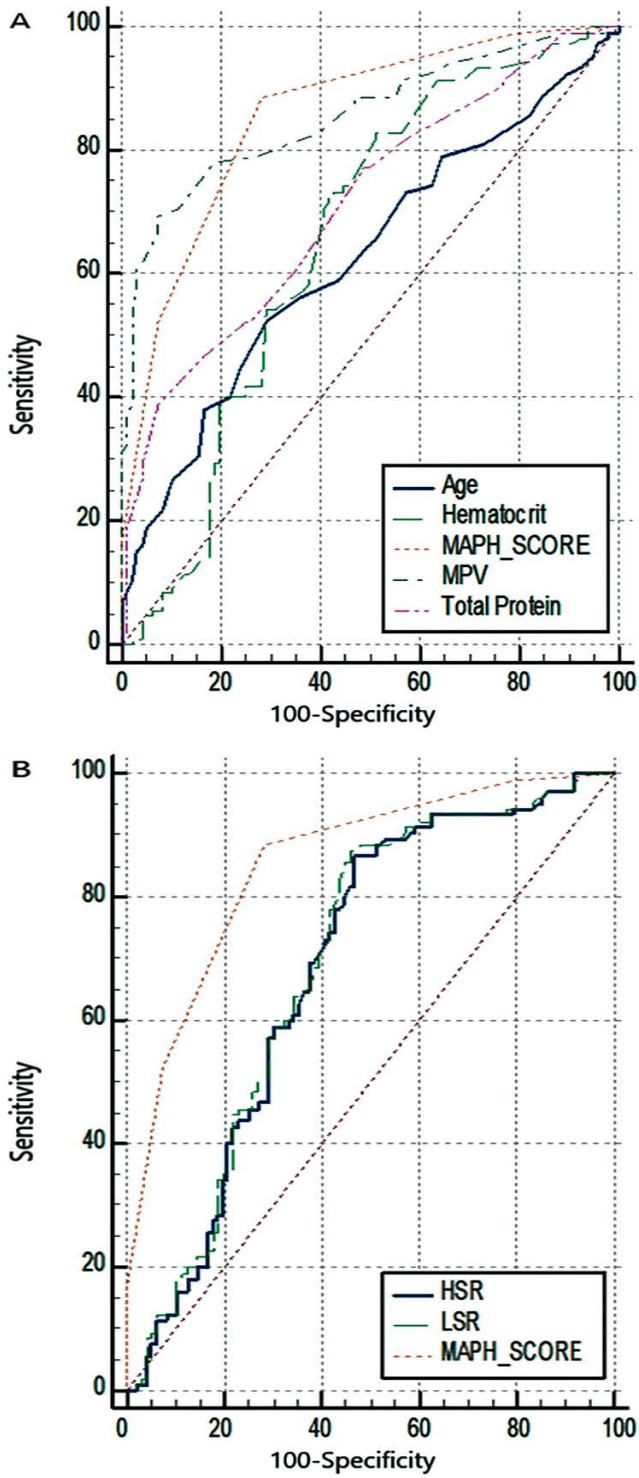
MPV, mean platelet volume; MAPH, MPV: age+total protein+hematocrit; HSR, high shear rate; LSR, low shear rate; c-TFC, corrected TIMI frame count.

Table 3. Pairwise comparisons of ROC curve analysis

	Difference between AUC	95% CI	Z statistic p value
Model I			
Age-hematocrit	0.003	– 0.0856–0.155	0.567; 0.5709
Age-MPV	0.231	0.135–0.327	4.698; <0.0001
Age-total protein	0.088	– 0.023–0.200	1.547; 0.1218
Age-MAPH score	0.232	0.155–0.310	5.866; <0.0001
Hematocrit-MPV	0.196	0.101–0.292	4.022; 0.0001
Hematocrit-total protein	0.053	– 0.044–0.151	1.066; 0.2865
Hematocrit-MAPH score	0.202	0.123–0.280	5.016; <0.0001
MPV–MAPH score	0.002	– 0.050–0.055	0.008; 0.9356
MPV-total protein	0.143	0.058–0.228	3.311; 0.0009
MAPH score-total protein	0.147	0.078–0.216	4.212; <0.0001
Model II			
HSR-LSR	0.006	0.001–0.011	2.658;0.007
HSR-MAPH score	0.172	0.096–0.248	4.474;<0.0001
LSR-MAPH score	0.166	0.091–0.240	4.361; <0.0001

ROC, receiver operating characteristic; MPV, mean platelet volume; MAPH, MPV+age+total protein+hematocrit; HSR, high shear rate; LSR, low shear rate.

Figure 2. Pairwise comparisons of variables for coronary slow flow.
A: comparisons of MAPH score, age, hematocrit, total protein, and MPV.
B: comparisons of MAPH score, HSR, and LSR. MPV, mean protein volume; HSR, high shear rate; LSR, low shear rate



Discussion

In this study, CSF was associated with smoking, increased age, higher total cholesterol, hematocrit, total protein, and MPV. Importantly, the MAPH score, which includes age, total protein, and hematocrit, was also higher in the CSF group. The most valuable finding of this study is that the best correlation was between the MAPH score and CSF. Furthermore, the ability of the MAPH score to predict CSF was higher than the individual performance of each component factor.

Although CSF is considered an innocent angiographic finding, it may cause acute chest pain syndromes and impair the quality of life [17, 18]. Despite numerous studies to elucidate the underlying pathophysiology of CSF, its mechanism remains unclear. Dysfunction of microvascular tone, imbalance of vasoconstrictive substrates, inflammation, metabolic factors, and hemorheological parameters have been blamed [2, 19–21]. A previous study revealed that WBV is an independent and significant predictor factor for CSF. This relationship was also shown in the current study. Blood viscosity is the major component of endothelial shear stress, and it plays a key role in endothelial function [22, 23]. In addition to the mechanical effect of viscosity on blood flow velocity, increased blood viscosity may cause endothelial dysfunction, leading to CSF. De Simone et al. [24] demonstrated an association of WBV with demographic, physiologic, and cardiovascular risk factors. They also investigated the contribution of rheological factors,

ISSN 0022-9040. Кардиология. 2024;64(2). DOI: 10.18087/cardio.2024.2.n2322

i.e., hematocrit, plasma viscosity, erythrocyte aggregability, and rigidity, to WBV. In their study, the major determinant of blood viscosity was WBV, and that it is possible to measure blood viscosity with a low margin of error using the formula. In addition, blood viscosity measured with this calculation has been tested in large studies, and its validity has been proven [7]. In the current study, WBV was found to be an independent and important predictor factor of CSF.

Blood viscosity is a dynamic parameter that depends both on lumen diameter and on blood flow velocity. In addition to studies showing the role of blood viscosity in atherosclerosis and its hemodynamic effects have also been demonstrated in large arteries, including the carotid and peripheral arteries. For example, Dormandy et al. [25] showed that blood viscosity in patients with intermittent claudication was higher than in normal individuals. Perhaps the most important finding of that study was that a significant proportion of patients with claudication had normal peripheral angiography, but they had high blood viscosity. It has been emphasized that the cause of claudication in such patients may be increased blood viscosity rather than peripheral vascular disease [26]. Based on this information, Dormandy et al suggested the term "rheological claudication" [25]. According to the results of current study it is possible that hyperviscosity disrupts circulatory dynamics and causes symptoms in patients with CSF, considering that the only pathologic condition is slow flow in the coronary circulation.

MPV is an indicator of platelet activation and is thought to be involved in CSF pathophysiology [27–29]. Platelets containing dense granules are biochemically and functionally more active and are a risk factor for coronary thrombus and myocardial ischemia [30, 31]. In the current study, in parallel with the results of previous studies, MPV was found to be an independent predictor for CSF at values above 10.3 fl.

Another component of the MAPH score is total protein, which consists of important elements of blood viscosity including albumin, fibrinogen, and globulin [10]. A study by Huang et al. [32] found that the concentration globulin was correlated with high-sensitivity C-reactive protein in the patient group with CSF and was an independent predictor of CSF. In this study, total protein of patients with CSF was higher than that of patients with NCF.

Another parameter that should be discussed is age. In this study, the mean age of the patients in the CSF group was higher than that in the NCF group. Aging is a major risk factor for cardiovascular diseases. Many factors, including decreased nitric oxide, increased endothelium-derived vasoconstrictor substrate release, increased oxidative stress, and proinflammatory cytokine release may cause endothelial deterioration with age [33]. Another important finding of our study is that the patients in the CSF group had a higher smokers rate, which is consistent with the results reported by Ghaffari et al. [34].

Limitations

The main limitations of this study are its single center, small number of patients and retrospective nature.

Conclusions

MAPH is a score newly developed by us, and it has previously been shown to be effective in predicting high thrombus burden in patients with STEMI. In this study, we found that the MAPH score had better performance in predicting CSF than the individual performance of each of its component factors. We suggest that the MAPH score may be useful in the identification and treatment of CSF, although large-scale studies are needed for it to be used routinely in practice.

Author contributions

All authors have contributed significantly. Mustafa Kaplangoray designed the study, collected and analyzed the data, and wrote the manuscript. Cihan Aydın, Arafat Yıldırım, and Ozge Ozcan Abacıoglu contributed to the study design and provided guidance on the statistical methods. Kenan Toprak, Aykut Demirkıran, and Yusuf Cekici assisted with data collection. All authors agree with the content of the manuscript and its publication.

No conflict of interest is reported.

The article was received on 11/10/2022

REFERENCES

1. Zivanic A, Stankovic I, Ilic I, Putnikovic B, Neskovic AN. Prognosis of patients with previous myocardial infarction, coronary slow flow, and normal coronary angiogram. Herz. 2020;45(S1):88–94. DOI: 10.1007/s00059-019-4817-4
2. Li N, Tian L, Ren J, Li Y, Liu Y. Evaluation of homocysteine in the diagnosis and prognosis of coronary slow flow syndrome. Biomarkers in Medicine. 2019;13(17):1439–46. DOI: 10.2217/bmm-2018-0446
3. Tambe AA, Demany MA, Zimmerman HA, Mascarenhas E. Angina pectoris and slow flow velocity of dye in coronary arteries – A new angiographic finding. American Heart Journal. 1972;84(1):66–71. DOI: 10.1016/0002-8703(72)90307-9
4. Buber I, Nar R, Kaya D, Senol H, Adali MK, Nar G. Assessment of triglyceride/glucose index with respect to coronary slow flow. Bratislava Medical Journal. 2022;123(8):585–8. DOI: 10.4149/BLL_2022_095
5. Sezgin AT, Sigirci A, Barutcu I, Topal E, Sezgin N, Ozdemir R et al. Vascular endothelial function in patients with slow coronary flow. Coronary Artery Disease. 2003;14(2):155–61. DOI: 10.1097/00019501-200304000-00008

6. Cin VG, Pekdemir H, Ahmet Camsar, Dilek Cicek, Akkus MN, Parmakýz T et al. Diffuse Intimal Thickening of Coronary Arteries in Slow Coronary Flow. Japanese Heart Journal. 2003;44(6):907–19. DOI: 10.1536/jhj.44.907
7. Cetin MS, Ozcan Cetin EH, Canpolat U, Aydın S, Temizhan A, Topaloglu S et al. An overlooked parameter in coronary slow flow phenomenon: whole blood viscosity. Biomarkers in Medicine. 2015;9(12):1311–21. DOI: 10.2217/bmm.15.92
8. Mahfouz HM, Yassen IAEF, Mahgoub KAM. Noninvasive Predictors of Coronary Slow Flow Phenomenon in Patients Presenting with Chronic Coronary Syndrome. Cardiology and Angiology: An International Journal. 2022;11(1):11–20. DOI: 10.9734/ca/2022/v11i130185
9. Zhu X, Shen H, Gao F, Wu S, Ma Q, Jia S et al. Clinical Profile and Outcome in Patients with Coronary Slow Flow Phenomenon. Cardiology Research and Practice. 2019;2019:9168153. DOI: 10.1155/2019/9168153
10. Abacioglu OO, Yildirim A, Karadeniz M, Abacioglu S, Koyunsever NY, Dindas F et al. A New Score for Determining Thrombus Burden in STEMI Patients: The MAPH Score. Clinical and Applied Thrombosis/Hemostasis. 2022;28:107602962110737. DOI: 10.1177/10760296211073767
11. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. European Heart Journal. 2020;41(1):111–88. DOI: 10.1093/eurheartj/ehz455
12. Davies MJ, D'Alessio DA, Fradkin J, Kernan WN, Mathieu C, Mingrone G et al. Management of Hyperglycemia in Type 2 Diabetes, 2018. A Consensus Report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetes Care. 2018;41(12):2669–701. DOI: 10.2337/dci18-0033
13. Aciksari G, Cetinkal G, Kocak M, Atici A, Celik FB, Caliskan M. The relationship between triglyceride/high-density lipoprotein cholesterol ratio and coronary slow-flow phenomenon. The International Journal of Cardiovascular Imaging. 2022;38(1):5–13. DOI: 10.1007/s10554-021-02387-w
14. Sloop G, Holsworth RE, Weidman JJ, St Cyr JA. The role of chronic hyperviscosity in vascular disease. Therapeutic Advances in Cardiovascular Disease. 2015;9(1):19–25. DOI: 10.1177/1753944714553226
15. Pepe MS. The statistical evaluation of medical tests for classification and prediction. -Oxford ; New York: Oxford University Press;2003. - 302 p. ISBN 978-0-19-850984-4
16. Youden WJ. Index for rating diagnostic tests. Cancer. 1950;3(1):32–5. DOI: 10.1002/1097-0142(1950)3:1<32::AID-CNCR2820030106>3.0.CO;2-3
17. Candemir M, Şahinarslan A, Yazol M, Öner YA, Boyaci B. Determination of myocardial scar tissue in coronary slow flow phenomenon and the relationship between amount of scar tissue and NT-proBNP. Arquivos Brasileiros de Cardiologia. 2020;114(3):540–51. DOI: 10.36660/abc.2018149
18. Nwose EU. Cardiovascular risk assessment and support techniques: Whole blood viscosity assessment issues I: Extrapolation chart and reference values. North American Journal of Medical Sciences. 2010;2(4):165–9. DOI: 10.4297/najms.2010.2165
19. Pekdemir H, Cin VG, Cicek D, Camsari A, Akkus N, Döven O et al. Slow coronary flow may be a sign o diffuse atherosclerosis. Contribution of FFR and IVUS. Acta Cardiologica. 2004;59(2):127–33. DOI: 10.2143/AC.59.2.2005166
20. Ugwoke CK, Cvetko E, Umek N. Skeletal Muscle Microvascular Dysfunction in Obesity-Related Insulin Resistance: Pathophysiological Mechanisms and Therapeutic Perspectives. International Journal of Molecular Sciences. 2022;23(2):847. DOI: 10.3390/ijms23020847
21. Beltrame JF. Defining the Coronary Slow Flow Phenomenon. Circulation Journal. 2012;76(4):818–20. DOI: 10.1253/circj.CJ-12-0205
22. Yaylali YT, Susam I, Demir E, Bor-Kucukatay M, Uludag B, Kilic-Toprak E et al. Increased red blood cell deformability and decreased aggregation as potential adaptive mechanisms in the slow coronary flow phenomenon. Coronary Artery Disease. 2013;24(1):11–5. DOI: 10.1097/MCA.0b013e32835b0bdf
23. Cutri N, Zeitz C, Kucia AM, Beltrame JF. ST/T wave changes during acute coronary syndrome presentation in patients with the coronary slow flow phenomenon. International Journal of Cardiology. 2011;146(3):457–8. DOI: 10.1016/j.ijcard.2010.10.120
24. De Simone G, Devereux RB, Chien S, Alderman MH, Atlas SA, Laragh JH. Relation of blood viscosity to demographic and physiologic variables and to cardiovascular risk factors in apparently normal adults. Circulation. 1990;81(1):107–17. DOI: 10.1161/01.CIR.81.1.107
25. Dormandy JA, Hoare E, Colley J, Arrowsmith DE, Dormandy TL. Clinical, Haemodynamic, Rheological, and Biochemical Findings in 126 Patients with Intermittent Claudication. BMJ. 1973;4(5892):576–81. DOI: 10.1136/bmj.4.5892.576
26. Cho YI, Cho DJ, Rosenson RS. Endothelial Shear Stress and Blood Viscosity in Peripheral Arterial Disease. Current Atherosclerosis Reports. 2014;16(4):404. DOI: 10.1007/s11883-014-0404-6
27. Ösken A, Haci R, Dinç Asarcikli L, Arikan ME, Onuk T, Ünal Dayi Ş et al. Mean platelet volume/platelet count ratio as a predictor of stent thrombosis in patients with ST-segment–elevation myocardial infarction. Irish Journal of Medical Science (1971 -). 2021;190(3):1095–102. DOI: 10.1007/s11845-021-02626-y
28. Korkmaz L, Ata Korkmaz A, Akyuz AR, Agac MT, Acar Z, Kiris A et al. Association between mean platelet volume and coronary artery calcification in patients without overt cardiovascular disease: an observational study. The Anatolian Journal of Cardiology. 2012;12(1):35–9. DOI: 10.5152/akd.2012.007
29. Damaske A, Muxel S, Fasola F, Radmacher MC, Schaefer S, Jabs A et al. Peripheral hemorheological and vascular correlates of coronary blood flow. Clinical Hemorheology and Microcirculation. 2011;49(1–4):261–9. DOI: 10.3233/CH-2011-1476
30. Rao AK, Goldberg RE, Walsh PN. Platelet coagulant activities in diabetes mellitus. Evidence for relationship between platelet coagulant hyperactivity and platelet volume. The Journal of Laboratory and Clinical Medicine. 1984;103(1):82–92. PMID: 6690642
31. Gunebakmaz O, Kaya MG, Kaya EG, Ardic I, Yarlioglues M, Dogdu O et al. Mean platelet volume predicts embolic complications and prognosis in infective endocarditis. International Journal of Infectious Diseases. 2010;14(11):e982–5. DOI: 10.1016/j.ijid.2010.05.019
32. Huang Q, Zhang F, Chen S, Dong Z, Liu W, Zhou X. Clinical characteristics in patients with coronary slow flow phenomenon: A retrospective study. Medicine. 2021;100(6):e24643. DOI: 10.1097/MD.0000000000024643
33. Herrera MD, Mingorance C, Rodríguez-Rodríguez R, Alvarez De Sotomayor M. Endothelial dysfunction and aging: An update. Ageing Research Reviews. 2010;9(2):142–52. DOI: 10.1016/j.arr.2009.07.002
34. Ghaffari S, Tajlil A, Aslanabadi N, Separham A, Sohrabi B, Saeidi G et al. Clinical and laboratory predictors of coronary slow flow in coronary angiography. Perfusion. 2017;32(1):13–9. DOI: 10.1177/0267659116659918

ISSN 0022-9040. Кардиология. 2024;64(2). DOI: 10.18087/cardio.2024.2.n2322