European Review for Medical and Pharmacological Sciences 2023; 27: 5515-5521

The effect of interarm blood pressure difference on coronary collateral flow in patients with ST-segment elevation myocardial infarction who had undergone primary percutaneous coronary intervention

R. ZEHIR[1], B. GÜVENDI ŞENGÖR[1], C. YILMAZ[2]

[1]Department of Cardiology, Kartal Kosuyolu Research and Education Hospital, Kartal, Istanbul, Turkey
[2]Department of Cardiology, Mus Malazgirt State Hospital, Malazgirt, Mus, Turkey

Abstract. – OBJECTIVE: In patients with acute myocardial infarction, coronary collateral circulation (CCC) is associated with reduced infarct size, preserved cardiac function, and decreased mortality. An interarm blood pressure difference (IABPD) is shown to be independently associated with cardiovascular and all-cause of mortality. We aimed to determine the effect of IABPD on coronary collateral flow in patients with ST-segment elevation myocardial infarction (STEMI) who had undergone primary percutaneous coronary intervention (p-PCI).

PATIENTS AND METHODS: We prospectively investigated 1,348 consecutive patients who were hospitalized for STEMI and underwent p-PCI. The Rentrop classification was used to assess CCC. According to this classification, we defined Rentrop 0 and 1 as poor CCC, and Rentrop 2 and 3 as good CCC. A 10 mm Hg difference is considered the upper limit of IABPD.

RESULTS: Patients were divided into two groups according to the collateral circulation, 325 patients (24%) had good collateral, while 1,023 patients (76%) had poor collateral. IABPD was significantly higher in the poor collateral group (57 patients, 5.6%) than in the good collateral group (9 patients, 2.8%) (p=0.04). Pre-infarction angina and IABPD were identified as independent predictors of poor collateral (OR: 0.516, 95% CI 0.370-0.631, p=0.007; OR: 3.681, 95% CI: 1.773-7.461, p=0.01, respectively) in multivariate analysis.

CONCLUSIONS: The IABPD was shown as an independent predictor of poor collateral circulation in patients with STEMI who underwent p-PCI.

Key Words:
Blood pressure, Myocardial infarction, Coronary collateral flow.

Introduction

Acute myocardial infarction (AMI) is one of the most important and fatal coronary artery disease (CAD) presentations. The infarct size is a major indicator of mortality among patients with AMI[1]. Coronary collateral circulation (CCC) serves as an alternative source of blood supply and connects the severely narrowed or occluded coronary arteries when ischemia jeopardizes the myocardium. In patients with AMI, CCC is associated with reduced infarct size, preserved cardiac function, and decreased mortality[2,3].

An interarm blood pressure difference (IABPD) is shown[4] to be independently associated with cardiovascular and all-cause of mortality. A 10 mm Hg difference is considered to be the upper limit of IABPD. The incidence of elevated IABPD (≥10 mm Hg) is 4.4% in the general population with no vascular disease[5]. The IABPD level in patients with CAD is higher than in patients without CAD and the severity of CAD was found to be correlated with IABPD[6]. In this study, we aimed to determine the effect of IABPD on coronary collateral flow in patients with ST-segment elevation myocardial infarction (STEMI) who had undergone primary percutaneous coronary intervention (p-PCI).

Patients and Methods

Study Design and Population

This prospective study included 1,592 patients who had undergone p-PCI for STEMI between January 2019 and September 2021. Demograph-

Corresponding Author: Regayip Zehir, MD; e-mail: regayipz@outlook.com

ic, clinical, and laboratory characteristics were obtained from patients' medical records and telephone interviews. Inclusion criteria were STEMI patients who underwent p-PCI within 12 hours of symptom onset and aged ≥18 years. Exclusion criteria were cardiogenic shock (Killip class IV), severe valvular disease, prior valvular surgery, severe peripheral artery disease, thoracic aortic dissection, aortic coarctation, active infection, atrial fibrillation or atrial flutter, chronic renal failure on hemodialysis. After applying exclusion criteria, the remaining 1,348 patients comprised the study population shown in the consort diagram (Figure 1). The study protocol was approved by the Local Institutional Review Board and all participants gave their written informed consent.

Angiographic Procedure

After administering a 600-mg single-loading dose of clopidogrel and 300 mg of acetylsalicylic acid, the patients underwent coronary angiography *via* a femoral approach. All coronary angiograms included at least two views of the right coronary artery and four views of the left coronary artery. The cine time was adequate to interpret coronary collateral flow. Standard percutaneous coronary intervention (PCI) techniques were carried out using a 7-French guiding catheter (Launcher; Medtronic, Minneapolis, MN, USA) to treat culprit coronary lesions. Unfractionated heparin was administered to achieve an activated clotting time >250 seconds, bare metal or drug-eluting stents were implanted and glycoprotein IIb/IIIa antagonists were administered according to the discretion of the interventional cardiologist. All patients were admitted to the coronary care unit after the procedure and received a dose of 75 mg clopidogrel and 100 mg acetylsalicylic acid daily.

Blood Pressure Measurement

The systolic and diastolic blood pressures were simultaneously measured in both arms at admission to the Emergency Department using a validated blood pressure (BP) device (Omron HEM-7,001-E; Omron Corp, Tokyo, Japan). A standard tourniquet (a width of 12-13 and a length of 35 cm) was used based on the current guideline[7]. A

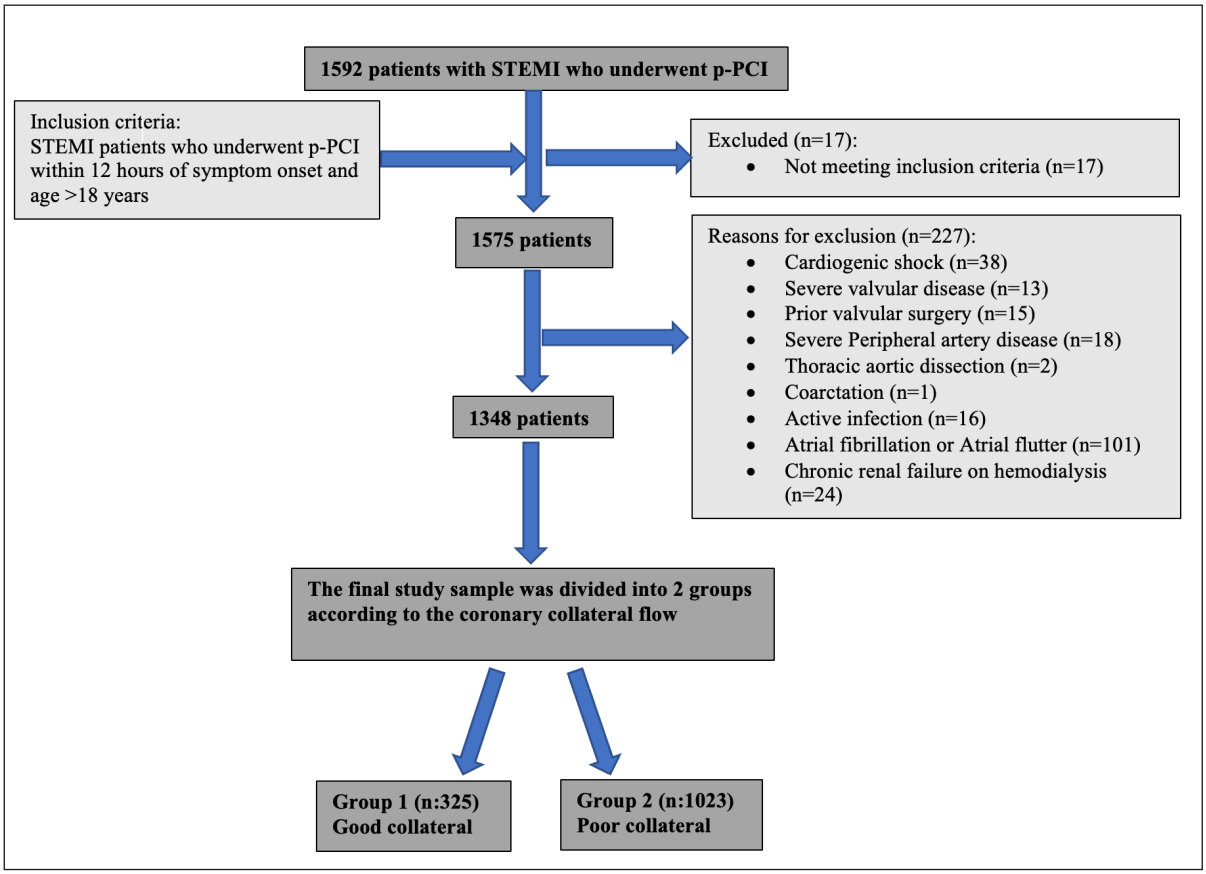

Figure 1. Consort diagram of study population.

larger or smaller size tourniquet was used as needed. After 5 minutes of rest, BP was measured three times at 5-minute intervals in the supine position. The arms of patients were kept at the heart level during measurement. IABPD <10 mm Hg was defined as normal.

Definitions

Hypertension was defined as a systolic BP ≥140 mm Hg or diastolic BP ≥90 mm Hg or the use of antihypertensive medications. IABPD ≥10 mm Hg was considered high. Current smokers or with a history of smoking were defined as smokers. Diabetes mellitus was defined depending on patients' medical history. Hyperlipidemia was defined as total cholesterol levels >200 mg/dl, or low-density lipoprotein (LDL) levels >130 mg/dl, or triglyceride levels >150 mg/dl, or the use of lipid-lowering drugs. STEMI was defined as the presence of ST-segment elevation of at least 1 mm in 2 or more contiguous leads (2 mm for V_1-V_3), at least 0.5 mm in leads V_3R and V_4R, or ≥0.5 mm in leads V_7-V_9, or new onset left bundle branch block. The manifestation of AMI was classified according to the Killip classification: Killip I no evidence of heart failure, Killip II heart failure, Killip III severe heart failure or acute pulmonary edema, Killip IV cardiogenic shock.

Coronary angiography views were independently evaluated by at least two expert interventional cardiologists. Coronary flow was graded according to Thrombolysis in Myocardial Infarction (TIMI) criteria: TIMI 0 no antegrade flow, TIMI I penetration without perfusion, TIMI II partial perfusion, TIMI III complete perfusion. The Rentrop classification was used to assess CCC. On the basis of this classification, the collateral flow was graded as follows: Rentrop 0 absent, Rentrop 1 weak collateral flow, Rentrop 2 partial collateral flow, Rentrop 3 complete collateral flow. After the grading, we defined Rentrop 0 and 1 as poor CCC, Rentrop 2 and 3 as good CCC.

Statistical Analysis

Data were processed using SPSS version 19.0 (IBM Corp., Armonk, NY, USA). Data were presented as the median (interquartile range) for continuous variables or the number (percentage) for categorical variables. The Shapiro-Wilk test of normality was used to identify the distribution of data. The Mann-Whitney U test was utilized to compare two groups for continuous variables without normal distribution. Student *t*-test was utilized to compare two groups for continuous variables with normal distribution. χ^2 test was used to compare categorical variables. Univariate regression analysis was performed to identify risk factors for poor collateral development. Multivariate logistic regression models were used to determine independent predictors of poor collateral development. The odds ratios and 95% confidence intervals (CI) were calculated. A two-tailed *p*-value <0.05 was considered statistically significant.

Results

The baseline demographic, clinical, laboratory, and angiographic characteristics of the two patient groups are shown in Table I. A total of 1,348 patients who met the inclusion criteria were enrolled in the study and the majority of the patients were males (77%). Patients were divided into two groups according to collateral circulation, 325 patients (24%) had good collateral, and 1,023 patients (76%) had poor collateral. The median age was 64±12 years in the good collateral group and 62±11 years in the poor collateral group. The body mass index, ejection fraction, hypertension, diabetes mellitus, hyperlipidemia, smoking status, and laboratory parameters were similar in the two groups. The ratios of radial access, predilatation, tirofiban usage, and unsuccessful intervention also did not differ between the two groups. IABPD was significantly higher in the poor collateral group (57 patients, 5.6%) than in the good collateral group (9 patients, 2.8%) (p=0.04). The poor collateral group had significantly higher rates of anterior infarction, PCI history, chest pain to revascularization time >6 h, Killip class II or III, and in-hospital mortality (p=0.03, p=0.02, p=0.009, p=0.04, p=0.05, respectively). Multivessel disease, pre-infarction angina, pre-TIMI flow 0-I, and stent length were significantly higher in the good collateral group (p=0.01, p<0.001, p<0.001, and p=0.04, respectively).

In univariate analysis (Table II), poor collateral development was found to significantly correlate with multivessel disease [odds ratio (OR): 0.477, 95% confidence interval (CI): 0.354-0.642, p<0.001], anterior infarct (OR: 2.15, 95% CI: 1.64-2.81, p<0.001), pre-infarct angina (OR: 0.46, 95% CI: 0.356-0.59, p<0.001), PCI history (OR: 1.59, 95% CI: 1.07-2.35, p=0.02), IABPD (OR: 2.07, 95% CI: 1.02-4.24, p=0.03), and Killip class II or III (OR: 1.46, 95% CI: 1.06-2.02, p=0.02).

Multivariate logistic regression analysis was performed using significant variables on univar-

Table I. The differential genes of bone marrow with osteoporosis and normal bone marrow in the geriatric patients were screened by using the limma package in R language.

Variable	Good Collateral n=325 (24%)	Poor Collateral n=1,023 (76%)	*p*-value
Age (years)	64±12	62±11	0.23
Sex (male), n (%)	245 (75.3)	788 (77)	0.64
Body mass index (kg/m^2)	24.6±7.1	23.9±7.7	0.62
IABPD	9 (2.8%)	57 (5.6%)	**0.04**
GFR ≤60, n (%)	158 (48.6)	468 (45.7)	0.43
Anterior infarction, n (%)	96 (29.5)	485 (47.4)	**0.03**
Multivessel disease, n (%)	89 (27.3)	156 (15.2)	**<0.001**
PCI history, n (%)	34 (10.5)	160 (15.6)	**0.02**
Pre-infarction angina, n (%)	162 (49.8)	323 (31.5)	**<0.001**
Chest pain to revascularization time >6 h	48 (14.7)	176 (17.2)	**0.009**
Killip class 2 or 3, n (%)	56 (17.2)	239 (23.3)	**0.04**
EF (%)	42.4±4.7	41.7±1.3	0.33
DM, n (%)	76 (23.3)	260 (25.4)	0.52
HT, n (%)	250 (76.9)	764 (74.6)	0.48
HL, n (%)	54 (16.6)	181 (17.6)	0.47
Smoking, n (%)	148 (45.5)	480 (46.9)	0.42
Anemia, n (%)	100 (30.7)	353 (34.5)	0.34
Radial access, n (%)	160 (49.2%)	509 (49.7%)	0.33
Predilatation, n (%)	275 (84.6)	873 (85.3)	0.42
Trofiban usage, n (%)	78 (24%)	250 (24.4%)	0.84
Pre-TIMI flow 0-1	264 (81.2%)	735 (71.8%)	**<0.001**
Stent length, mm	23.1±6.8	21.5±6.4	**0.04**
Unsuccessful intervention, n (%)	24 (7.3%)	77 (7.5%)	0.81
In-hospital mortality, n (%)	9 (2.5)	28 (2.7)	**0.05**
WBC (10^3/μL)	14±7	14±4.6	0.42
Hemoglobin (g/dl)	12.9±2.1	13.6±1.7	0.09
Neutrophil (10^3/μL)	9.1±6.5	10.5±3.8	0.43
Platelet (10^3/μL)	171.2±42.3	179.5±51.8	0.52
Total cholesterol (mg/dl)	178.5±32.4	178.9±43.4	0.74
LDL (mg/dl)	105±35.1	108.9±37.2	0.51
HDL (mg/dl)	37.4±10.2	38.2±9.8	0.44
TG (mg/dl)	153±76.1	158.9±96.7	0.94
Peak troponin (ng/mL)	36.8±16.8	37.4±18.4	0.41
Basal creatinine (mg/dl)	1.18±0.13	1.02±0.46	0.72
CRP (mg/L)	3.6±0.3	3.5±0.4	0.45

CIN, contrast-induced nephropathy; CRP; C reactive protein; DM, diabetes mellitus; EF, left ventricular ejection fraction; GFR, glomerular filtration rate; HT, hypertension; HL, hyperlipidemia; HDL, high-density lipoprotein; LAD, left anterior descending artery; LDL, low-density lipoprotein; PCI, percutaneous coronary intervention; TG, triglyceride; WBC, white blood cell; TIMI, thrombolysis in myocardial infarction; IASBD, interarm blood pressure difference.

iate analysis to find the predictors of poor collateral development (Table III). Pre-infarction angina and IABPD were identified as independent predictors of poor collateral (OR: 0.516, 95% CI: 0.370-0.631, p=0.007; OR: 3.681, 95% CI: 1.773-7.461, p=0.01, respectively). The multivariate analysis showed that multivessel disease, anterior infarction, PCI history, Killip class II or III did not predict poor collateral development.

Discussion

IABPD is associated with coronary artery disease and its severity. To our knowledge, this is the first study to evaluate the relationship between IABPD and coronary collateral development in patients with STEMI. We found that the IABPD ≥10 mm Hg is an independent predictor of poor collateral development in patients with STEMI who underwent p-PCI.

In patients with STEMI, the remodeling process and enlargement of preexisting arterioles begins after an abrupt coronary occlusion. Subsequently, the redistribution of blood flow and the increase of shear stress leads to the development of CCC[8,9]. CCC has positive effect on clinical outcomes. Good collaterals are shown[1-3,10,11] to preserve left ventricular function, reduce infarct size

and ventricular arrhythmias, improve myocardial salvage, and reduce the risk of mortality in patients with STEMI. That CCC may be associated with a poor prognosis due to being a marker of advanced CAD remains to be controversial[12,13]. In the current study, the poor collateral group had significantly higher Killip class II or III and in-hospital mortality, while the good collateral group had significantly higher rates of multivessel disease (p=0.04, p=0.05, p<0.001, respectively). Because of stimulating collateral development, pre-infarction angina was also significantly higher in the good collateral group, consistent with previous studies[14,15] (p<0.001).

The underlying mechanism of CAD and the acute coronary syndrome is inflammation[16]. Therefore, inflammation-related markers have provided benefits regarding the diagnosis and prognosis. Mansiroglu et al[17] showed that neutrophil/lymphocyte ratio, an important inflammation marker, was significantly higher in the STEMI group that developed collaterals. Liu et al[18] also reported that the higher triglyceride to high-density lipoprotein (HDL)-cholesterol ratio was an independent risk factor for poor collateral development in elderly patients with STEMI because it mediated endothelial function and the growth of collateral circulation by affecting some inflammatory reactions. Acute hyperglycemia was shown[18,19] to result in decreased collateral circulation due to oxidative stress and inflammation. Unlike the previous studies[18,19] in literature, we found no significant differences between the good and poor collateral groups with respect to the laboratory parameters, the history of diabetes mellitus, or hyperlipidemia. Although previous studies[17-19] have shown that blood laboratory parameters have negative effects on endothelial function and collateral circulation, we think that the results are more generalizable since our study included a larger number of patients. However, in order to reach more realistic results in this regard, it may be more beneficial to perform histological evaluations rather than angiographic classification.

The IABPD measurement is a simple, low-cost, fast and noninvasive test that can be easily applied in our daily practice. Some recent trials[20-22] showed that IABPD ≥10 mm Hg was associated with cardio-cerebrovascular disease and cardiovascular mortality. Although the exact mechanism underlying this relation still remains unclear, atherosclerosis and increased vascular stiffness have been considered to be responsible pathologies[23,24]. One cross-sectional study[25] revealed that the SYNTAX score was significantly higher in patients with a high IABPD. We found that the IABPD was significantly higher in the poor collateral group, and the univariate and multivariate analysis revealed that the high IABPD was an independent predictor of poor collateral circulation in patients with STEMI (p=0.04; OR: 2.07, 95% CI: 1.02-4.24, p=0.03; OR: 3.681, 95% CI: 1.773-7.641, p=0.01, respectively).

Based on our findings, routine IABPD measurement may be considered in STEMI patients undergoing p-PCI. Considering the relationship between poor coronary collateral development and IABPD, we think that measuring the IABPD at the time of diagnosis is very important in terms of prognosis prediction in patients with STEMI. Patients with IABPD may have greater post-infarction damage due to poor collateralization. For this reason, more arrhythmia and heart failure symptoms may be seen in these patients in the first 48 hours after infarction and in the following period. These patients may be more prone to mechanical complications due to increased damage and the devastating effects of the damage. Extending the monitored follow-up period of these patients in the coronary intensive care unit and

Table II. Univariate analysis: predictors of poor collateral development.

Parameters	OR (95% CI)	p-value
Pre-TIMI flow 0-1	0.992 (0.752-1.31)	0.95
Multivessel disease	0.477 (0.354-0.642)	<0.001
Anterior infarction	2.15 (1.64-2.81)	<0.001
Pre-infarction angina	0.46 (0.356-0.59)	<0.001
PCI history	1.59 (1.07-2.35)	0.02
IABPD	2.07 (1.02-4.24)	0.03
Killip class II or III	1.46 (1.06-2.02)	0.02
Chest pain to revascularization time >6 h	1.23 (0.86-1.74)	0.24

TIMI, thrombolysis in myocardial infarction; IASBD, interarm blood pressure difference; PCI, percutaneous coronary intervention.

Table III. Multivariate analysis: predictors of poor collateral development.

Parameters	OR (95% CI)	*p*-value
Multivessel disease	0.957 (0.788-1.147)	**0.29**
Anterior infarction	1.150 (0.729-1.950)	0.30
Pre-infarction angina	0.516 (0.370-0.631)	**0.007**
PCI history	1.190 (0.606-1.964)	**0.41**
IABPD	3.681 (1.773-7.641)	**0.01**
Killip class II or III	1.453 (0.898-2.140)	0.63

TIMI, thrombolysis in myocardial infarction; IASBD, interarm blood pressure difference; PCI, percutaneous coronary intervention.

closer follow-up in terms of complications may be considered. In addition, these patients may need a rehabilitation program. Larger randomized follow-up studies are needed in the future to evaluate the early and late prognostic effects of IABPD in STEMI patients. We believe that the valuable results of our study will shed light on future studies.

Limitations

This study was a single-center study and included only angiographically visualized coronary collaterals which were more than 100 μm in diameter.

Conclusions

The IABPD has been shown to be an independent predictor of poor collateral circulation in patients with STEMI who underwent p-PCI. The likely mechanism could be inflammation, as in atherosclerosis. The IABPD is a straightforward and easy-to-measure parameter, and physicians should keep in mind the possible association between higher IABPD and poor collateral circulation in the process of diagnosis and therapy.

Conflict of Interest

The Authors declare that they have no conflict of interests.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Ethics Approval

All the investigators ensure that the study has been conducted according to the Declaration of Helsinki Guidelines. Approval was granted by the Ethics Committee of Kartal Kosuyolu Training and Research Hospital Ethics Committee (Protocol number: 17/644).

Informed Consent

Informed consent was obtained from all patients.

Availability of Data and Materials

The study data are available in our University Hospital archive.

Funding

None.

Authors' Contributions

All authors approved the final version before submission. Conception and design of the study: RZ; Acquisition of data: CY, BGS; Analysis and interpretation of data: RZ. Drafting the article or making critical revisions related to the relevant intellectual content of the manuscript: BGS, CY; Supervision, validation and final approval of the version of the article to be published: RZ. The corresponding author attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted.

ORCID ID

Regayip Zehir: 0000-0003-2729-884X
Busra Guvendi Sengor: 0000-0001-7946-1229
Cemalettin Yilmaz: 0000-0003-4140-9139

References

1) Yoon SJ, Ko YG, Kim JS, Moon JY, Kim YJ, Park S, Ha JW, Choi D, Jang Y, Chung N, Shim WH, Cho SY. Impact of coronary artery collaterals on infarct size assessed by serial cardiac magnetic resonance imaging after primary percutaneous coronary intervention in patients with acute myocardial infarction. Coron Artery Dis 2009; 20: 440-445.
2) Elsman P, van 't Hof AW, de Boer MJ, Hoorntje JC, Suryapranata H, Dambrink JH, Zijlstra F; Zwolle Myocardial Infarction Study Group. Role of collateral circulation in the acute phase of ST-segment-elevation myocardial infarction treated with primary coronary intervention. Eur Heart J 2004; 25: 854-858.

3) Cui K, Lyu S, Song X, Yuan F, Xu F, Zhang M, Zhang M, Wang W, Zhang D, Tian J. Effect of Coronary Collaterals on Prognosis in Patients Undergoing Primary Percutaneous Coronary Intervention for Acute ST-Segment Elevation Myocardial Infarction: A Meta-Analysis. Angiology 2018; 69: 803-811.
4) Clark CE, Taylor RS, Shore AC, Ukoumunne OC, Campbell JL. Association of a difference in systolic blood pressure between arms with vascular disease and mortality: a systematic review and meta-analysis. Lancet 2012; 379: 905-914.
5) Clark CE, Taylor RS, Shore AC, Campbell JL. Prevalence of systolic inter-arm differences in blood pressure for different primary care populations: systematic review and meta-analysis. Br J Gen Pract 2016; 66: e838-e847.
6) Tokitsu T, Yamamoto E, Hirata Y, Fujisue K, Sugamura K, Maeda H, Tsujita K, Kaikita K, Hokimoto S, Sugiyama S, Ogawa H. Relationship between inter-arm blood pressure differences and future cardiovascular events in coronary artery disease. J Hypertens 2015; 33: 1780-1790.
7) Coca Payeras A, Williams B, Mancia G. Response to 'Comment on 2018 ESC/ESH Guidelines for the management of arterial hypertension'. Eur Heart J 2019; 40: 2093.
8) Schaper W, Ito WD. Molecular mechanisms of coronary collateral vessel growth. Circ Res 1996; 79: 911-919.
9) Carmeliet P. Mechanisms of angiogenesis and arteriogenesis. Nat Med 2000; 6: 389-395.
10) Nicolau JC, Pinto MA, Nogueira PR, Lorga AM, Jacob JL, Garzon SA. The role of antegrade and collateral flow in relation to left ventricular function post-thrombolysis. Int J Cardiol 1997; 61: 47-54.
11) Meier P, Gloekler S, de Marchi SF, Zbinden R, Delacrétaz E, Seiler C. An indicator of sudden cardiac death during brief coronary occlusion: electrocardiogram QT time and the role of collaterals. Eur Heart J 2010; 31: 1197-1204.
12) Rechciński T, Jasińska A, Peruga JZ, Foryś J, Krzemińska-Pakuła M, Bednarkiewicz Z, Trzos E, Kasprzak JD. Presence of coronary collaterals in ST-elevation myocardial infarction patients does not affect long-term outcome. Pol Arch Med Wewn 2013; 123: 29-37.
13) Hara M, Sakata Y, Nakatani D, Suna S, Nishino M, Sato H, Kitamura T, Nanto S, Hori M, Komuro I; OACIS Investigators. Impact of coronary collaterals on in-hospital and 5-year mortality after ST-elevation myocardial infarction in the contemporary percutaneous coronary intervention era: a prospective observational study. BMJ Open 2016; 6: e011105.
14) Fujita M, Nakae I, Kihara Y, Hasegawa K, Nohara R, Ueda K, Tamaki S, Otsuka K, Sasayama S. Determinants of collateral development in patients with acute myocardial infarction. Clin Cardiol 1999; 22: 595-599.
15) Kim EK, Choi JH, Song YB, Hahn JY, Chang SA, Park SJ, Lee SC, Choi SH, Choe YH, Park SW, Gwon HC. A protective role of early collateral blood flow in patients with ST-segment elevation myocardial infarction. Am Heart J 2016; 171: 56-63.
16) Balta S, Celik T, Mikhailidis DP, Ozturk C, Demirkol S, Aparci M, Iyisoy A. The Relation Between Atherosclerosis and the Neutrophil-Lymphocyte Ratio. Clin Appl Thromb Hemost 2016; 22: 405-411.
17) Mansiroglu AK, Sincer I, Gunes Y. Assessment of neutrophil and neutrophil/lymphocyte ratio in coronary collateral developed patients with acute coronary syndrome. Rev Assoc Med Bras (1992) 2020; 66: 954-959.
18) Liu GY, Meng XX, Zhang Z. Triglyceride to HDL-cholesterol ratio as an independent risk factor for the poor development of coronary collateral circulation in elderly patients with ST-segment elevation myocardial infarction and acute total occlusion. Medicine (Baltimore) 2018; 97: e12587.
19) Kurmus O, Aslan T, Ekici B, Baglan Uzunget S, Karaarslan S, Tanindi A, Erkan AF, Akgul Ercan E, Kervancıoglu C. Impact of Admission Blood Glucose on Coronary Collateral Flow in Patients with ST-Elevation Myocardial Infarction. Cardiol Res Pract 2018; 2018: 4059542.
20) Li M, Fan F, Zhang Y, Ma W, Huo Y. Elevated Interarm Systolic Blood Pressure Difference Is Positively Associated with Increased Likelihood of Coronary Artery Disease. Int J Hypertens 2021; 2021: 5577957.
21) Yu S, Zhou Y, Wu K, Zhou X, Yang Y, Qiu H, Liu X, Ke J, Wang X, Li Z, Chen X, Ruan X. Association of interarm blood pressure difference with cardio-cerebral vascular disease: A community-based, cross-sectional study. J Clin Hypertens (Greenwich) 2019; 21: 1115-1123.
22) Clark CE, Warren FC, Boddy K, McDonagh STJ, Moore SF, Goddard J, Reed N, Turner M, Alzamora MT, Ramos Blanes R, Chuang SY, Criqui M, Dahl M, Engström G, Erbel R, Espeland M, Ferrucci L, Guerchet M, Hattersley A, Lahoz C, McClelland RL, McDermott MM, Price J, Stoffers HE, Wang JG, Westerink J, White J, Cloutier L, Taylor RS, Shore AC, McManus RJ, Aboyans V, Campbell JL. Associations Between Systolic Interarm Differences in Blood Pressure and Cardiovascular Disease Outcomes and Mortality: Individual Participant Data Meta-Analysis, Development and Validation of a Prognostic Algorithm: The INTERPRESS-IPD Collaboration. Hypertension 2021; 77: 650-661.
23) Su HM, Lin TH, Hsu PC, Chu CY, Lee WH, Chen SC, Lee CS, Voon WC, Lai WT, Sheu SH. Association of interarm systolic blood pressure difference with atherosclerosis and left ventricular hypertrophy. PLoS One 2012; 7: e41173.
24) Canepa M, Milaneschi Y, Ameri P, AlGhatrif M, Leoncini G, Spallarossa P, Pontremoli R, Brunelli C, Strait JB, Lakatta EG, Ferrucci L. Relationship between inter-arm difference in systolic blood pressure and arterial stiffness in community-dwelling older adults. J Clin Hypertens (Greenwich) 2013; 15: 880-887.
25) Durmuş G, Belen E, Bayyigit A, Kalyoncuoğlu M, Can MM. The Relationship between Inter-Arm Blood Pressure Difference and Coronary Artery Disease Severity Calculated by the SYNTAX Score. Int J Hypertens 2018; 2018: 9370417.