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ORIGINAL ARTICLE

Association between coronary microvascular dysfunction indices and infarction size following primary percutaneous coronary intervention

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Summary

Introduction: Coronary microvascular dysfunction (CMD) has been associated with impaired prognosis in patients with ST-elevation myocardial infarction (STEMI) despite timely and successful primary percutaneous coronary intervention (PCI). Our aim was to compare the ability of two different CMD indices, coronary flow reserve (CFR) and hyperemic microvascular resistance (HMR), to predict infarct size.

Methods: The analysis included 31 patients with STEMI in whom valid invasive measurements of coronary blood flow velocity with the Doppler-tipped coronary wire had been performed at the end of primary PCI, and in whom infarct size was determined by cardiac magnetic resonance (CMR) after at least 3 months. Receiver operating (ROC) curves were used to comparatively assess the capacity of HMR and CFR to predict large infarction, defined as the highest quartile of the included population (≥16% of the left ventricular mass).

Results: Invasive measurements at the end of primary PCI revealed an average HMR value of 2.72±0.86 mmHg/cm/s, and that of CFR to be 1.65± 0.36. Both indices of microvascular function significantly correlated with infarct size (rho=0.569, p<0.01, for HMR, and rho= -0.391, p=0.029, for CFR). Comparative assessment of ROC curves revealed a similar capacity of HMR and CFR to predict large myocardial infarction (AUC=0.669, 95%CI 0.472-0.866 for HMR vs. AUC=0.712, 95%CI 0.517-0.907 for CFR; p=0.718 for comparison).

Conclusion: Doppler wire-derived HMR and CFR, measured simultaneously at the end of primary PCI, exhibited similar capacities for predicting large myocardial infarction.

Keywords: coronary microcirculation, infarct size, primary PCI

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INTRODUCTION

Primary percutaneous coronary intervention (PCI) with the resulting recanalization of the infarct-related artery (IRA) is the recommended standard of care for patients presenting with ST-elevation myocardial infarction (STEMI) (1). Early research during the emergence of recanalization therapies in the 1990s revealed that, in a substantial proportion of patients, the myocardium within the risk area remained inadequately perfused despite timely and successful epicardial artery recanalization (2). Contemporary research has confirmed that in around 50% of patients undergoing primary PCI, there is residual coronary microvascular injury leading to impaired reperfusion at the level of cardiac myocytes, which is ultimately associated with increased mortality (3, 4). The injury to the coronary microcirculation post primary PCI is nowadays most frequently expressed as microvascular obstruction (MVO) on cardiac magnetic resonance (CMR) imaging or as coronary microvascular dysfunction (CMD) estimated by an invasive measurement of coronary flow velocity either directly by a Doppler-tipped coronary wire or indirectly by the thermodilution method (5). Both MVO and invasive indices of CMD, such as coronary flow reserve (CFR) or microvascular resistance, have been associated with increased mortality following primary PCI (6-9). As microvascular injury has been found to be an independent predictor of mortality after STEMI (10), it becomes a therapeutic target, with ongoing research efforts to define optimal treatment strategies to decrease the degree of microvascular damage post-primary PCI (11, 12). Infrastructural constraints associated with the inability to routinely perform CMR in STEMI have shaped the current research paradigm towards stratifying patients at the end of primary PCI according to the presence of CMD as assessed by invasive indices of microvascular function, such as the index of microcirculatory resistance (IMR) (13, 14). This approach is based on a series of studies demonstrating that different indices of microvascular function, such as IMR, hyperemic microvascular resistance (HMR), and CFR, have been associated with mortality and heart failure following primary PCI (6, 7, 14). Several comparative studies indicated that resistance indices (IMR or HMR) may have been more closely related to MVO or infarct size than CFR (15). Pressure at zero flow (PzF), an index considering extravascular compression, had the best capacity to predict structural myocardial damage post MI (6, 16). However, the invasive nature of indices of microvascular resistance and PzF constrains their use in daily practice. Given the correlation of non-invasive, transthoracic doppler echocardiography (TTDE)-derived CFR with the invasive, Doppler wire-derived CFR (17), similar predictive ability of invasive CFR and HMR may open the door towards a more widespread use of CFR to risk-stratify patients after STEMI according to their risk of mortality and heart failure. Hence, our aim was to comparatively assess the ability of HMR and CFR, measured simultaneously at the end of a timely and successful primary PCI, to predict large infarct size, as a surrogate of an increased risk of future mortality and heart failure (18).

METHODS

Patients

This was a prospective, observational study, which included 31 patients with valid simultaneous invasive measurements of coronary blood flow velocity and distal coronary pressure with a Doppler-tipped intracoronary guidewire in the recanalized IRA immediately post primary PCI, for whom infarct size assessment on CMR was also available, at least 3 months after the acute event (CMR was available for 32 patients). Patients who presented with STEMI, within 12 hours of chest pain onset, were included if undergoing a successful primary PCI, with TIMI 3 flow and residual stenosis <20-30% post-PCI. Exclusion criteria were acute heart failure or a known history of chronic heart failure, a known prior coronary artery disease and the presence of a critical stenosis in a non-IRA territory. All patients were treated with a loading dose of aspirin (300mg) and either ticagrelor 180mg or clopidogrel 600mg, prior to primary PCI, and with standard maintenance doses afterwards. Use of Glycoprotein IIb/IIIa inhibitors at any point of the procedure or post-procedurally, and manual thromboaspiration were at the discretion of the operator. Ethics Committee of the University Clinical Center of Serbia (protocol code 747/11, 19 July 2018) approved the protocol, and the research was conducted in accordance with the Declaration of Helsinki.

Study procedures

Immediately after a successful primary PCI, patients underwent simultaneous coronary flow velocity and distal coronary pressure measurements by positioning a Doppler-tipped coronary guide wire (ComboWire, Philips Volcano, San Diego, California) in the distal segment of the recanalized IRA. The simultaneous coronary pressure and flow velocity measurements were performed at rest and again after inducing hyperemia by the intracoronary injection of adenosine, at least 200 mcg for the left anterior descending (LAD) and 100 mcg for the right coronary artery (RCA). Blood flow velocity was expressed as average peak velocity (APV) over three heart cycles. From those measurements, CFR and HMR were automatically calculated by the ComboMap system (Philips Volcano, San Diego, California), which processed the data obtained by the ComboWire together with the simultaneous ECG and aortic pressure tracings. Fractional flow reserve (FFR) was also calculated from the available data, as the ratio of distal coronary pressure (Pd) over the aortic pressure (Pa).

After 3 months (median 7.5 months), patients underwent cardiac magnetic resonance (CMR) imaging with a 1.5 Tesla MR-scanner (Magnetom Avanto, Siemens, Erlangen, Germany). Infarct size was measured as a proportion of the LV mass that is infarcted, as delineated by late gadolinium enhancement (LGE) after the administration of 0.2 mmol/kg of Gadobutrol (Gadovist, Bayer Inc, Canada). In addition, CMR measurements included the assessment of left ventricular size and function, as expressed by left ventricular ejection fraction (LVEF), end-diastolic volume (EDV) and end-systolic volume (ESV).

Statistics

After testing for the normality of their distribution with the Shapiro-Wilk test, variables with normal distribution were presented as means with standard deviation and compared with the student t test, whereas variables with non-normal distribution were presented as median with interquartile range and compared with the Mann-Whitney U test. Counts were used to present categorical variables, which were compared with the chi-squared test. If necessary, correlations between continuous variables were assessed using the Pearson (r) or Spearman (rho) coefficient. We constructed receiver-operating characteristic (ROC) curves to assess the ability of HMR and CFR to predict large infarct size, representing the highest quartile of the included population (≥16% of the mass of the left ventricle). Optimal HMR and CFR cut-off values for the prediction of a large infarction were determined using the Youden index. The Delong method was used to compare the ROC curves constructed for HMR and CFR. The degree of association of HMR and CFR with infarct size was explored by constructing multivariable regression models with the inclusion of other key predictor variables known at the time of the primary PCI procedure. P-value <0.05 was adopted as the marker of statistical significance. SPSS 26.0 (IBM, Chicago, Illinois) and GraphPad Prism (La Jola, California) were used for all statistical analyses and graph constructions.

RESULTS

Table 1 lists baseline characteristics of the included patients. The study population consisted predominantly of patients with STEMI who underwent primary PCI early after chest pain onset (median time to reperfusion was just under 3 hours). Median age was 59 and 20% were females. The procedure was performed via transradial route in all cases, and thromboaspiration was utilized in 16%, whereas Glycoprotein inhibitors (GPI) were administered in 13% of cases. The culprit artery was the LAD in two-thirds of patients. In almost all of the included patients (90%), the culprit artery was occluded at the start of the procedure. In-hospital echocardiography, several days following primary PCI, revealed an average LVEF of 45%. Finally, post-PCI ECG established the presence of incomplete ST-segment resolution in close to 50% of patients, which has been an accepted surrogate for coronary microvascular impairment.

Table 1. Baseline characteristics.

Baseline clinical and angiographic variables	n=31
Age, years, median (IQR)	59 (53-62)
Male, n (%)	26 (84)
Diabetes mellitus, n (%)	6 (19)
Hyperlipidemia, n (%)	24 (77)
Hypertension, n (%)	18 (58)
Time, symptom onset to reperfusion, min, median, (IQR)	175 (125-270)
Infarct-related artery LAD, n (%)	21 (68)
TIMI flow grade 0 before primary PCI, n (%)	28 (90)
Trans-radial access, n (%)	31 (100)
Manual aspiration thrombectomy, n (%)	5 (16)
Glycoprotein IIb/IIIa inhibitors, n (%)	4 (13)
Echocardiography during index hospitalization	
Left ventricular ejection fraction, %, mean ± SD	45 ±11
End-systolic diameter, mm, mean ± SD	54 ± 5
End-diastolic diameter, mm, mean ± SD	38 ± 6
Electrocardiography after 90 minutes of primary PCI	
Incomplete ST-segment resolution, n (%)	15 (48)

IQR – interquartile range, LAD – left anterior descending, PCI – percutaneous coronary intervention, SD – standard deviation.

Table 2 shows the average parameters of intracoronary physiology at the end of the primary PCI procedure. Mean HMR was 2.72±0.86 mmHg/cm/s, above the earlier threshold of 2.50 mmHg/cm/s, implying the presence of CMD in an average patient undergoing primary PCI. Similarly, the mean CFR was 1.65±0.36, which is below the established threshold of 2.50, also implying the presence of CMD. It should be noted that the average value of FFR was 0.92, which likely suggested no significant residual disease, confirming that the obtained CFR values primarily reflected microvascular function.

Table 2. Invasive measurements of coronary pressure and flow at the end of primary PCI.

Coronary physiology by Doppler wire	n=31
APV resting, cm/s, mean ± SD	21.00 ± 6.05
APV hyperemic, cm/s, mean ± SD	34.58 ± 12.48
Pa, resting, mmHg, mean ± SD	93.48 ± 11.71
Pd, resting, mmHg, mean ± SD	89.45 ± 12.29
Pa, hyperemic, mmHg, mean ± SD	88.71 ± 12.52
Pd, hyperemic, mmHg, mean ± SD	81.90 ± 11.64
CFR, mean ± SD	1.65 ± 0.36
HMR, mmHg/cm/s, mean ± SD	2.72 ± 0.86
FFR, mean ± SD	0.92 ± 0.04

APV – average peak velocity, Pa – pressure aorta, Pd – pressure distal, CFR – coronary flow reserve, HMR – hyperemic microvascular resistance, FFR – fractional flow reserve, SD – standard deviation.

Table 3. Invasive coronary physiology in the culprit artery at the end of primary PCI and parameters of left ventricular structure and function on CMR after 3 months according to the infarct size.

	Small infarct size (<16% of the LV) n=22	Large infarct size (≥16% of the LV) n=9	p-value
Invasive coronary physiology at the end of primary PCI			
APV resting, cm/s, mean ± SD	21.55 ±6.89	19.67 ±3.16	0.442
APV hyperemic, cm/s, mean ± SD	36.77 ±13.73	29.22 ±6.59	0.128
Pa, resting, mmHg, mean ± SD	93.32 ±11.14	94.80 ±13.26	0.745
Pd, resting, mmHg, mean ± SD	90.00 ±11.86	88.90 ±13.38	0.817
Pa, hyperemic, mmHg, mean ± SD	88.59 ±11.93	89.00 ±14.64	0.936
Pd, hyperemic, mmHg, mean ± SD	81.64 ±11.75	82.56 ±12.04	0.846
CFR, mean ± SD	1.73 ±0.38	1.48 ±0.25	0.084
HMR, mmHg/cm/s, mean ± SD	2.58 ±0.84	3.04 ±0.84	0.177
FFR, mean ± SD	0.92 ±0.05	0.91 ±0.03	0.508
Left ventricular structure and function at follow-up			
Ejection fraction, %, mean ± SD	55.00 ±6.22	36.89 ±7.90	<0.001
End-diastolic volume, ml, mean ± SD	150.86 ±33.14	220.33 ±59.77	<0.001
End-systolic volume, ml, mean ± SD	71.14 ±22.13	142.33 ±57.57	<0.001
Infarct size, %, median, IQR	10.50, 3.75-12.25	19.50, 16.00-23.75	<0.001

APV – average peak velocity, Pa – pressure aorta, Pd – pressure distal, CFR – coronary flow reserve, HMR – hyperemic microvascular resistance, FFR – fractional flow reserve, SD – standard deviation, IQR – interquartile range.

When intracoronary physiology parameters were compared between patients with large and small infarct size (**Table 3**), a tendency was observed towards higher HMR values and lower CFR values in patients with large infarcts, pointing towards an association of CMD with the size of infarction (2.58 ±0.84 mmHg/cm/s vs. 3.04 ±0.84 mmHg/cm/s, p=0.177, for HMR and 1.73 ±0.38 vs. 1.48 ±0.25, p=0.084, for CFR, in small and large infarctions, respectively).

This tendency was confirmed by a finding of moderate but significant correlation between both HMR (rho=0.569, p<0.01) and CFR (rho=-0.391, p=0.029) with infarct size expressed as a continuous variable (**Figure 1**).

Both HMR (AUC 0.669, 95%CI 0.472-0.866) and CFR (AUC 0.712, 95%CI 0.517-0.907) had similar capacity (p=0.718 for the comparison of ROC curves) to predict large infarct size (defined as ≥16% of the LV mass) (**Figure 2**).

Optimal cut-off values to predict large infarct size were 2.25 mmHg/cm/s for HMR and 1.45 for CFR. Median infarct size in the overall population was 11.50% (6.75-16.00) and it was significantly larger in patients with HMR ≥2.25mmHg/cm/s (15.00% vs. 4.00%, p<0.001). Similarly, patients with CFR <1.45 had significantly larger median infarct size compared with those with values above this cut-off (15.50% vs. 9.00%, p=0.007). In the univariable regression analysis, both HMR≥2.25 mmHg/cm/s and CFR≤1.45 were significantly associated with the infarct size as a continuous variable. However, when adjusted for clinical variables known at the time of primary PCI, HMR≥2.25 mmHg/cm/s remained an independent predictor, whereas CFR≤1.45 was not (**Table 4**).

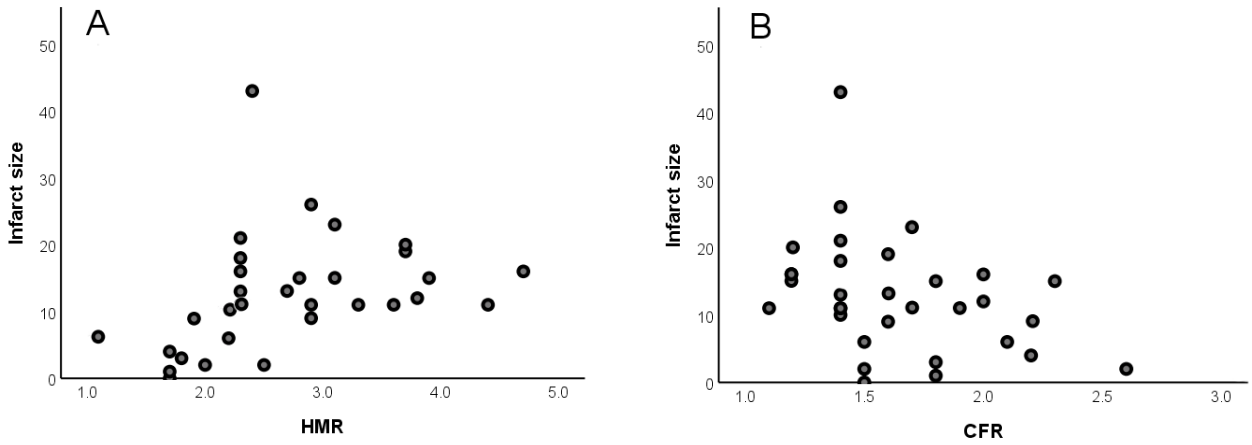

Figure 1. Significant correlation between coronary microvascular dysfunction and infarct size. **Panel A.** Increase in HMR values correlates with an increase in infarct size. **Panel B.** Decrease in CFR values correlates with an increase in infarct size.

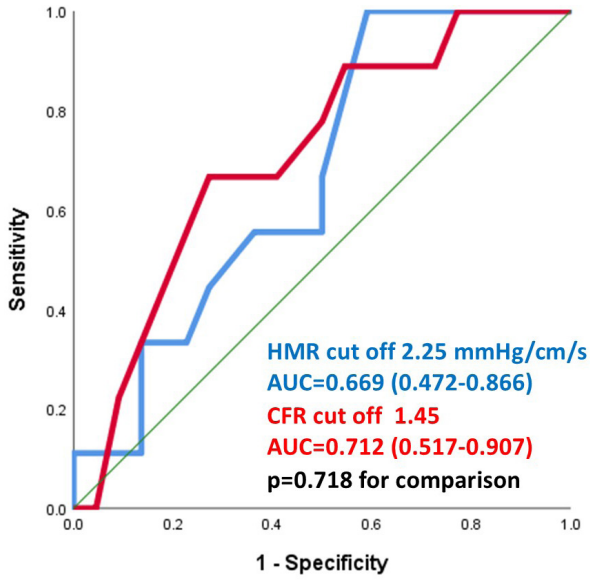

Figure 2. ROC curves comparison for the capacity of HMR vs. CFR to predict large infarct size.
Comparison of ROC curves showing similar capacity (p=0.718) of HMR (blue curve) and CFR (red curve) to predict large infarct size (≥16% of the left ventricular mass).

DISCUSSION

The main findings of our study are the following. First, both HMR and CFR, when measured at the end of a successful primary PCI, correlate significantly with infarct size. Second, HMR and CFR showed similar ability to predict large infarct size, defined as the highest quartile of the study population. Those findings need to be interpreted in the context of previous research in this field, which has both evaluated the prognostic value of different indices of microvascular function and explored its practical application as a means of patient stratification and therapy guidance to achieve better outcomes post primary PCI (5, 12, 13, 15, 16, 19, 20).

Teunissen et al. established the ability of HMR to predict microvascular injury (MVI) in a contemporary primary PCI setting, with values ≥2.5 mmHg/cm/s independently predicting MVI on CMR (19). MVI was defined akin to MVO, as hypointense areas within the infarcted, hyperintense myocardium on late gadolinium enhancement, assessed 4-6 days after primary PCI. The average HMR in the IRA immediately after recanalization was 2.87 ± 1.45 mmHg/cm/s, closely aligning with our findings of 2.72 ± 0.86 mmHg/cm/s. Post-primary PCI CFR was 1.80±0.80, also similar to our findings (1.65± 0.36). Although the primary result of Teunissen et al. was to demonstrate the ability of HMR, unlike CFR, to predict MVI, it also reported a significant correlation of HMR with infarct size on CMR on day 4-6 (r=0.41, p<0.01) and the significantly larger infarcts at 3 months in patients with HMR≥2.5 mmHg/cm/s (15.1% vs. 9.9%) (19). In our study, the correlation between HMR and infarct size on CMR at least 3 months after primary PCI was even stronger (rho=0.569, p<0.01; **Figure 1**). More-

Table 4. Predictors of infarct size*.

	Univariable** analysis		Multivariable analysis	
	Beta (95% CI)	p	Beta (95% CI)	p
Age	-0.045 (-0.685 – 0.594)	0.885		
Sex	2.000 (-9.492 – 13.492)	0.724		
Diabetes	-7.000 (-15.559 – 1.559)	0.105		
Hypertension	-2.000 (-11.136 – 7.136)	0.658		
Hyperlipidemia	0.000 (-10.109 – 10.109)	1.000		
Time to reperfusion	0.008 (-0.022 – 0.038)	0.573		
Infarct-related artery LAD	6.000 (2.369 – 15.631)	0.010	3.455 (0.967 – 11.033)	0.021
Incomplete ST-segment resolution	5.000 (-2.330 – 12.330)	0.174		
HMR≥2.25mmHg/cm/s	11.000 (5.413 – 16.587)	<0.001	8.000 (2.638 – 13.362)	0.005
CF≤1.45	-7.000 (-13.942 – 0.058)	0.048	-3.000 (-8.110 – 2.110)	0.239

*The reported results are from the quantile regression analysis with a quantile value set at 0.5.
**Baseline variables routinely obtained at the start of primary PCI or in the immediate periprocedural period are tested as predictors of infarct size, as measuring microvascular function during primary PCI may be actionable if it is of added value above and beyond the already known parameters. Those variables with p<0.100 from the univariable regression analysis are included into the multivariable regression analysis.

over, the ability to classify patients with large final infarct size (after at least 3 months) according to the study-specific HMR cut offs was very similar in both studies. In our study, patients with HMR≥2.25 mmHg/cm/s had average infarct size of 16% of the left ventricle mass, whereas patients with HMR≥2.5 mmHg/cm/s had average infarct size of 15.1% in the study by Teunissen et al (6). On the other hand, our study did demonstrate similar ability to predict infarct size by CFR, whereas no such data were reported by Teunissen et al. Our results reinforce earlier studies, which showed the ability of CFR measured with the Doppler wire in the IRA immediately after recanalization, as in our study, to predict LV structural and functional recovery after STEMI (21). At the same time, CFR was shown to predict MVO on CMR in other studies utilizing the thermodilution method, but with a very low specificity (34% and sensitivity of 79%) (22), likely explaining why in the study by Teunissen et al. HMR was a better predictor, especially of the high-extent MVO (specificity 65% and sensitivity 93%) (19). A possible explanation is that different indices, such as microvascular resistance (IMR or HMR) or flow reserve (CFR), may reflect different pathways of microvascular destruction (4), resulting in a variable association of individual indices with MVO or infarct size on CMR.

In practical terms, if invasive indices of microvascular function, which are available immediately after primary PCI, are associated with clinical outcomes, they can be used to guide adjunctive therapeutic strategies (13). Although previous studies suggested that CFR, HMR and IMR are associated with mortality and heart failure after primary PCI (6, 7, 22), recent comparisons indicated better ability of HMR (Doppler-derived hyperemic microvascular resistance) (6) and IMR (thermodilution-derived index of microcirculatory resistance) (15) over CFR to stratify patients according to the risk of mortality and heart failure. Recent efforts to standardize risk stratification after primary PCI favored IMR due to the wealth of studies utilizing this thermodilution-derived index, which has been seen as a more practical alternative to Doppler wire-based measurements that may require more time and effort to obtain (5, 13). However, the quest for an optimal index of microvascular injury to guide any adjunctive therapies targeting coronary microcirculation after STEMI is still on-going, as more information become available about the multifactorial process of microvascular injury and its impact on subsequent clinical events (4, 23, 24). The injury to coronary microvasculature may be associated with myocardial edema and eventually lead to intramyocardial hemorrhage, which has been more closely associated with increased mortality and heart failure than MVO (25). Intracardial edema and hemorrhage may exert extravascular pressure on the microvasculature (26), which may be best captured by pressure at zero flow (PzF), i.e., pressure at which antegrade coronary flow would cease, an index derived from simultaneous coronary pressure and flow tracing of a Doppler-tipped coronary guide wire. In a study by Patel et al., PzF (AUC=0.94) has been more closely related to large infarct size, defined as ≥24% of the LV mass, than HMR (AUC=0.74) or IMR (AUC=0.54) (16).

From the above-described studies comparing different indices of microvascular function, it seems to follow that a) PzF has the strongest capacity to predict infarct size (16), b) MVO on CMR can be predicted by both HMR (6) and IMR (15) better than CFR, and c) HMR and IMR seem to be superior to CFR in predicting mortality and heart failure (6, 22). Our study adds that CFR may have comparable ability to predict large infarct size as HMR. As large infarct size is associated with mortality and heart failure (18), our results support some of the previous studies showing the association of post-primary-PCI CFR with clinical outcomes (7). But the weaker association of CFR with MVO and intramyocardial hemorrhage, which both have been shown to predict mortality and HF independently of infarct size (10, 25), may explain the inferiority of CFR when compared to HMR and IMR in other studies.

In summary, our study did show comparable ability of invasively obtained HMR and CFR to predict large infarct size, which may be of practical relevance given the possibility of non-invasive echocardiography-derived CFR assessment (17) and the ability of CFR to provide prognostic information across a broad spectrum of cardiac diseases (27). However, our results should be interpreted with caution and in the aforementioned context of previous comparative analyses demonstrating better microvascular resistance indices (HMR and IMR) ability to predict MVO on CMR and clinical outcomes in the follow-up. Given the heterogeneity of post-STEMI changes in the myocardial structure and function, with infarct size, MVO and intramyocardial hemorrhage all being associated with mortality and heart failure, any one index of microvascular function may only represent a partial assessment of the damage conferred by the sequence of IRA occlusion and recanalization. Future research is needed to demonstrate which of the available invasive indices of coronary microcirculation may provide optimal patient stratification and therapeutic guidance at the end of the primary PCI.

The presented results need to be interpreted bearing in mind several important limitations. First, a limited number of patients. However, most of the original research that assessed indices of microvascular function in STEMI included a comparable patient population size (n=30-60). Second, no detailed MVO data were available for this analysis. Although our analysis did show a comparable predictive capacity between HMR and CFR in terms of predicting large infarct size, the correlation of CFR with infarct size was weaker, and CFR, unlike HMR, did not remain independently associated with infarct size after adjusting for other baseline predictors. Third, PzF was not available for this analysis. Given previous data on the supremacy of PzF over HMR and IMR, comparing PzF with HMR and CFR would have provid-

ed additional value to our analysis. Fourth, the known practical difficulty of routinely obtaining adequate intracoronary Doppler signals limits the applicability of our findings to everyday practice.

CONCLUSION

Invasive CFR and HMR, obtained by a Doppler-tipped coronary guide wire in the recanalized infarct-related artery, have similar capacity to predict large infarct size. Further research is needed to understand which index of microvascular function can be effectively used at the end of primary PCI to guide adjunctive therapies aimed at ameliorating microvascular injury and improving overall prognosis post-STEMI.

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Conflict of Interest Statement: No conflict of interest to report.

Author Contributions: DM performed data collection and analysis, as well as writing the darft and the final version of the manuscript.

Ethical approval: Ethics Committee of the University Clinical Center of Serbia (protocol code 747/11, 19 July 2018), and the research was conducted in accordance with the Declaration of Helsinki.

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POVEZANOST RAZLIČITIH INDEKSA DISFUNKCIJE KORONARNE MIKROCIRKULACIJE SA VELIČINOM INFARKTA NAKON PRIMARNE PERKUTANE KORONARNE INTERVENCIJE

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Sažetak

Uvod: Ranija istraživanja pokazala su da je kod bolesnika sa infarktom miokarda sa elevacijom ST-segmenta (STEMI), stepen disfunkcije koronarne mikrocirkulacije nakon primarne perkutane koronarne intervencije (PCI) povezan sa lošijom prognozom. Cilj ovog rada bio je da uporedi sposobnost dva različita indeksa disfunkcije koronarne mikrocirkulacije, koronarne rezerve protoka (CFR) i hiperemijske mikrovaskularne rezistencije (HMR), da predvide veličinu infarkta nakon primarne PCI.

Metode: Analiza je obuhvatila 31 bolesnika sa STEMI i validnim invazivnim merenjima brzine koronarnog protoka Dopler metodom na kraju primarne PCI, a kod kojih je magnetnom rezonancom srca (CMR) nakon najmanje 3 meseca procenjena veličina infarkta. Poređenjem ROC krivih ispitana je sposobnost CFR i HMR da predvide veliki infarkt miokarda, koji je bio definisan kao najviši kvartil analizirane populacije (≥16% mase leve komore).

Rezultati: Merenjem na kraju primarne PCI utvrđena je srednja vrednost HMR od 2.72±0.86 mmHg/cm/s, dok je u isto vreme srednja vrednost CFR bila 1.65± 0.36. Oba indeksa mikrovaskularne disfunkcije značajno su korelisala sa veličinom infarkta (rho=0.569, p<0.01, za HMR, i rho= -0.391, p=0.029, za CFR). Uporedna analiza ROC krivih pokazala je sličan kapacitet HMR i CFR da klasifikuju bolesnike prema riziku od velikog infarkta miokarda (AUC=0.669, 95%CI 0.472-0.866 za HMR vs. AUC=0.712, 95%CI 0.517-0.907 za CFR; p=0.718 za poređenje dva indeksa).

Zaključak: Vrednosti HMR i CFR dobijene invazivnim merenjem Dopler žicom nakon rekanalizacije infarktne arterije imaju sličnu sposobnost da predvide veliki infarkt nakon primarne PCI.

Ključne reči: koronarna mikrocirkulacija, veličina infarkta, primarna PCI

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