JACC: CARDIOVASCULAR INTERVENTIONS VOL. 3, NO. 7, 2010

ISSN 1936-8798/$36.00
PUBLISHED BY ELSEVIER INC. DOI: 10.1016/j.jcin.2010.04.009

The Index of Microcirculatory Resistance Measured Acutely Predicts the Extent and Severity of Myocardial Infarction in Patients With ST-Segment Elevation Myocardial Infarction

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Objectives This study investigated the relationship between the index of microcirculatory resistance (IMR) with myocardial injury and microvascular obstruction (MVO) assessed by contrast-enhanced cardiac magnetic resonance (ceCMR) imaging in a broad range of ST-segment elevation myocardial infarction (STEMI) patients undergoing emergency percutaneous coronary intervention (PCI).

Background Contrast-enhanced cardiac magnetic resonance imaging is the gold standard for assessment of microvascular obstruction (MVO), left ventricular (LV) ejection fraction, and infarct volumes in ST-segment elevation myocardial infarction (STEMI). However, ceCMR is not available acutely. The index of microcirculatory resistance is a simple invasive measure of microvascular function available at the time of emergency PCI. We investigated the relationship between IMR with myocardial injury and MVO assessed by ceCMR in STEMI patients undergoing emergency PCI.

Methods Fifty-seven patients with STEMI were included and 53 (93%) and 47 (82%) patients had complete ceCMR scans 2 days and 3 months following MI, respectively. Microvascular obstruction was defined as a dark core of hypoenhancement within the area of hyperenhanced infarct tissue 10 to 15 min following intravenous gadolinium (0.1 mmol/kg).

Results The median IMR (interquartile range [IQR]) was 35 (24 to 63) U. Twenty-seven patients (46%) had MVO. We found that IMR (median [IQR]) was higher in patients with MVO (38 [29 to 55] U) than in patients without MVO (27 [18 to 36] U); $p = 0.003$). The index of microcirculatory resistance was a negative multivariable predictor of LV ejection fraction, ($p \leq 0.001$) and infarct volume ($p = 0.01$) on the ceCMR scan 2 days after MI, and IMR was a multivariable predictor of LV ejection fraction ($p = 0.028$) and infarct volume ($p = 0.048$) at 3 months.

Conclusions The index of microcirculatory resistance measured acutely was higher in patients with MVO on ceCMR, and IMR independently predicted LV function and infarct volume. This easily measured physiological parameter provides important prognostic information at the time of emergency PCI. (J Am Coll Cardiol Intv 2010;3:715–22) © 2010 by the American College of Cardiology Foundation

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Manuscript received December 8, 2009; revised manuscript received March 9, 2010, accepted April 5, 2010.

Emergency percutaneous coronary intervention (PCI) is the established treatment for ST-segment elevation myocardial infarction (STEMI). Despite achieving normal epicardial artery flow in the majority of patients, up to one-third of patients do not achieve myocardial microvascular reperfusion (1,2). Microvascular dysfunction in this setting is associated with an adverse prognosis (3–5). However, there is no established method for evaluating the coronary microcirculation in STEMI patients in the cardiac catheterization laboratory.

The index of microcirculatory resistance (IMR) is a novel pressure-/temperature-tipped guidewire-based quantitative measure of coronary microvasculature function. The index of microcirculatory resistance has been validated in animal models and tested in stable patients (6–8), and in a recent study of acute STEMI patients, IMR was a better predictor of left ventricular (LV) function 3 months after MI than current standard methods for evaluating the microcirculation (9).

Abbreviations and Acronyms

ceCMR = contrast-enhanced cardiac magnetic resonance imaging
IMR = index of microcirculatory resistance
LV = left ventricular
MVO = microvascular obstruction
PCI = percutaneous intervention
STEMI = ST-segment elevation myocardial infarction
TIMI = Thrombolysis In Myocardial Infarction

Contrast-enhanced cardiac magnetic resonance imaging (ceCMR) is the gold standard noninvasive technique for assessment of the coronary microcirculation (10). Microvascular obstruction (MVO) on ceCMR following STEMI (Fig. 1) is associated with an adverse prognosis (11,12). However, for safety reasons, ceCMR is generally not performed until 2 or more days after hospital admission, limiting its clinical utility in the initial post-MI period.

In a broad range of STEMI patients, we aimed to determine whether IMR and other invasive physiological markers of microvascular dysfunction acquired immediately after stent deployment in emergency PCI for STEMI might predict the nature and severity of myocardial injury using ceCMR as the gold standard comparison. Specifically, we first hypothesized that an elevated IMR at the time of reperfusion would be associated with the nature of MI, as revealed by the occurrence of MVO on ceCMR. Second, we hypothesized that IMR would be independently predictive of the severity of MI, as subsequently revealed by infarct size and LV function on ceCMR during long-term follow-up.

Methods

Study design and population. The study population included patients with STEMI referred to our institution for primary PCI or after failed intravenous thrombolytic therapy for rescue PCI, or who underwent cardiac catheterization within 24 h of successful thrombolytic therapy. Patients were prospectively enrolled when the following inclusion criteria were present: 1) age ≥18 years with electrocardiographic and symptomatic evidence of acute STEMI and in whom emergency PCI was intended; and 2) written informed consent. Exclusion criteria were: 1) standard contraindications to CMR: pacemakers; cochlear implants; some types of prosthetic heart valves, surgical prostheses, or vascular clips; metal intraocular foreign bodies; 2) contraindications to gadolinium including estimated glomerular filtration rate <30 ml/min/1.73 m^2; sickle cell anemia; hemolytic anemia; 3) contraindications to adenosine; 4) cardiogenic shock; 5) previous MI in the index territory; and 6) pregnancy. The protocol was approved by the Institutional Review Board and informed consent was obtained from each patient.

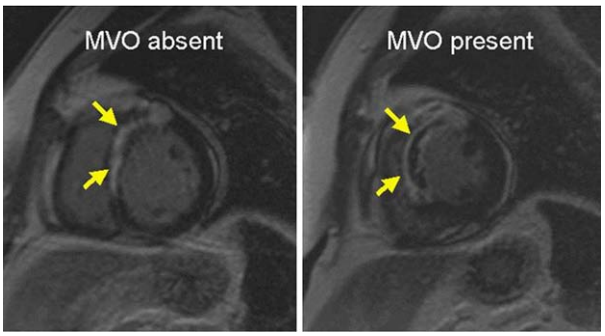

Figure 1. Late Gadolinium Enhancement of an Anterior MI Following Reperfusion

Late gadolinium enhancement of an anterior myocardial infarction 48 h following reperfusion. A microvascular obstruction (MVO) can be seen **(left)** as hypoenhancement within the hyperenhanced area. Axial views of anterior myocardial infarction (MI) revealed by late gadolinium contrast-enhanced magnetic resonance imaging (MRI). In the **left** image, there is no microvascular obstruction within the infarct zone, whereas in the **right** image there is a microvascular obstruction, as indicated by the **yellow arrows**. The central dark zone is due to the failure of the contrast agent to penetrate within the infarct core.

PHYSIOLOGICAL ASSESSMENT. Percutaneous coronary intervention was performed in line with current international guidelines. Glycoprotein IIb/IIIa inhibitors and thrombectomy catheters were used at the discretion of the primary operator. In the majority of cases, the coronary pressure-/temperature-sensitive guidewire (Radi Medical Systems, Uppsala, Sweden) was used as the primary guidewire. The guidewire was calibrated outside the body, equalized within the guide catheter, with the pressure sensor positioned at the ostium of the guide catheter. In all cases, the guidewire microsensor position was advanced into the distal third of the culprit artery. Great care was taken to ensure the position of the microsensor was constant throughout the procedure.

The IMR was calculated at the end of the procedure using the mean transit time (in seconds) of 3 × 3 ml bolus of room-temperature saline injected into the coronary ostium during maximal hyperemia as previously described (13). Meticulous attention was paid to guide catheter engagement. Adenosine 140 μg/kg/min was used to induce maximal hyperemia via a large peripheral vein. The mean aortic and distal coronary pressures were recorded during maximal hyperemia.

We defined IMR as the distal coronary pressure multiplied by the mean transit time of the 3-ml saline bolus at maximal hyperemia, measured simultaneously (mm Hg × s, or units). Subsequently, the stented segment was occluded with a short noncompliant balloon, and mean distal coronary wedge pressure was recorded. The pressure-derived collateral flow index was calculated as the ratio of the mean wedge pressure to mean aortic pressure at hyperemia (14).

CECMR PROTOCOLS. The initial scan was performed 24 to 48 h after PCI. Scanning was performed using a Siemens Sonata 1.5-T (Erlangen, Germany) scanner using a 6-channel anterior chest coil and spinal coils within the gantry table. Before scanning, electrocardiograph electrodes were positioned to achieve the best possible signal within the magnetic field of the scanner. Following the acquisition of localizing images, we obtained long- and short-axis cine images using a retrospectively gated breath-hold true fast imaging with steady precession sequence. The short-axis cine scans of 10-mm slices were used to determine LV mass, volume, and function.

A bolus of contrast medium, gadolinium-diethylenetriamine penta-acetic acid bismethylamide (Omniscan, Amersham Health, Oslo, Norway) was injected at a dose of 0.1 mmol/kg. Following the first pass, early gadolinium-enhancement images were obtained using a segmented true fast imaging with steady precession sequence across the LV short axis at 1-min intervals from 2 up to 5 min following the bolus. We then collected a series of images in both the long- and short-axis planes looking for delayed gadolinium enhancement using a turbo fast low angle shot sequence allowing assessment for the presence of MVO. We defined MVO as an area of hypoenhancement within the gadolinium hyperenhanced area of infarcted tissue present early after contrast injection and persistent when reimaged 15 min following contrast injection (Fig. 1) (12).

CECMR ANALYSIS. Left ventricular mass, volume, and function analyses were performed by a cardiologist experienced in ceCMR who was blinded to all clinical and pressure wire data. Results were obtained using Argus Dynamic Signal software (Siemens, Erlangen, Germany) by drawing endocardial and epicardial contours using the short-axis cine images at both end diastole and end systole. Images with late gadolinium enhancement were analyzed for the presence of MVO as previously defined by 2 experienced blinded observers, and a third experienced observer adjudicated when disagreement occurred. Infarct territories and MVO were manually delineated (11,12). Additionally, 2 other cardiologists experienced in ceCMR (S.W., C.B.) also reviewed the MVO analyses. For categorical analyses, MVO was defined as present or absent.

BIOCHEMICAL ASSESSMENT. Troponin I concentration in cubital venous blood was measured 12 to 24 h after MI using an automated analyzer (Advia Centaur, Bayer Diagnostics, Indianapolis, Indiana). The limit of detection was <0.2 μg/l.

STATISTICAL ANALYSIS. Invasive markers of microvascular function (coronary wedge pressure, pressure-derived collateral flow index, and IMR) were non-normally distributed and are summarized with the median value and interquartile range (IQR). The Mann-Whitney *U* test was used to compare these data between patients with or without MVO as revealed by ceCMR. An elevated IMR was defined as greater than the median value. Log-transformed invasive markers of microvascular function and other clinical features (such as those presented in Tables 3 and 4) were compared with ceCMR outcomes using univariate regression analysis. The univariable predictors with a p value of less than 0.1 were entered into a multivariate model. Because our focus was to evaluate the predictive value of IMR compared with other clinical characteristics that were available at the time of primary PCI, only variables that were clinically available at that time, such as time-to-reperfusion but not troponin concentration, were included in the multivariable models. A p value <0.05 was considered statistically significant. Statistical analyses were performed on MINITAB 14 software (Minitab, Inc., State College, Pennsylvania).

Results

Patient characteristics and procedure outcomes. Fifty-seven patients were prospectively enrolled and their clinical characteristics are presented in Table 1. Physiological measurements were acquired in all patients. In 1 patient, the coronary wedge pressure was not obtained due to a poor quality signal. As this patient underwent balloon angioplasty without stenting, it was not clinically appropriate to perform further balloon inflations necessary for wedge pressure measurement.

The culprit artery was the right coronary artery in 24 cases, the left anterior descending artery in 28 cases, the circumflex coronary artery in 4 cases, and the diagonal artery in 1 patient. Emergency PCI was for primary intervention in 26 cases, rescue in 16 cases, and prognostic (defined as

Table 1. Characteristics of 57 STEMI Patients

Mean age, yrs	58 (32–83)
Male sex, %	88
Current smoker, %	50
History of diabetes, %	6
Treated hypertension, %	27
History of hypercholesterolemia, %	27
TIMI flow grade 3 after PCI, %	94
Mean transit time (hyperemia), s	0.45 (0.35–0.68)
Distal coronary wedge pressure, mm Hg*	23 (17–30)
Pressure-derived collateral flow index*	0.28 (0.2–0.32)
Index of microcirculatory resistance	34.6 (22.8–51.1)
Fractional flow reserve after PCI	0.90 (0.85–0.97)
Adjunctive therapy during PCI, %	
Aspirin (300 mg)	100
Clopidogrel loading prior to PCI (600 mg)	100
Thrombectomy	46
Glycoprotein IIb/IIIa inhibitor therapy†	75
Medications at discharge, %	
ACE inhibitor	94
Beta-blocker	87
Statin	100

Continuous data for coronary microvascular function are expressed as median (interquartile range). *Available in 56 patients. †Glycoprotein IIb/IIIa inhibitor therapy was initiated in the cardiac catheter laboratory at the beginning or during PCI.

ACE = angiotensin-converting enzyme; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction; TIMI = Thrombolysis In Myocardial Infarction.

PCI within 24 h of successful thrombolytic therapy) in 15 cases. The median (IQR) time from onset of pain to reperfusion for patients undergoing primary or rescue PCI was 4.3 h (2.2 to 7.0 h). The IMR, pressure-derived collateral flow index, and distal coronary wedge pressure were not related to pain-to-reperfusion time.

Relationships between acute invasive measures of infarct severity and subsequent ceCMR findings. The median IQR for time from PCI to the ceCMR scan was 29 h (24 to 44) and the majority of patients (90%) underwent ceCMR within 48 h after PCI. In 2 patients, the magnetic resonance imaging scan was not well tolerated and gadolinium was not administered.

The coronary microcirculatory data are shown in Table 1. The median (IQR) IMR was 35 (24 to 63) U with a range of 7 to 154 U. The ceCMR findings 2 days and 3 months after MI are shown in Table 2.

The median IMR (IQR) in patients with MVO was higher (38 [29 to 55] U) than in patients without MVO (27 [18 to 36] U; p = 0.003) (Fig. 2). The pressure-derived collateral flow index and distal coronary wedge pressure were similar in both groups. Coronary microcirculatory characteristics were not related to MVO volume.

Relationship between acute invasive measures of infarct severity and a biochemical measure of myocardial injury. We found that IMR correlated with peak troponin I (r = 0.52, p = 0.01) (Fig. 3) but not with either the pressure-derived collateral flow index or distal coronary wedge pressure.

Table 2. Contrast-Enhanced CMR Findings in 57 STEMI Patients

ceCMR Variables	2 Days After MI	3 Months After MI
Left ventricular ejection fraction, %	55.2 (11.9)	61.9 (10.8)
Left ventricular end-diastolic volume index, ml/m^2	68.2 (13.6)	74.3 (17.8)
Left ventricular end-systolic volume index, ml/m^2	30.5 (10.8)	28.9 (12.4)
Left ventricular infarct volume, ml	23.1 (22.6)	12.1 (11.2)
Microvascular obstruction, n (%)	27 (51)	—

Continuous data are expressed as mean (SD). Data for MVO were available in 53 (93%) patients. ceCMR = contrast-enhanced cardiac magnetic resonance imaging; MI = myocardial infarction; MVO = microvascular obstruction; other abbreviations as in Table 1.

Univariable and multivariable regression models of IMR and ceCMR outcomes. In univariable analyses, an elevated IMR (i.e., IMR greater than median value of 35 units) was a negative predictor of LV ejection fraction and a positive predictor of LV end-systolic volume index (p = 0.035). In multivariable analyses, an elevated IMR was an independent predictor of LV ejection fraction 2 days and 3 months after MI (Table 3). An elevated IMR was a multivariable predictor of infarct size 2 days and 3 months after MI (Table 4).

Comparison of IMR in patient subgroups. The median IMR (IQR) was 40 (30 to 55) U in those who underwent rescue PCI, 36 (21 to 60) U in those who had primary PCI and 24 (17 to 35) U in those in whom PCI was performed within 24 h of successful reperfusion therapy on prognostic grounds (rescue PCI vs. prognostic PCI, p = 0.009; primary PCI vs. prognostic PCI, p = 0.04).

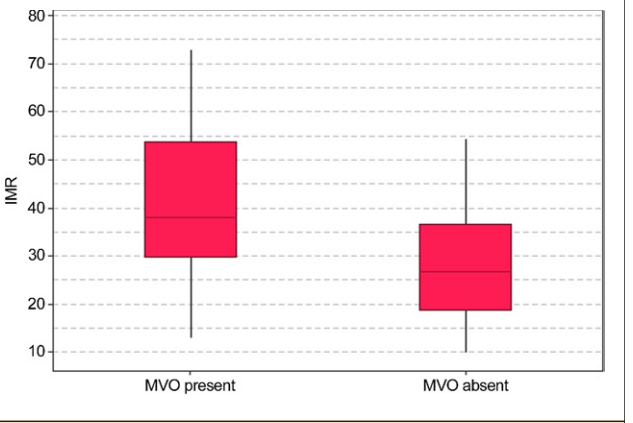

Figure 2. IMR in Patients With and Without MVO

The median (interquartile range) index of microcirculatory resistance (median [interquartile range]) was higher in patients with microvascular obstruction (38 [29 to 55] U) than in patients without microvascular obstruction (27 [18 to 36] U); p = 0.003). The **box limits** represent the interquartile range. The **horizontal line** within the box represents the median value. Abbreviation as in Figure 1.

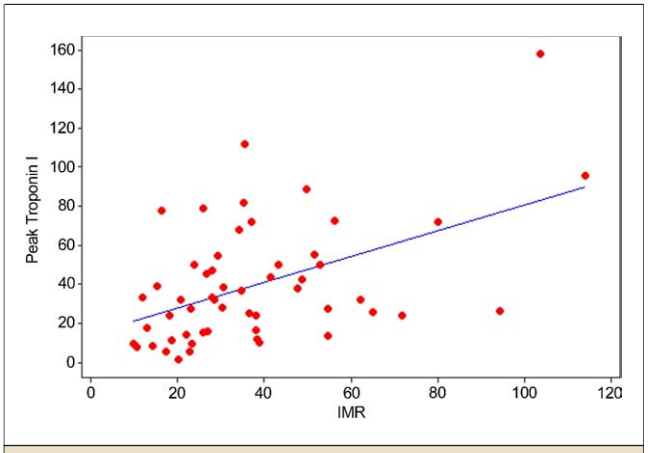

Figure 3. IMR Correlates With Peak Troponin I Concentration

The index of microcirculatory resistance correlates moderately well with peak troponin I concentration (r = 0.52; p = 0.01). The scatter plot shows peak troponin I concentration (μg/l) after myocardial infarction and the index of microcirculatory resistance obtained at the end of the primary percutaneous coronary intervention. Abbreviation as in Figure 2.

Relationships between IMR and initial TIMI flow grade. We found that IMR varied in relation to initial TIMI (Thrombolysis In Myocardial Infarction) flow grade and was higher in patients with no or minimal antegrade coronary flow (TIMI 0/1) than in patients with normal coronary flow (TIMI 3) at pre-PCI angiography ($p < 0.05$) (Table 5). However, IMR was similar in patients treated with or without aspiration thrombectomy or glycoprotein IIb/IIIa inhibitor therapy.

Table 3. Results of Multivariable Regression Analyses for LVEF Measured by ceCMR Performed 2 Days and 3 Months After MI

	Univariate R^2 (%)	Univariate p Value	Multivariate p Value*
Predictors of LVEF at day 2			
Cigarette smoking	5.4	0.08	0.37
Diabetes	12.1	0.008	0.025
IMR	29.1	<0.001	<0.001
Time to reperfusion	9.5	0.02	0.096
ST-segment resolution >70%	7.7	0.037	0.21
Predictors of LVEF at 3 months			
Hypercholesterolemia	9.4	0.01	0.016
Glycoprotein IIb/IIIa inhibitor	10.8	0.02	0.031
IMR	14.5	0.02	0.028
Time to reperfusion	9.1	0.035	0.446
ST-segment resolution >70%	12.3	0.013	0.175
Culprit artery	16.8	0.003	0.039

The variables entered into univariable models for LVEF measured by ceCMR at 2 days and 3 months follow-up, respectively, were age, male sex, cigarette smoking, hypercholesterolemia, hypertension, diabetes, glycoprotein IIb/IIIa inhibitor therapy, aspiration thrombectomy, IMR, fractional coronary collateral supply, coronary wedge pressure, culprit artery, time to reperfusion, ST-segment resolution >70%, and culprit artery. The strongest univariate predictors with a p value less the 0.1 were entered into a multivariate model to determine independent predictors. *The rise in R^2 value for each multivariate model including IMR as compared to all other variables was 37.1% to 50.8% at 2 days and 54.5% to 58% at 3 months.

IMR = index of microcirculatory resistance; LVEF = left ventricular ejection fraction; other abbreviations as in Table 2.

Table 4. Results of Multivariable Regression Analyses for Myocardial Infarct Volume Measured by ceCMR Performed 2 Days and 3 Months After MI

	Univariate R^2 (%)	Univariate p Value	Multivariate p Value*
Predictors of infarct volume at day 2			
Glycoprotein IIb/IIIa inhibitor	5.3	0.09	0.29
IMR	18.6	<0.001	0.01
Time to reperfusion	11.1	0.016	0.32
ST-segment resolution >70%	14.7	0.005	0.06
Culprit artery	14.2	0.006	0.03
Predictors of infarct volume at 3 months			
Hypercholesterolemia	9.1	0.04	0.112
IMR	15.6	0.006	0.048
ST-segment resolution >70%	9.9	0.031	0.384
Time to reperfusion	6.0	0.097	0.545
Culprit artery	21.3	0.001	0.021

The units of myocardial infarct volume are milliliters. The following variables were entered into the multivariate models for myocardial infarct volumes measured by ceCMR 2 days and 3 months after MI: age, male sex, cigarette smoking, hypercholesterolemia, hypertension, diabetes, glycoprotein IIb/IIIa inhibitor therapy, aspiration thrombectomy, IMR, fractional coronary collateral supply, coronary wedge pressure, time to reperfusion, ST-segment resolution >70%, and culprit artery. The strongest univariate predictors with a p value less the 0.1 were entered into a multivariate model to determine independent predictors. *The rise in R^2 value for each multivariate model including IMR as compared to all other variables was 51.3% to 53.9% at 2 days and 40.7% to 45.1% at 3 months.

Abbreviations as in Tables 2 and 3.

Discussion

Microcirculatory injury is a determinant of LV function (3,15) and prognosis following acute MI (4,12). Whereas emergency PCI represents 1 of the first opportunities to evaluate and treat an acute MI patient, the optimal method for evaluating the microcirculation in this setting is uncertain (16).

The key findings in our study are as follows. First, in a broad range of STEMI patients undergoing emergency PCI, an invasive measure of microvascular function, the

Table 5. The Relationships Between IMR and pre-PCI TIMI Flow Grade

TIMI Flow Grade	n	Median IMR (IQR)
0	21	37 (26.9–53.1)
1	17	34.8 (24.9–63.6)
2	10	32.1 (15–50.9)
3	9	23.1 (17.3–31.6)

IQR = interquartile range; other abbreviations as in Tables 1 and 3.

IMR, was linked with the pathological nature of MI, because IMR was higher in patients with MVO than in those without MVO, as revealed by ceCMR. Second, IMR independently predicted the severity of MI as revealed by infarct volume and LV function 2 days and 3 months after MI. Third, IMR, but not coronary wedge pressure or fractional collateral supply, independently predicted infarct size.

To date, IMR has been subject to pre-clinical and clinical validation, and these studies have demonstrated that IMR measurement is largely independent of variations in hemodynamic state (6–8). A seminal study by Fearon et al. (9) confirmed the IMR to be superior to other commonly used clinical markers of microvascular dysfunction for predicting LV function assessed by echocardiography.

Our study is the first to compare invasively acquired measurements of microvascular injury using the coronary pressure wire with LV function and MVO assessed by paired ceCMR studies obtained acutely and after longer-term follow-up. Contrast-enhanced CMR is recognized as the current noninvasive gold standard for assessment of coronary microvascular damage and LV function and dimensions (10). A recent study by Hirsch et al. (17) quantified coronary blood flow characteristics using an intracoronary Doppler wire and related these findings to microvascular injury assessed by ceCMR. Twenty-seven anterior STEMI patients underwent repeat cardiac catheterization 4 to 8 days after primary PCI with ceCMR performed the preceding day. This study demonstrated that the extent of MVO was a multivariable predictor of abnormal coronary flow velocity characteristics, providing further functional evidence of the validity of ceCMR as a noninvasive tool to assess microcirculatory function. These observations complement those in the current study.

Here, IMR predicted LV injury as assessed by ceCMR and correlated significantly with troponin I, which is a biochemical marker of infarct size. Our study is also the first direct comparison of IMR with other physiological measures of microvascular dysfunction (18). The other postulated coronary pressure/temperature wire-derived markers of microvascular injury (pressure-derived collateral flow index and distal coronary wedge pressure) did not correlate with the severity or nature of MI as assessed by ceCMR.

Our findings complement those of the other previously published studies in a number of important ways. Our study had a larger sample size and included patients with a broader range of acute MI types (e.g., nonanterior STEMI). The time to presentation from the onset of symptoms for patients in our study was less restricted than in previous studies that excluded patients with symptoms >12 h (9,17,19). Therefore, our results extend the potential clinical relevance of IMR measurement to a larger group of patients

Figure 4. Thermodilution Curves at Rest and in Hyperemia

Thermodilution curves under resting conditions **(blue lines)** and during hyperemia induced by intravenous adenosine infusion. Graphical representation of thermodilution curves at baseline and hyperemia (seconds) as seen in the catheter laboratory at the time of percutaneous coronary intervention displayed on the RADI analyzer (Radi Medical Systems, Uppsala, Sweden).

with MI. The median IMR in our study population was 35 units, which is similar to that reported by Fearon et al. (9) (median IMR: 32 U). Our findings are consistent with and extend the results of these earlier studies and support the notion that standardized measurement of IMR may be valid in clinical practice. Consequently, our results support the notion that invasive measures of microvascular function may be useful clinical tools to predict the initial severity of myocardial injury.

Only a minority of patients had a history of hypertension or diabetes and patients with previous MI in the culprit artery territory were not included. Therefore, the microvascular dysfunction observed in our patients was most likely influenced by the effects of acute coronary thrombosis and reperfusion, rather than the effects of chronic microvascular disease related to pre-existing cardiac disease.

Study limitations. Microvascular obstruction is a dynamic phenomenon following coronary reperfusion, and MVO that may be apparent on ceCMR initially may resolve by 48 h. In fact, MVO that is detectable 2 days after MI is more correctly termed persistent MVO. The dynamic nature of MVO may in part explain why elevated IMR values >35 may occur in some patients with no visible MVR on ceCMR scanning 2 days later (20). Serial ceCMR up to 48 h after MI would be required to answer this question. The time point used to assess MVO by ceCMR is consistent with other studies of this subject, and MVO measured at this time point is an adverse prognostic marker (4).

The study is limited by its small sample size; however, IMR remains a significant predictor of myocardial damage. A larger sample size may have resulted in other predictors becoming significant. Larger studies will be needed to determine whether IMR can predict clinical outcomes.

Conclusions

The index of microcirculatory resistance is a simple wire-based technique that can provide a quantitative assessment of microvascular function at the time of emergency PCI. We have shown that an elevated IMR is linked to MVO as revealed by ceCMR. MVO independently predicts long-term prognosis following MI. Accordingly, we suggest measurement of IMR represents a new approach to risk assessment at the very earliest stage of acute MI management, and, therefore, potentially enables a triage of higher risk patients to more intensive therapy (Fig. 4).

Acknowledgments

The authors thank the staff in our Interventional Service and the Chief Scientist Office (Scotland) for funding this work.

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Key Words: myocardial infarction ■ index of microcirculatory resistance ■ magnetic resonance imaging.