A simple angiographic index to predict adverse clinical outcome associated with acute myocardial infarction

Akut miyokart enfarktüsüne bağlı kötü klinik sonlanımı öngörmede basit bir anjiyografik endeks

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ABSTRACT

Objectives: The major determinant of final infarct size for a given coronary occlusion is the size of the myocardial area-at-risk. We propose herein a new index 'Relative Importance Index (RII)' to predict area-at-risk in patients with anterior myocardial infarction (MI). The aim of the study was to assess the predictive value of RII in left ventricle (LV) systolic function reduction and its relation to adverse clinical outcome.

Study design: One hundred twenty-three acute anterior MI patients with their first acute coronary syndrome incident were consecutively and prospectively enrolled in to the study. RII was calculated by dividing the culprit segment diameter by the sum of diameters of the left anterior descending, circumflex, and right coronary arteries at their proximal segments. We evaluated the one-month follow-up rates of major clinical endpoints, which were defined as death, non-fatal MI, stroke, and new congestive heart failure (CHF).

Results: RII was significantly and negatively correlated with left ventricular ejection fraction (LVEF) (r=-0.65, p<0.001). Likewise, RII was significantly correlated with 72 hour troponin I (TnI) (r=0.48, p<0.001). Patients were dichotomized according to the median value of RII (median RII: 0.30). Supra-median RII was associated with lower EF (32.8±8.6 *vs.* 42.8±9.4, p<0.001) and higher incidence of composite major adverse cardiac events (33.9% *vs.* 13.1%, p=0.01). The mortality, non-fatal MI and new CHF rates in the supra-median RII group trended higher but they did not reach statistical significance. An RII >0.30 had an 88% sensitivity and 60% specificity (ROC area: 0.82, p<0.001, CI: 0.73-0.90) for predicting severe LV dysfunction (LVEF <30%).

Conclusion: A simple index derived from coronary angiography at the time of primary percutaneous coronary intervention can predict LV systolic function loss and adverse clinical outcome in patients with acute anterior MI.

ÖZET

Amaç: Akut miyokart enfarktüsünde, nihai enfarkt büyüklüğünün ana belirteçlerinden birisi ilgili damarın beslediği risk altındaki miyokart alanının büyüklüğüdür. Akut ön duvar miyokart enfarktüsü (ME) geçiren hastalarda risk altındaki miyokart alanını öngörebilecek yeni bir endeks tanımladık. Çalışmamızın amacı, "Kısmi önemlilik endeksi (KÖE)" adını verdiğimiz bu endeksin klinik sonlanım ve sol ventrikül (SV) sistolik fonksiyonlarındaki düşme ile ilişkisini araştırmaktır.

Çalışma planı: Daha önce akut koroner sendrom hikayesi olmayan ve ön duvar ME'si geçiren ardışık 123 hasta ileriye dönük bir şekilde çalışmaya alındı. KÖE, sorumlu segmentteki damar çapının sol ön inen, sirkumfleks ve sağ koroner arter çaplarının toplamına bölünmesi ile hesaplandı. Birinci aydaki ölüm, ölümcül olmayan ME, inme ve konjestif kalp yetersizliğini (KKY) içeren klinik sonlanım noktalarına bakıldı.

Bulgular: Kısmi önemlilik endeksi ile SV ejeksiyon fraksiyonu (EF) arasında anlamlı ve negatif bir korelasyon saptandı (r=-0.65, p<0.001). Benzer şekilde KÖE, 72. saat troponin I (TnI) ile anlamlı bir şekilde korele idi (r=0.48, p<0.001). Hastalar ortanca KÖE değerine (Ortanca KÖE=0.30) göre iki gruba ayrıldı. Ortanca üstü grupta ortalama EF daha düşük (32.8±8.6 ve 42.8±9.4, p<0.001) ve birleşik sonlanım noktasına erişme sıklığı daha fazla idi (%33.9 ve %13.1, p=0.01). Ortanca üstü grupta ölüm, ölümcül olmayan ME ve KKY sıklığı daha fazla olmakla birlikte fark istatistiksel olarak anlamlı değildi. Kısmi önemlilik indeskinin >30 olması ileri derece SV işlev bozukluğunu (SVEF <0.30) öngörmede %88 duyarlılık ve %60 özgüllüğe sahipti (ROC alanı 0.82, p<0.001, GA: 0.73-0.90).

Sonuç: Akut ön duvar ME'li hastalarda, primer perkütan koroner girişim esnasında hesaplanabilen basit bir endeks SV işlev bozukluğunu ve kötü klinik gidişi öngörebilir.

Received: August 06, 2013 *Accepted:* November 20, 2013

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Acute myocardial infarction (MI) indicates irreversible myocardial injury resulting in necrosis of a significant portion of the myocardium secondary to occlusion of an epicardial artery. The area of infarct occurs in the distribution of the occluded artery, which typically starts at the subendocardium and progresses towards the epicardium in a wave-front fashion.[1] Clinical outcome after acute MI depends largely on final infarct size and is improved with early reperfusion. The major determinant of final infarct size for a given coronary occlusion is the size of the myocardial area-at-risk (AAR).[2,3] Therefore, determination of both initial AAR and final infarct size after acute MI has major clinical implications since it permits an accurate assessment of myocardial salvage provided by reperfusion therapies. Final infarct size is always less than the initial AAR, and can reliably be assessed by delayed enhanced cardiac magnetic resonance imaging (DE-CMR).[4] However, determination of AAR in the clinical setting remains challenging. Although single-photon emission computed tomography (SPECT) is an accepted technique, endocardial surface area on DE-CMR has been proposed recently for measurement of AAR.[5,6] Likewise, a number of angiographic jeopardy scores have been developed to estimate the amount of myocardium at risk and predict patient-based clinical outcomes.[7] However, the clinical applicability of the current angiographic scoring systems is limited, especially in the setting of acute MI, owing to their sophisticated algorithm. Therefore, we propose herein a simple angiographic index, the 'Relative Importance Index (RII)', to predict the relative contribution of an arterial segment to the perfusion of the heart by proportioning the culprit segment diameter to the total coronary artery diameter.

The primary aim of this study was to determine whether there is a relationship between the extent of left ventricular ejection fraction (LVEF) reduction and RII of the left anterior descending artery (LAD) in patients with anterior MI. We secondarily assessed the relation of RII with adverse clinical outcome.

Abbreviations:

AAR	*Area-at-risk*
CHF	*Congestive heart failure*
DE-CMR	*Delayed enhanced cardiac magnetic resonance imaging*
EF	*Ejection fraction*
IM	*Intermediary*
IRA	*Infarct- related artery*
LAD	*Left anterior descending artery*
LVEF	*Left ventricular ejection fraction*
MCE	*Major clinical endpoints*
MI	*Myocardial infarction*
PCI	*Percutaneous coronary intervention*
QCA	*Quantitative Coronary Analysis*
RCA	*Right coronary artery*
RII	*Relative Importance Index*
ROC	*Receiver operating characteristic*
SPECT	*Single-photon emission computed tomography*
TIMI	*Thrombolysis in Myocardial Infarction*
TnI	*Troponin I*

PATIENTS AND METHODS

Study population

From May 2012 to February 2013, we prospectively and consecutively enrolled 123 subjects with their first ST-segment elevation acute anterior wall MI admitted for primary percutaneous coronary intervention (PCI) at our high-volume invasive heart center. We did not include subjects with a clinical history of congestive heart failure (CHF), valvular heart disease, previous coronary revascularization, or prior MI. Criteria for inclusion in the current study were (1) more than 30 minutes (min) of chest pain and electrocardiographic ST-segment elevation ≥0.1 mm in at least two adjacent anterior leads, (2) PCI within 12 hours (h) from symptom onset, and (3) presence of Thrombolysis in Myocardial Infarction (TIMI) 0 and 1 flow at time of PCI. We excluded subjects with pre-procedural TIMI flow ≥2 in the infarct- related artery (IRA) and subjects in whom symptoms-to-reperfusion time could not be determined precisely. Subjects with chronic total occlusion of another arterial territory and with angiographically visible collateral flow to the IRA (those with >0 flow grade according to Rentrop's classification) were also excluded. The study was approved by the local ethics committee, and all individuals included in the study provided written consent for their participation.

Coronary angiography and PCI procedure

All subjects were pre-treated with aspirin and a weight-adjusted dose of unfractionated heparin. A standard catheterization procedure with multiple selective contrast injections in the right and left coronary artery system was performed with size 6F diagnostic catheters of the same manufacturer (Boston Scientific Corporation; Natick, MA, USA) before PCI of the culprit lesion in all cases. Predilatation of the culprit lesion, PCI technique, and administration of glycoprotein IIb/IIIa receptor inhibitors were left to the discretion of the operator. With the exception of failed procedures and operator's discretion to withhold stenting, all subjects received at least one stent.

Additional PCI for a lesion other than the culprit lesion was discouraged at the time of primary PCI.

Blood sampling

Troponin I (TnI) was obtained serially on presentation, every 12 h in the first 2 days, and every 24 h for 4 days or longer as is routine according to our coronary care unit (CCU) standards. As a serological estimate of infarct size, TnI concentration 72 h after MI was chosen.[8] Cardiac TnI was determined by a commercially available assay (Siemens Ultra assay, ADVIA Centaur immunoassay system, Siemens Healthcare Diagnostics) with a limit of detection of 0.006 lg/L (=6 ng/L) (measuring range 6-5,000 ng/L), a 99th percentile cut-off value of 0.04 lg/L (=40 ng/L), and a coefficient of variation of <10% at 30 ng/L, as specified by the manufacturer. All measurements in this study were performed at our hospital's central core laboratory.

Echocardiography

All the patients underwent echocardiographic evaluation for determination of residual LVEF at the 1st month follow-up. Echocardiographic examinations were done according to the recommendations of the American Society of Echocardiography and European Association of Echocardiography guidelines[9] by two operators who were blinded to the angiographic data. For those who did not survive until the 1st month, in-hospital echocardiography records were used. LVEF was measured by biplane Simpson's method. Patients with residual LVEF <30% were considered to have severe systolic dysfunction.

Clinical endpoints and definitions

We evaluated one-month follow-up rates of major clinical endpoints (MCE), which were defined as death, non-fatal MI, stroke, and new CHF. New CHF was defined as any new diagnosis of CHF with one or more of the following disorders necessitating treatment with diuretics: cardiogenic shock, pulmonary edema or congestion on chest radiograph, rales more than 1/3 from lung base (Killip class ≥2), pulmonary capillary wedge pressure greater than 25 mmHg, and dyspnea with oxygen saturation lower than 90% without supplemental oxygen in the absence of lung disease. In patients discharged after the index event, any CHF necessitating readmission to the hospital within the first month was recorded.

Quantitative coronary analysis

Quantitative Coronary Analysis (QCA) was performed with Scientific QCA Analysis software incorporated to an Artis Zee Angiography System (Siemens Medical Systems; Forchheim, Germany), by one investigator who was blinded to the echocardiographic results. Coronary end-diastolic frames from views without shortening or overlap were selected for QCA measurement. Care was exercised to choose a projection where the vessel and the catheter tip run in a plane nearly parallel to the X-ray tube. Calibration was performed by contrast-filled 6 F diagnostic catheter of the same manufacturer (Boston Scientific Corporation; Natick, MA, USA) as a scaling device. The proximal diameters of the major coronary vessels (LAD, left circumflex artery (LCx), right coronary artery (RCA), and intermediary artery (IM) if present) were measured in all patients. The proximal segment of the LAD and LCx was defined as vessel segment immediately beyond the bifurcation of the left main coronary artery (LMCA). In case of any disease in this segment, the first disease-free segment before the major bifurcation of the individual vessel was used for measurement. When LAD is ostially occluded thereby precluding measurement, we used an appropriately inflated stent size as a surrogate of the LAD diameter. The proximal segment of the RCA was defined as the vessel segment 1 to 2 cm distal to the coronary ostium before any major branch take-off except for conus branch. Patients with diffuse disease at proximal segments of coronary arteries that precluded defining a reference segment were excluded from the study. The culprit segment diameter was measured at the first disease-free reference segment before the occlusion. Total coronary artery diameter was defined as the sum of the diameters of LAD, LCx, RCA, and IM if present. RII for the culprit lesion was calculated by dividing the culprit segment diameter by the total coronary artery diameter (Figure 1). Twenty angiograms were randomly selected and measurements were repeated by another investigator for assessment of the reproducibility of RII measurements.

Statistical analysis

Continuous variables were tested for normal distribution using the Kolmogorov–Smirnov test. Variables not normally distributed were expressed as medians (interquartile ranges). Normally distributed continuous variables were expressed as mean values ± 1

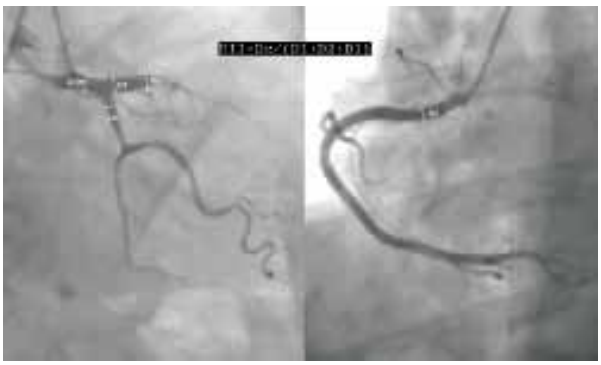

Figure 1. Calculation of RII. Dc, culprit segment diameter; D1, left anterior descending artery diameter; D2, circumflex artery diameter; D3, right coronary artery diameter; D lm, left main coronary artery diameter; RII, relative importance index.

standard deviation. Categorical variables were summarized as frequency percentages and absolute numbers. Correlation of RII with LVEF and 72-h TnI were assessed by Spearman's correlation analysis. Patients were dichotomized according to median value of RII. The Pearson χ^2-test was used to compare categorical variables. The means for normally distributed continuous variables were compared by Independent-Samples T-test. Skew-distributed continuous variables were compared using a Mann-Whitney U-test. Receiver operating characteristic (ROC) curve analysis was performed to define corresponding specificity and sensitivity of RII ≥30 for predicting severe systolic dysfunction (LVEF <30%). Intra- and inter-observer reproducibility was assessed by Cronbach's alpha coefficient. A two-tailed p value of less than 0.05 was considered statistically significant. SPSS software (Statistical Package for the Social Sciences, version 15.0, SSPS Inc.; Chicago, IL, USA) was used for all statistical calculations.

RESULTS

There were 29 females and 94 males in the study population. The mean age was 62.1±14.5 years. The

Table 1. Demographic and clinical characteristics of the study participants (n=123)

	n	%	Mean±SD
Age, years			61.2±14.5
Sex			
Female	29	23.6	
Male	94	76.4	
Hypertension	49	39.8	
Dyslipidemia	35	28.5	
Diabetes mellitus	20	16.3	
Smokers	52	42.3	
LVEF (%)			37.8±10.2
TIMI flow before PCI			
0	112	91.1	
1	11	8.9	
Affected arterial segment			
Proximal LAD	54	43.9	
Mid LAD	51	41.5	
Distal LAD	14	11.4	
Diagonal branch	4	3.3	
Mean door-to-balloon time, min			49±9
Median symptom-to-balloon time, h*	4	3-6	

LAD: Left anterior descending artery; LVEF: Left ventricular ejection fraction; PCI: Percutaneous coronary intervention; TIMI: Thrombolysis in myocardial infarction. Data are expressed as number (%) or mean±standard deviation. *Data are expressed as median and interquartile ranges.

demographic and clinical characteristics of the study participants are presented in Table 1. Affected arterial segments were the proximal LAD in 54 (43.9%) cases, mid LAD in 51 (41.5%) cases, distal LAD in 14 (11.4%) cases, and diagonal branch in 4 (3.3%) cases. Mean RIIs for corresponding lesions at the proximal, mid and distal LAD and diagonal branch were different (0.33±0.04, 0.28±0.04, 0.25±0.03, and 0.20±0.07, respectively, p≤0.001).

Figure 2 shows the negative correlation between RII and EF. As RII of the culprit lesion increased, there was tendency to end with a lower EF (r=-0.65, p<0.001). Likewise, RII was significantly correlated with 72-h TnI (r=0.48, p<0.001) (Figure 3). Patients were dichotomized according to median value of RII (median RII: 0.30) (Table 2). Supra-median RII was associated with lower EF (32.8±8.6% *vs.* 42.8±9.4%, p<0.001), higher 72-h TnI (42±24 ng/ml *vs.* 24±16 ng/ml, p<0.001) and higher incidence of composite MACE (33.9% *vs.* 13.1%, p=0.01). Infra-median RII was associated with higher incidence of preserved systolic function, whereas supra-median RII was associated with higher incidence of severe systolic dysfunction. There were no significant differences between supra- and infra-median RII groups with regard to age, sex, smoking status, and presence of hypertension and diabetes mellitus. PCI technique, use of interventional device (coronary stents, thrombectomy catheters), PCI success, and medications were similar in both groups. The mortality (12.9% *vs.* 6.6%), non-fatal MI (6.5% *vs.* 3.3%), and new CHF (12.9% *vs.* 3.3%) rates in the supra-median RII group trended higher but did not reach statistical significance, probably due to the small sample size. An RII ≥0.30 had

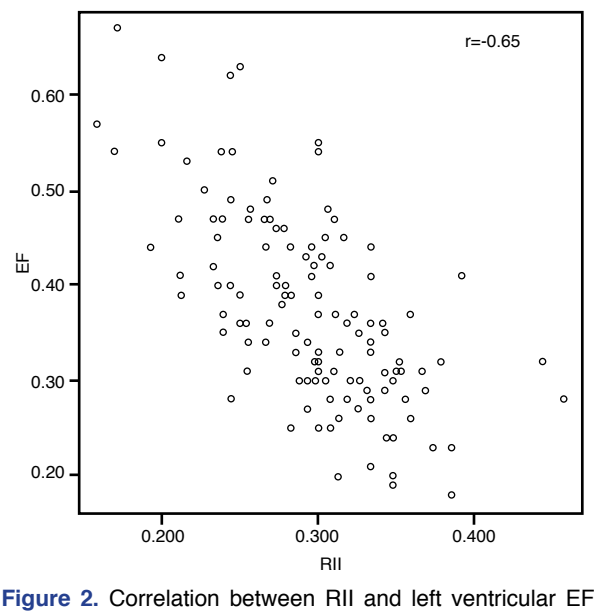

Figure 2. Correlation between RII and left ventricular EF (r=-0.65, p<0.001). RII: Relative importance index of culprit lesion.

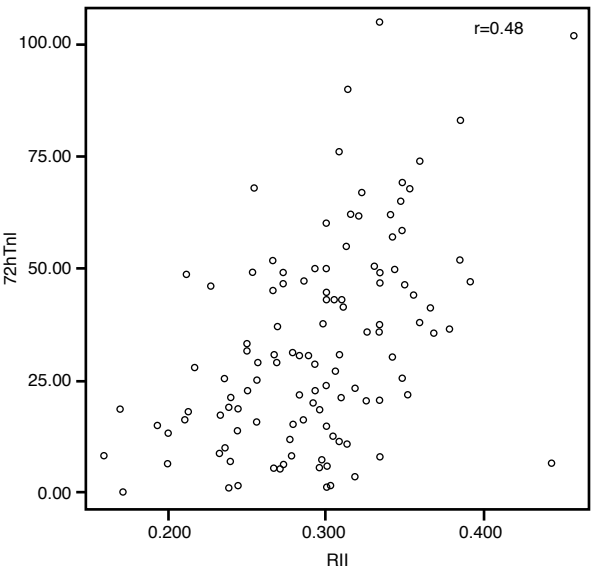

Figure 3. Correlation between RII and 72-h TnI (r=0.48, p<0.001). RII: Relative importance index of culprit lesion.

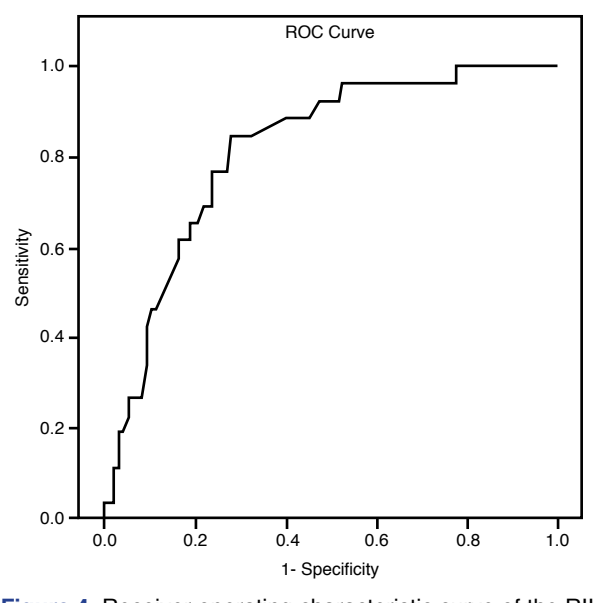

Figure 4. Receiver operating characteristic curve of the RII in predicting severe LV dysfunction (EF <30%). RII: Relative importance index; LV: Left ventricle.

Table 2. Clinical and laboratory characteristics in the two RII groups

	RII <0.30 (n=61)		RII ≥0.30 (n=62)		p
	n	%	n	%	
LVEF (%)	42.8±9.4		32.8±8.6		**<0.001**
LVEF ≥45%	24		7		**<0.001**
LVEF <30%	3		23		**<0.001**
Age (years)	62±14		63±15		NS
Sex (Female/Male)	17/44		12/50		NS
Hypertension	26		23		NS
Dyslipidemia	16		19		NS
Diabetes mellitus	9		11		NS
Smokers	24		28		
72-h TnI (ng/ml)	24±16		42±24		**<0.001**
Final TIMI flow					
TIMI 3	46	75.4	45	72.6	NS
TIMI 2	7	11.5	8	12.9	NS
TIMI 1	1	1.6	1	1.6	NS
TIMI 0	7	11.5	8	12.9	NS
Door-to-balloon time (min)	48±10		50±9		NS
Symptom-to-balloon time (h*)	4	3-6	3.5	3-6	NS
Drug use					
ASA	61	100	61	98.4	NS
Clopidogrel	61	100	62	100	NS
β-blocker	58	95.1	54	87.1	NS
ACE inhibitor	59	96.7	57	91.9	NS
Statin	60	98.4	62	100	NS
Glycoprotein IIb/IIIa inhibitors	6	9.8	8	12.9	NS
Device use					
Thrombus aspiration	3	4.9	5	8.1	NS
Patients with BMS	18	29.5	22	35.5	NS
Patients with DES	31	50.8	32	51.6	NS
MCE					
Death	4	6.6	8	12.9	NS
Stroke	0	0	1	1.6	NS
Non-fatal MI	2	3.3	4	6.5	NS
New CHF	2	3.3	8	12.9	NS
Composite MCE	8	13.1	21	33.9	0.01

ACE: Angiotensin converting enzyme; ASA: Acetylsalicylic acid; BMS: Bare metal stent; CHF: Congestive heart failure; DES: Drug-eluting stent; LVEF: Left ventricular ejection fraction; MCE: Major clinical events; MI: Myocardial infarction; NS: Non-significant; RII: Relative importance index; TnI: Troponin I; TIMI: Thrombolysis in myocardial infarction. Data are expressed as number (%) or mean ± standard deviation. *Data are presented as median and interquartile ranges.

an 88% sensitivity and 60% specificity (ROC area: 0.82, p<0.001, confidence interval (CI): 0.73-0.90) for predicting severe LV dysfunction (LVEF <30%) (Figure 4).

Intra- and inter-observer correlation coefficients for coronary artery diameter measurement were found to be 0.95 and 0.91, respectively. Intra- and inter-observer correlation coefficients of RII measurements were 0.95 and 0.92, respectively.

DISCUSSION

In the present study, we defined an index to predict LV systolic function loss and adverse clinical outcome in patients with acute anterior MI. Final infarct size following acute MI is the main predictor of clinical outcome and mainly determined by the extent of collateral flow, myocardial metabolic demand, duration of coronary occlusion, and size of the initial AAR.[1,2] Among those factors, AAR represents the main constant predictor of the potential infarct size given that the anatomical distribution of the coronary arteries cannot be altered.[3,6]

Initial efforts to quantify AAR stem back to morphometric studies of the coronary arteries. The studies by Seiler et al.[10,11] established the basis of angiographically measuring the anatomic AAR. They stated that regional myocardial mass supplied by a coronary artery was closely and linearly related to the sum of coronary artery branch lengths (crown length) distally. Therefore, the sum of arterial branch lengths distal to the point of occlusion has been proposed for estimating the corresponding regional myocardial mass at risk. Similarly, they have shown a close relation between the cross-sectional lumen areas at any point in the coronary artery tree with the size of the dependent vascular bed. Likewise, Le et al.[12] studied morphometric relationships of the coronary artery tree using micro-computed tomography. The volumetric micro-CT dataset was used to quantify coronary arterial branch length and lumen volume and relating them to regional myocardial mass. They showed a power relationship of 0.75 between crown length and corresponding regional myocardial mass [log (L) α 0.75 log (M)]. A similar power relationship between crown length and summed coronary arterial volume (crown volume) was also established [log (L) α 0.75 log (V)]. Defining myocardial mass at risk from morphological surrogates such as crown length and crown volume requires detailed analysis and complex instrumentation, and thus it is of experimental value. Instead, a number of angiographic jeopardy scores have been developed to estimate the amount of myocardium at risk and predict patient-based clinical outcomes.[13-15] However, the clinical applicability of the current angiographic scoring systems is limited, especially in the setting of acute MI, due to their complex processes. Therefore, our simple RII can become a speedy, practical predictor of the relative contribution of an arterial segment to the perfusion of the heart.

Relation of RII with LV function

We assumed that the loss of the culprit segment's contribution to perfusion of the heart would have a decremental effect on LV systolic function in patients with MI. To test this hypothesis, we studied 123 subjects with their first ST-segment elevation acute anterior wall MI admitted for primary PCI. In order to eliminate the effect of collateral flow to the final infarct size, we excluded patients with angiographically visible collateral flow to the IRA. Likewise, we excluded subjects with pre-procedural TIMI flow ≥2 in the infarct-related artery. In this MI sub-population, we have shown a significant negative correlation between RII and EF (r=-0.65, p<0.001).

Relation of RII with infarct size

In order to test the hypothesis that the size of the artery is related to the mass it perfuses, we used 72-h TnI as a surrogate for flow-ceased myocardial mass. It was previously shown that a 72-h TnI provides a more reliable method to quantify myocardial damage than serial creatine kinase (CK) and early Tn values, as this reflects intramyocardial protein degradation, which is relatively unaffected by early reperfusion.[8] In this model, we have shown a modest but significant correlation between 72-h TnI and RII (r=0.48, p<0.001).

Relation of RII with MCE

In order to understand the effect of functional loss associated with higher RII to adverse clinical outcome, we evaluated one-month follow-up rates of MCE. We have shown a significantly higher composite MCE rate in patients with supra-median RII as compared to patients in the lower group. There was a tendency for higher mortality and non-fatal MI rates in the supra-median group, which did not reach statistical signifi-

cance, probably due to the small sample size and short follow-up duration.

Relation of RII with proximity of the culprit segment

We have shown a gradual increase in RII as the culprit lesion approaches more proximal segments of the LAD. It was previously shown that residual LVEF following MI was lower for proximal LAD lesions than that for distal ones.[16] Moreover, proximal culprit lesion location was shown to associate with increased risk of adverse outcome after adjustment of baseline factors.[17,18] We believe that, as a quantitative index, RII would offer more precise estimation of proximity of the arterial segment than simply categorizing the culprit lesion in boundaries of three segments.

Limitations

There are three major limitations of our study. The first is related to our patient enrollment criteria. In order to display the pure effect of RII -which is used as a surrogate of AAR- on LV function, we excluded patients with potential confounding factors such as collateral flow and early spontaneous reperfusion (pre-procedural TIMI flow ≥2). Likewise, we excluded patients with MI of any territory other than LAD, as in contrast to RCA and LCx, LAD is exclusively responsible for the perfusion of the LV. Thus, we ended up with a highly selected MI sub-population that precludes deriving a conclusive statement that can be extrapolated to the whole MI population. The other major limitation of the present study is the lack of a comparative imaging modality such as CMR or SPECT. Thus, it was not possible for us to compare RII with more accurate methods for assessment of AAR. This was due to technical and financial feasibility issues and the pilot nature of our study. Instead, we used 72-h TnI as an estimate of infarct size, which was shown previously to correlate with the final infarct size assessed by CMR.[8] The third limitation is related to the method and context of coronary artery size measurements. Measurements were done in the setting of MI, which is pathophysiologically linked with various vasoactive substances that can affect coronary artery size. Also, coronary artery measurements with angiography may not be appropriate in diffuse coronary artery disease as it depicts the lumen of the vessel. In addition, other determinants of coronary artery size must also be addressed, such as age, gender, body mass, height, and body surface area. However, we believe that use of a proportional index would inherently correct the coronary artery size for the aforementioned anthropometric measurements.

In conclusion, we defined a simple angiographic index that has predictive value on LV systolic function loss and adverse clinical outcome in patients with acute anterior MI. Given the fact that residual LV function in MI survivors is the best predictor of mortality, one can assume that a higher RII might predict mortality. Nevertheless, our study was underpowered to show this hypothetical relation, due to the limited number of patients enrolled and the short follow-up duration. We believe that this issue is worth further study in future trials.

Conflict-of-interest issues regarding the authorship or article: None declared

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Key words: Acute myocardial infarction; ejection fraction; relative importance index.

Anahtar sözcükler: Akut miyokart enfarktüsü; ejeksiyon fraksiyonu; kısmi önemlilik endeksi.