ORIGINAL ARTICLE

Relationship between the infarct localization and left ventricular rotation parameters following acute ST-segment elevation myocardial infarction

Akut ST yükselmeli miyokart enfarktüsü sonrası enfarkt yeri ile sol ventrikül rotasyon parametrelerinin ilişkisi

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ABSTRACT

Objective: This study was an investigation of the role of left ventricular (LV) apical rotation seen in the early period after myocardial infarction (MI) in predicting infarct localization.

Methods: A total of 124 patients with a ST-Segment elevation myocardial infarction (STEMI) diagnosis who underwent primary percutaneous coronary intervention (PCI) and 50 healthy volunteers with similar demographic characteristics were included in the study. The relationship between 2-dimenstional speckle tracking echocardiography (STE)-guided LV apical rotation angle measurements and technetium-99m sestamibi-single-photon emission computed tomography (SPECT)-guided infarct localization was evaluated. Conventional echocardiography and STE were performed on average 2 days after PCI, and gated SPECT myocardial perfusion imaging (MPI) was performed within an average of 60 days.

Results: The apical rotation angle was lower in patients with an anterior MI compared with those who had an inferior MI and the control group (AntMI-InfMI: 6.51±2.4°, AntMI-Control: 13.20±2.5°, InfMI-Control: 14.3±2.1°; p value: 0.00, 0.00, 0.15, respectively). SPECT MPI analysis revealed the presence of an LV apical scar in all patients with acute anterior MI, but only 14 of those with inferior MI group (usually the inferoapical wall). The apical rotation angle recorded in patients with apical scar was lower than that of the patients without apical scar (7.6±2.8° and 14.5±2°, respectively; p=0.00). Receiver operating characteristic curve analysis yielded an area under the curve for apical rotation of 0.799 (p<0.01). The optimal cutoff value of 12.1° had a sensitivity of 78.3% and a specificity of 68.2% for predicting LV apical scar following STEMI.

Conclusion: Detection of apical rotation angle decrease in the early period after STEMI may be useful in predicting extension of infarct scarring to the LV apex.

ÖZET

Amaç: ST yükselmeli miyokart enfarktüsü (STYME) sonrası erken dönemde ölçülmüş sol ventriküler (LV) apikal rotasyon değerinin gelişecek olan enfarkt lokalizasyonunu öngörmedeki rolünü araştırdık.

Yöntemler: Yüz yirmi dört hasta ve benzer demografik özelliklere sahip 50 kişilik sağlıklı gönüllü değerlendirildi. 2D-Speckle Tracking ekokardiyografi (STE) kılavuzluğundaki apikal rotasyon açıları ile tc-99m sestamibi-tek-foton emisyon bilgisayarlı tomografi (SPECT) kılavuzluğundaki infarkt lokalizasyonu arasındaki ilişki değerlendirildi. Başarılı primer perkütan koroner girişimden (PPCI) itibaren 2D-STE ortalama 2 gün, SPECT görüntüleme ise ortalama 59 gün sonra yapılmıştı.

Bulgular: Apikal rotasyon açısı anteriyor miyokart enfarktüsü geçirmiş hastalarda inferiyor miyokart enfarktüsü geçirmiş hastalara ve kontrol grubuna göre anlamlı olarak daha düşük saptandı (sırasıyla, 6.51±2.4°, 13.20±2.5°, 14.3±2.1°, p değerleri; AntMI-InfMI: 0.000, AntMI kontrol: 0.000, InfMI kontrol: 0.150). SPECT görüntülemede perfüzyon kusurları değerlendirildiğinde, anteriyor miyokart enfarktüslü tüm hastalarda LV apikal duvarda skar gözlendi, ancak inferiyor miyokart infarktüslü sadece 14 hastada apikal skar saptanmıştı (genellikle inferoapikal duvarda). Apikal rotasyon açısının, apikal skar saptanmış hastalarda apikal skar saptanmamış hastalara göre azaldığını saptadık (sırasıyla, 7.6±2.8° ve 14.5±2° p=0.00). ROC analizinde, apikal rotasyon açısı için eğri altındaki alan 0.799, p<0.01; en iyi kestirim değeri olan 12.1° %78.3 duyarlılık ve %68.2 özgüllük ile STYME sonrası LV apikal skar oluşumunu öngörebilmektedir.

Sonuç: STYME sonrası erken dönemde apikal rotasyon açısındaki azalmanın tespiti, infarkt skarının sol ventrikül apeksine uzanacağını öngörmede faydalı olabilir.

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Infarct size (IS) is a predictor of left ventricular (LV) remodeling, and it has been demonstrated in clinical trials that it can be a useful endpoint.[1] A smaller IS has been associated with improved survival.[2] Therefore, IS might be a useful clinical endpoint in upcoming trials and a prognostic factor for patients with ST-elevation myocardial infarction (STEMI).[3] The results of 1 study examining single-photon emission computed tomography (SPECT) quantification indicated that IS, but not myocardial ischemia, was independently associated with cardiac death and/or reinfarction in STEMI patients who underwent primary percutaneous coronary intervention (PCI) and were treated with optimized medical therapy during follow-up.[4]

Abbreviations:

2D	*Two-dimensional*
GLS	*Global longitudinal strain*
GTOR	*Global torsion angle*
IS	*Infarct size*
LV	*Left ventricle*
MI	*Myocardial infarction*
MIBI	*Methoxyisobutyl isonitril*
MPI	*Myocardial perfusion imaging*
PCI	*Percutaneous coronary intervention*
SPECT	*Single-photon emission computed tomography*
STE	*Speckle-tracking echocardiography*
STEMI	*ST-elevation myocardial infarction*
Tc-99m	*Technetium-99m*

Noninvasive measurement of LV twist or rotation can be performed using magnetic resonance imaging or speckle-tracking echocardiography (STE). Cardiac torsion, a result of the architecture of the heart, is conventionally defined as the difference in the rotation of apical and basal cross-sections. Noninvasive assessment of LV twist or rotation could be applied in several clinical fields. One is evaluation of LV systolic function.[5,6] Distinct changes in ventricular twist dynamics are now seen to accompany a wide variety of disease states. Hearts with anterolateral infarctions, for example, tend to generate less apical torsion during systole. There has been research investigating whether the mechanical restoration and augmentation of apical torsion can serve to improve function in failing hearts.[7]

The ability to predict apical infarct scarring will be important. The focus of this study was patients with STEMI. The role of cardiac rotation parameters and longitudinal strain in the early period were analyzed with regard to predicting IS and localization.

The location and size of infarct scars were defined using SPECT myocardial perfusion imaging (MPI). The influence of an infarct scar on the motion pattern of the LV was examined with 2-dimensional (2D) STE. The relationship between 2D STE-guided measurements of LV global torsion angle (GTOR), apical rotation, basal rotation angles, and technetium-99m (tc-99m) SPECT-guided IS/localization was evaluated.

METHODS

This was a retrospective analysis of patients from between 2013 and 2019 who experienced a first STEMI and underwent primary PCI followed by SPECT MPI within 2 to 3 months. In all, 168 patients with STEMI were investigated. Patients with ischemia (n=10), atrial fibrillation/flutter (n=5), complete bundle branch block (n=2), a New York Heart Association functional class III-IV (n=15), chronic alcoholism (n=1), or poor image quality (n=11) were excluded from the study. The patients were divided into 2 groups based on the electrocardiographic findings. A total of 55 patients with an anterior myocardial infarction (AntMI group) and 69 with an inferior myocardial infarction (InfMI group) were included. The inclusion criteria for the InfMI group were a right coronary artery lesion and no ischemia in the region of the left anterior descending artery or the circumflex artery. The AntMI group consisted of patients with a left anterior descending artery lesion and no ischemia in the right coronary artery or circumflex artery regions.

Patients with a recent history of STEMI who were then referred for gated SPECT MPI as a result of clinical indications were retrospectively enrolled in the study. Conventional echocardiography and STE were performed on average 2 days (range: 1-5 days) after the PCI procedure, and gated SPECT MPI was performed within an average of 2 months (range: 1–4 months). In addition to routine echocardiographic measurements, the peak global longitudinal strain (GLS), GTOR angle, apical rotation angle, and basal rotation angle were measured from recorded images.

Fifty healthy participants without any history of cardiovascular disease (Control group) were examined to define normal motion of the LV. The upper limit of the 95% confidence interval of the average GLS, GTOR angle, apical rotation angle, and basal rotation angle in this group was used to distinguish between normal and abnormal motion of the heart.

Echocardiographic images were obtained in the left lateral decubitus position using the EPIQ 7 system (Koninklijke Philips N.V., Amsterdam, Netherlands) and a 3.5-MHz transducer. Torsion measurements were

performed offline using dedicated software (EPIQ QLAB; Koninklijke Philips N.V., Amsterdam, Netherlands). Images of 3 consecutive cardiac cycles were recorded, measured, and averaged. The mean frame rate of the images was 70 frames/second (range: 50–90 frames/second). Basal and apical short-axis views were used for the measurement of rotation. The software then selected stable speckles within the myocardium and tracked these speckles frame by frame throughout the cardiac cycle. Counterclockwise rotation viewed from the LV apex was denoted with a positive value, and clockwise rotation with a negative value. The GTOR angle was defined as the difference between the basal rotation and apical rotation angles (twist°).

Longitudinal myocardial deformations were evaluated using standard 2D images (frame rate, 60–90 frames/second). GLS was calculated as the average of 16 segmental strain values from the apical 4-, 3-, and 2-chamber views. The time to maximal myocardial shortening, including post systolic shortening if present, was measured from the electrocardiographic onset Q/onset R wave in 16 LV segments. If speckle tracking could not be performed from any chamber view, the patient was excluded from the study. Hence, 3 apical projections were available for all of the study participants. Interobserver variabilities were measured using the analysis of 10 random patients conducted by 2 independent and blinded observers. Intraobserver variabilities were assessed using the analysis of 10 patients by the same observer at 2 different time points.

SPECT MPI

A 2-day stress-rest protocol was performed with all of the study patients. At the moment of peak stress during an exercise test, 500 MBq Tc-99m methoxyisobutyl isonitril (MIBI) was administered intravenously. On the second day, 500 MBq Tc-99m MIBI was administered intravenously to the patient for rest imaging. SPECT MPI was performed 45 minutes after the administration of Tc-99m MIBI for both stress and rest images, and recorded with a dual head SPECT gamma camera system (E.cam; Siemens Healthineers GmbH, Erlangen, Germany) equipped with a low-energy, high-resolution collimator. A 20% window was used around the 140 keV energy peak of 99mTc, and the data were stored in a 64x64 matrix. The SPECT stress and rest data sets were post-processed and reconstructed in vertical and horizontal long-axis and short-axis views perpendicular to the heart axis. The 17-segment model was used to define vascular territories. Quantitative analysis (IS) was performed using the Quantitative Perfusion SPECT application (Cedars-Sinai Medical Center, Los Angeles, CA, USA).

Statistical analysis

A total of 124 patients with STEMI (55 AntMI+69 InfMI) and 50 healthy controls (174 consecutive individuals) were included in the study. The PASW Statistics for Windows, Version 18.0 (SPSS Inc., Chicago, IL, USA) program was used to analyze the data. Distribution of continuous variables was evaluated using histograms and normal Q–Q plots. Continuous variables distributed normally were presented as mean±SD. Categorical variables were presented as number and percentage. The Kolmogorov-Smirnov test was used to assess the normality assumption. The Student's t-test was used for normally distributed data, and the Mann-Whitney U test (age, creatine kinase, left ventricular end-diastolic volume) for non-normally distributed data. All of the statistical analyses were performed with the IBM SPSS Statistics for Windows, Version 23.0 software package (IBM Corp., Armonk, NY, USA). A statistical test was considered significant if the p value was <0.05.

Table 1. Patient characteristics and risk factors

Characteristic	STEMI (n=124)	Control (n=50)	*p* value
Age (years), mean±SD	50±10	52±8	0.06
Male sex, n (%)	75 (60)	30 (60)	0.01
Current smoker, n (%)	99 (79)	39 (78)	0.02
Hypercholesterolemia, n (%)	53 (42)	22 (44)	0.05
Hypertension, n (%)	100 (80)	41 (82)	0.01
Diabetes mellitus, n (%)	25 (20)	10 (20)	0.01

Data are presented as mean±SD or number. Hypercholesteremia was defined as use of a statin, and hypertension was defined as use of an antihypertensive medication. STEMI: ST-elevation myocardial infarction; SD: Standard deviation.

Table 2. Peak values of biomarkers

	AntMI	InfMI	p value
Troponin I (ng/mL)	51	36.5	0.03
Brain natriuretic peptide (pg/mL)	123	92	0.00
Creatine kinase-MB (mg/dL)	251	142	0.01
Creatine kinase (mg/dL)	2498	1277	0.00

AntMI: Anterior myocardial infarction; InfMI: Inferior myocardial infarction.

RESULTS

A total of 55 patients with an anterior MI (87% male; mean age: 50.5±10.8 years) and 69 patients with an inferior MI (81% male; mean age 52.2±8.2 years) were included in the research. The baseline characteristics are shown in Table 1. Anterior wall infarction typically resulted in a greater area of necrotic myocardium and higher peak values of biomarkers than inferior wall infarction (Table 2).

The GLS was lower in the AntMI group compared with the InfMI group, and lower in the InfMI patients than in the control group (AntMI-InfMI:

Table 3. Infarct size, strain, rotation values, and basic echocardiographic parameters

	AntMI	InfMI	Control	*p* value
	Mean±SD	Mean±SD	Mean±SD	
GLS (-) (%)	11.1±2.5	14.6±1.2	18.5±1.1	AntMI-InfMI: 0.00 AntMI-Control: 0.00 InfMI-Control: 0.00
GTOR angle (°)	11.3±3.0	17.8±3.0	18.7±3.0	AntMI-InfMI: 0.00 AntMI-Control: 0.00 InfMI-Control: 0.32
Apical rotation angle (°)	6.5±2.4	13.2±2.5	14.3±2.1	AntMI-InfMI: 0.00 AntMI-Control: 0.00 InfMI-Control: 0.15
Basal rotation angle (°) (-)	5.3±1.2	4.9±1.8	5.0±1.3	AntMI-InfMI: 0.99 AntMI-Control: 0.91 InfMI-Control: 0.93
LVEDV (mL)	124.4±12.9	115.3±14.8	109.8±9.0	AntMI-InfMI: 0.01 AntMI-Control: 0.01 InfMI-Control: 0.10
LVESV (mL)	68.3±12.0	66.3±9.80	61.6±7.0	AntMI-InfMI; 0.61 AntMI-Control: 0.04 InfMI-Control: 0.04
EF (%)	42.3±7.8	50.6±8.60	59.2±4.2	AntMI-InfMI: 0.00 AntMI-Control: 0.00 InfMI-Control: 0.00
Infarct size (left ventricular mass %)	23.8±16.8	7.7±7.5		AntMI-InfMI: 0.00

AntMI: Anterior myocardial infarction; EF: Ejection fraction; GLS: Global longitudinal strain; GTOR: Global torsion; InfMI: Inferior myocardial infarction; LVEDV: Left ventricular end-diastolic volume; LVESV: Left ventricular end-systolic volume; SD: Standard deviation.

Table 4. Correlation table

	r	*p* value
Infarct size in anterior myocardial infarction group		
Global longitudinal strain	-0.66	0.00
Global torsion	-0.76	0.00
Apical rotation (°)	-0.81	0.00
Basal rotation (°) (–)	-0.24	0.24
Infarct size in inferior myocardial infarction group		
Global longitudinal strain	-0.72	0.00
Global torsion	-0.24	0.21
Apical rotation (°)	-0.20	0.30
Basal rotation (°) (–)	-0.10	0.60

Table 5. Strain and rotation values in the InfMI group according to apical scar presence

	With apical scar (n=14)	Without apical scar (n=55)	*p* value
	Mean±SD	Mean±SD	
GLS (-) (%)	13.9±0.5	14.9±0.2	0.08
GTOR angle (°)	12.6±0.6	18.7±0.3	0.00
Apical rotation angle (°)	7.6±2.8	14.5±2	0.00
Basal rotation angle (°) (-)	4.96±0.2	4.6±0.2	0.30

GLS: Global longitudinal strain; GTOR: Global torsion; InfMI: Inferior myocardial infarction; SD: Standard deviation.

-11.1±2.5%, AntMI-Control: -14.6±1.2%, - InfMI-Control: 18.5±1.1%; p value: 0.000 for all). The apical rotation angle was lower in the AntMI group compared with the InfMI group and the control group (AntMI-InfMI: 6.5±2.4°, AntMI-Control: 13.2±2.5°, InfMI-Control: 14.3±2.1°; p value: 0.00, 0.00, 0.15, respectively). The basal rotation angle was not significantly different between the AntMI, InfMI, and control groups (AntMI-InfMI: -5.3±1.2°, AntMI-Control: -4.9±1.8°, InfMI-Control: -5.0±1.2°; p value: 0.99, 0.91, 0.93, respectively). As expected, the GTOR angle, defined as the difference between the basal rotation and apical rotation angles, was lower in the AntMI group compared with the InfMI group and the control group (AntMI-InfMI: 11.3±3°, AntMI-Control: 17.8±3°, InfMI-Control: 18.7±3°; p value: 0.00, 0.00, 0.32, respectively) (Table 3). There was a negative correlation between IS and early GLS (p=0.00; r=-0.66), GTOR (p=0.00l; r=-0.76), and apical rotation (p=0.00; r=-0.81) in the AntMI group. There was also a negative correlation between IS and early GLS (p=0.00; r=-0.72) in the InfMI group (Table 4).

Evaluation of perfusion defects using SPECT MPI revealed an LV apical scar in all of the patients with an anterior MI, but only 14 of the inferior MI patients (usually inferoapical wall). An apical scar was noted in all of the patients with a decreased apical rotation angle (AntMI: n=55, InfMI: n=14). The apical rotation angle in the InfMI group with an apical scar was less than that of the patients without an apical scar (7.6±2.8° and 14.5±2°, respectively; p=0.00). A decrease in the apical rotation angle suggests that the infarct will be in the apex in the coming months (Table 5). Receiver operating characteristic curve analysis indicated that the area under the curve for apical rotation was 0.79 (p<0.01) and the best cutoff value of 12.1° had a sensitivity of 78.3% and a specificity of 68.2% for predicting an LV apical scar after STEMI (Fig. 1).

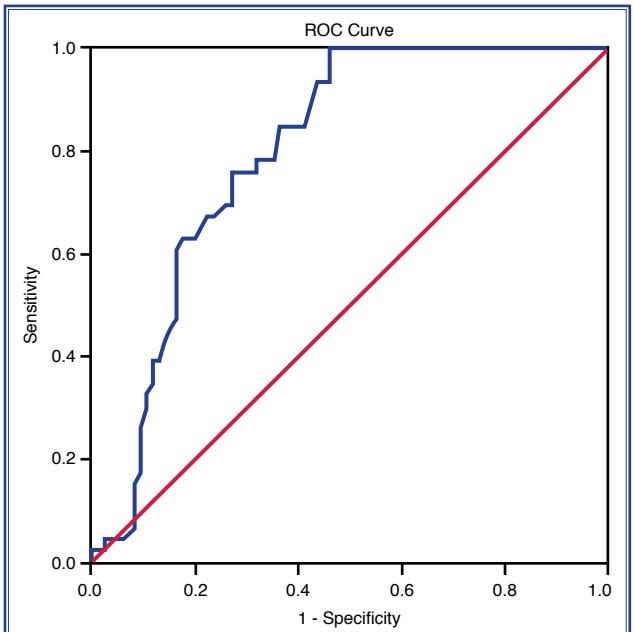

Figure 1. Receiver operating characteristic (ROC) curve testing the accuracy of left ventricular (LV) apical rotation in the prediction of apical scarring. The optimal LV apical rotation cutoff value of 12.1° provided the highest sensitivity (78.3%) and specificity (68.2%) for predicting an apical scar. The area under the curve for apical rotation was 0.799 (p=0.01).

DISCUSSION

We sought to contribute to the knowledge of the role of GLS and torsional parameters in the early post-MI period to predict IS and infarct location. The subendocardial region is more sensitive and is the first to be affected by the transmural dissemination of perfusion; therefore, the disseminating ischemia wave affects the contraction of longitudinal fibers in this region first.[5–8] Longitudinal fibers are typically oriented in the subendocardium and thus are more vulnerable to wall stress and fibrosis, in contrast to midwall circumferential fibers, which are not as greatly affected.[9] Long-axis function might be a potential indicator of subclinical LV dysfunction in numerous diseases.[10] The GLS value calculated in our study was significantly lower in STEMI patients (with a more prominent difference in the anterior MI group relative to the inferior MI group) compared with the control group. The correlation we observed between GLS values and IS could assist in early determination of IS. Our study results revealed that the GLS value after acute inferior MI did not predict apical scar development, but we also wanted to assess the apical longitudinal strain as a predictor. The apical strain values were significantly lower in the group with apical scarring (-19.5±0.4%) compared with the healthy group (-25.1±0.8%) (p=0.02), but there was no significant difference between the group with an apical scar and without apical scar (-22.2 ±0.8%) (p=0.40). The apical longitudinal strain value in the early MI period was a poor predictor of the infarct extending to the LV apex in the near future.

The results of this study demonstrate the value of apical rotation measurement, which reflects the effect of acute myocardial cell necrosis on the strain and twisting properties of the myocardium, in predicting LV apical scarring. Several studies have examined the incremental value of LV torsion to predict LV remodeling after MI.[8,11] Our research indicated that apical and basal rotation responded differently to STEMI. Apical rotation demonstrated clear changes, whereas basal rotation exhibited no significant alteration. The decrease in apical rotation angle may be an early marker of an apical scar. These results are valid for both anterior and inferior MIs.

We found no significant decrease or increase in the basal rotation angle after STEMI. Similarly, other studies of anterior MI patients have observed a decreased apical rotation in the infarcted LV, but a preserved LV basal rotation.[12,13] However, other research has suggested that basal rotation has an important role in LV function, finding that the apical rotation was significantly lower and basal rotation was significantly higher in acute anterior and inferior MIs.[14–16]

One of the limitations of our study is that echocardiographic examinations were not repeated at regular intervals, which would allow for detection of the change in basal rotation value in more detail. In addition, in the control (healthy) group, the absolute basal rotation angles were lower than the apical rotation angles, so the change in apical rotation angles may be more pronounced than basal rotation angles in the STEMI group.

Figure 2 shows the follow-up data from 2 patients with an inferior MI. In the first patient, the apical rotation angle was 3.8° and the basal rotation angle was -4.3°. After 2 months, SPECT MPI detected a fixed inferior perfusion defect (infarct) extending from the apical to the basal segments. In the second patient, the apical rotation angle was 16°. A fixed perfusion defect was observed in the inferior-inferolateral wall, not ex-

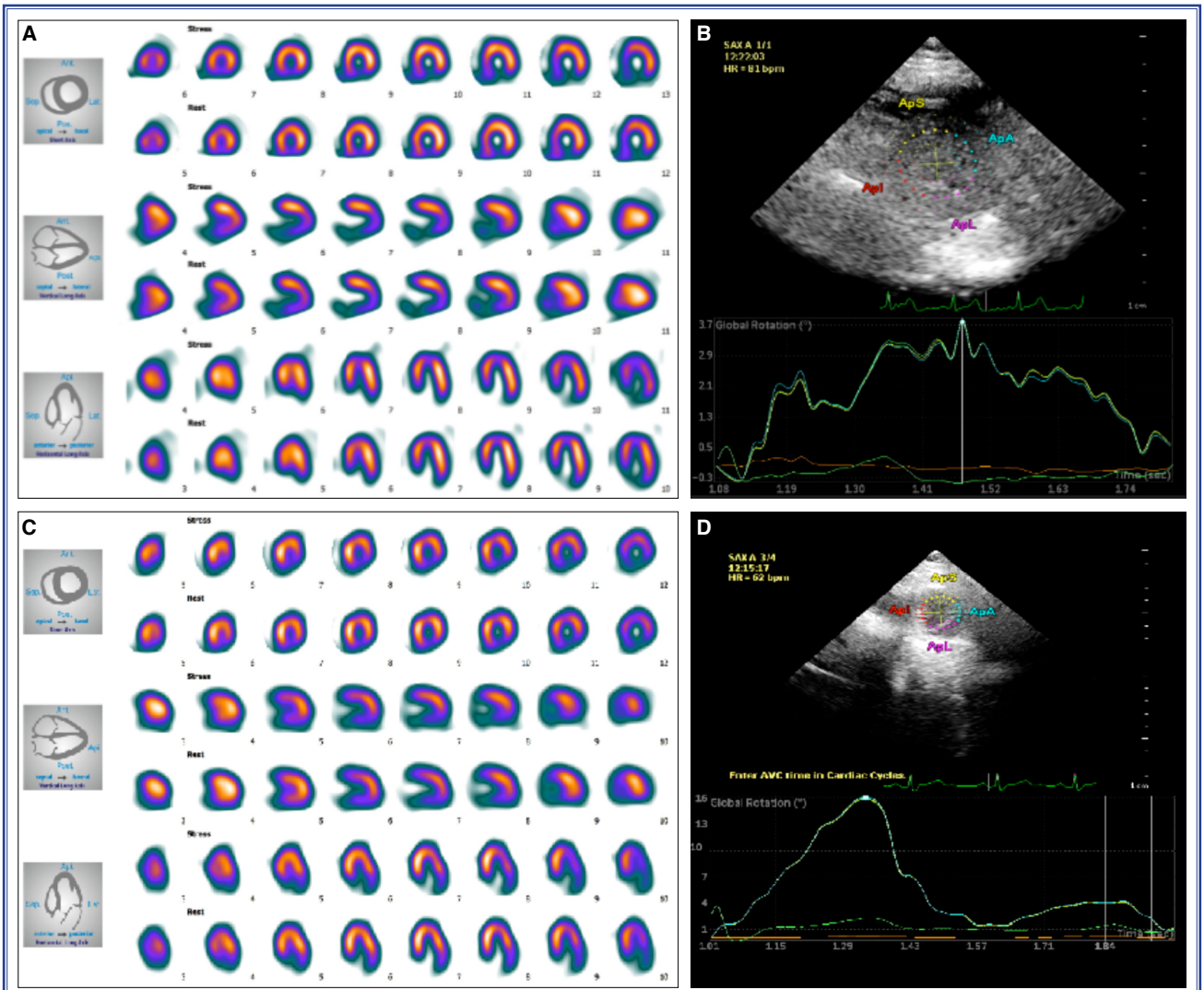

Figure 2. Reduced apical rotation due to apical perfusion defects illustrated with single-photon emission computed tomography (SPECT) data derived from 2 patients after a successful primary percutaneous coronary intervention for acute inferior myocardial infarction. **(A)** SPECT myocardial perfusion imaging shows a fixed inferior perfusion defect extending from the apical to the basal segment; **(B)** Decreased apical rotation angle (3.8°) observed in strain analysis; **(C)** In the second patient, a fixed perfusion defect was seen in the inferior-inferolateral wall, not extending to the apex; **(D)** Strain analysis of the apical rotation angle was 16° (within normal limits). Decreased global longitudinal strain was recorded in both patients.

tending to the LV apex. Both patients had a decreased GLS. The patients in our study were divided into 2 groups according to the presence of an LV apical infarct. We found apical scarring in all of the patients with an anterior MI (55) and in 14 of the patients with an inferior MI. There were significant differences in the apical rotation angle between the 2 groups. The detection of decreased apical rotation in the early MI period may indicate that the infarct will soon extend to the apex.

Previous studies have found that the impact of MI on LV rotational mechanics appeared to be related to scar extension (IS) rather than location.[1–5] In these studies, global LV twist was more impaired in patients with a large STEMI than in those with a small STEMI.[11–16] Our study findings support these results: Patients with an apical scar had a larger IS than those without an apical scar.

A second 2D-STE on day of SPECT MPI could be useful, but the aim of this study was to assess 2D-STE data in the early MI period that could predict LV apical scarring. Accurate tracking is dependent on image quality and there is interobserver variability; for example, the selection of LV apical and basal planes,

through-plane motion, and the transmural depth of the region of interest. These factors reduced the number of participants in our study.

In conclusion, GLS during the early post-MI period may be used to determine IS, but it may not help determine the location of the infarct. A decrease in the apical rotation angle may be useful in predicting that the infarct scar will extend to the LV apex. Early evaluation of LV apical rotation with STE after STEMI may be able to identify patients with reduced apical rotation who could benefit from aggressive medical therapy and revascularization in order to avoid LV apical infarct scarring. Prospective studies with more patients are needed to better understand the relationship between cardiac torsion and scarring.

Ethics Committee Approval: The study was approved by local clinical research ethics committee (date: 06.12.2018, approval number: 93966460-605.01).

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Keywords: Apical rotation; infarct location; infarct size; left ventricular torsion.

Anahtar sözcükler: Apikal rotasyon; enfarkt yerleşimi; enfarkt genişliği; sol ventriküler torsiyon.