Is Postsystolic Shortening a Marker of Viability in Chronic Left Ventricular Ischemic Dysfunction? Comparison with Late Enhancement Contrast Magnetic Resonance Imaging

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Background: During acute myocardial ischemia, myocardial postsystolic shortening (PSS) is considered as a sign of viability. In chronic left ventricular (LV) ischemic dysfunction, the value of PSS is less well established. In this study, PSS was compared with transmural extent of necrosis and contractile reserve in patients with chronic LV ischemic dysfunction.

Methods: A total of 25 patients (20 men, mean age: 63 ± 8 years) with LV dysfunction (mean ejection fraction: 32 ± 10%, range: 14%-47%) and stable coronary artery disease underwent rest color Doppler myocardial imaging, low-dose dobutamine echocardiography, and late enhancement gadolinium-magnetic resonance imaging. Strain (ε) curves were computed in 16 segments from color Doppler myocardial imaging sequences and were compared with transmural extent of necrosis and with contractile reserve. End-systolic ε was defined as ε value at aortic valve closure, peak ε (ε-peak) as maximal ε value during cardiac cycle, and time to ε-peak as time interval between aortic valve closure and ε-peak. PSS was considered when ε-peak occurred after aortic valve closure.

Results: Of 348 analyzable segments, 212 (61%) were graded as abnormal. In dysfunctional segments, PSS was more prevalent in transmural than in nontransmural infarcted segments (96% vs 50%, $P < .001$) and time to ε-peak was correlated to transmural extent of necrosis ($r = 0.69$, $P < .0001$). In nontransmurally infarcted segments, prevalence of PSS was similar in segments with or without contractile reserve (37% vs 45%, respectively).

Conclusion: In chronic LV dysfunction, PSS is not a specific marker of viability. These results suggest strongly that delayed myocardial shortening may be associated to scarred segments.

Risk stratification in patients with left ventricular (LV) ischemic dysfunction typically includes a combination of clinical, hemodynamic, and angiographic parameters.[1] During the past 10 years, several different studies have indicated that assessment of myocardial viability, ie, the ability of dysfunctional myocardium to improve in contraction after revascularization, also provides useful prognostic information in these patients,[2-6] with an effect additive to that of the usual clinical assessment.[2] Although a variety of different imaging modalities have been used to assess myocardial viability, these approaches are often limited by their availability, their cost, their technical difficulty, their subjective character, or a combination of these factors.

It has been recently proposed that postejection or postsystolic shortening (PSS) may serve as a marker of actively contracting and, therefore, viable myocardium.[7] PSS is defined as a myocardial deformation that occurs after aortic valve closure (AVC). It is typically seen in the early phases of acute myocardial ischemia, where its spatial distribution is consistent with that of the myocardium at risk. Several experimental and clinical studies have recently suggested that PSS could be a marker of residual myocardial viability.[7-10] Yet, results of several other studies suggest that it is a purely passive phenomenon that results from the interaction of ischemic with surrounding nonischemic segments.[11]

In view of these conflicting results, we designed the current study to evaluate the spatial and temporal relationships among PSS, derived from color Doppler myocardial imaging (CDMI) strain (ε) curves[12,13]; the transmural extent of necrosis, as delineated by late-enhancement gadolinium cardiac magnetic resonance (MR)[14-16]; and contractile reserve, as evaluated by low-dose dobutamine echocardiography (DbE).[17]

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METHODS

Study Population

The population consisted of 25 patients (20 men, mean age: 63 ± 8 years) with angiographic coronary artery disease and both regional and global LV dysfunction (mean ejection fraction: 32 ± 10%, range: 14%-47%). A total of 21 patients had experienced a previous myocardial infarction (16 anterior, 1 inferior, and 4 lateral). Five patients were diabetic, 11 had a history of hypertension, and two had a history of coronary artery bypass graft surgery. All patients gave written, informed consent to participate in the study, which had been approved by the ethical committee of our institution.

All patients underwent rest CDMI, contrast-enhanced (CE) low-dose DbE, and CE MR during the same hospital stay.

Cardiac Catheterization

Selective coronary arteriography and contrast left ventriculography were performed from the femoral approach, before the echocardiographic and CE MR studies. The presence and severity of coronary artery disease was assessed visually. Significant coronary disease was defined as a greater than 70% luminal diameter stenosis in any major coronary branch. Six patients had single-vessel disease, 6 others had 2-vessel disease, and 13 had 3-vessel disease.

CDMI

All CDMI studies were performed with the patients in a supine position. Data were acquired using a Sonos 7500 system, Philips Medical Systems, Andover, Mass) equipped with the broadband phased-array S3 transducer (1-3 MHz). CDMI myocardial velocity data were acquired at a frame rate of 80 ± 19 frames/s, using an imaging sector angle of 45 degrees. CDMI data from 3 consecutive cardiac cycles were acquired at rest in the apical 4-, 3-, and 2-chamber views and transferred onto a remote microcomputer for subsequent offline analysis.

CE DbE

For DbE, the patients were allowed to take their prescribed medications with an exception for β-blockers, which were withdrawn for at least 48 hours before the investigation. Before the test was started, a clinical history was recorded, a rest electrocardiogram (ECG) and echocardiogram were obtained, and a venous line was secured. To optimize LV contour delineation, a continuous intravenous infusion of PESDA (Perfluorocarbon-enhanced Sonicated Dextrose Albumin), a second-generation ultrasonic contrast agent, was then started. The rate of infusion was progressively adjusted to obtain homogenous cavity opacification during real-time scanning. Once an adequate LV opacification was obtained, dobutamine was infused in 3-minute dose increments from 5 to 40 μg/kg/min, under continuous ECG and echocardiographic monitoring. The test was concluded after achievement of the peak dose or earlier if the patient developed severe ischemia (either angina or impairment of LV function) or experienced intolerable side effects, as previously described.[17] Clinical signs, the ECG, and echocardiographic images were recorded at the beginning of the study and every 3 minutes thereafter until completion of the stress.

CE MR

MR imaging was performed in a 1.5-T magnet (Integra CV, Philips, Eindhoven, The Netherlands) using a phased-array coil wrapped around the chest. After localization of the heart, patients received an intravenous bolus of 0.2 mmol/kg of gadodiamide. Fifteen minutes later, delayed images were acquired in the apical 4-, 3-, and 2-chamber orientations using an inversion-recovery prepared gated fast gradient echo pulse sequence. Imaging was performed with the following parameters: repetition time, 4.6 milliseconds; echo time, 2.3 milliseconds; image matrix, 256 × 192; flip angle, 20 degrees; and inversion pulse of 180 degrees with an inversion time of 250 to 300 milliseconds. All image prescriptions had the same orientation as the echocardiographic images.

Data Analysis

The CDMI, CE DbE, and late-enhancement MR imaging data were analyzed using the American Society of Echocardiography 16-segment model.

CDMI

Longitudinal ε was computed from CDMI data using a prototype version of the Qlab software package (Philips Medical Systems). The software automatically tracks wall displacement and maintains the region in which measurements are made in midwall position. For ε computation, end diastole was chosen as the reference time point. This was defined to occur at the R-top on the ECG trace. The timing of end systole (AVC) was derived from M-mode echocardiographic recordings. End-systolic ε (ε-ES) was defined as the magnitude of deformation at the time of AVC. When further deformation occurred after AVC, this was measured as the peak ε (ε-peak). The difference between ε-ES and ε-peak was calculated as the PSS and, from this, the postsystolic index, expressed as a percentage, was derived from the equation; ([PSS/ε-peak]*100). The time delay from AVC to ε-peak was also calculated (Figure 1).

CE DbE

Images were interpreted qualitatively in accordance with previous guidelines by experienced observers who had no knowledge of the angiographic and clinical data. Regional function was defined as normal (1), hypokinetic (2), or akinetic (3).[17] Normal wall motion was defined as greater than or equal to 5 mm of endocardial excursion and obvious systolic wall thickening. Hypokinesis was defined as less than 5 mm of endocardial excursion and reduced wall thickening. Akinesis was defined as near absence of endocardial excursion or thickening.

A normal segmental response to dobutamine was defined as a progressive enhancement in contractility during stress. Ischemia was identified by a stress-induced wall-motion abnormality. Dysfunctional myocardium at baseline was considered as exhibiting contractile reserve if wall-motion score improved by at least one full grade with low-dose (5-10 μg/kg/min) dobutamine.

CE MR

Assessment of the transmural extent of delayed hyperenhancement was performed on the late (15-minute) gadolinium-DTPA–enhanced MR images as previously described.[16] Briefly, the images were binarized to a threshold of 2 SD above the mean value obtained in remote normal segments without hyperenhancement. The transmural extent of hyperenhancement was computed within each segment on these thresholded images and reported as the percentage of the segmental area that was hyperenhanced. Transmural necrosis was defined as greater than 50% hyperenhancement and nontransmural necrosis as less than or equal to 50% hyperenhancement.

Statistical Analysis

Continuous values were expressed as mean ± SD. Dichotomous data were presented as percentages. Linear regression was used to