
EXPERIMENTAL METHODS FOR CLINICAL PRACTICE

Prediction of Postinfarction Remodeling of the Left Ventricle from Parameters of Tissue Doppler Echocardiography

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The possibility of predicting undesirable postinfarction remodeling of the left ventricle from parameters of tissue doppler echocardiography was evaluated in 55 patients with a history of acute myocardial infarction. Low diastolic rates of normal segments and the absence of the peak of isovolumetric contraction in the dysfunction zone were the most significant predictors of unfavorable remodeling.

Key Words: *postinfarction remodeling; tissue doppler study*

Postinfarction remodeling is a complex of changes in the left-ventricular (LV) size, geometry, and function after myocardial infarction [3]. In some patients, remodeling eventuates in long-term stabilization of LV sizes and function, which is paralleled by a sufficiently favorable cardiac prognosis. However, in some cases the course of remodeling is unfavorable and leads to excessive LV enlargement and reduction of its contractility, which is associated with high risk of cardiac death, repeated myocardial infarction, and development of cardiac insufficiency [2]. One of the most important criteria of unfavorable remodeling is excessive increase in the end-diastolic index of LV [5].

The possibility of predicting the type of postinfarction remodeling of LV can be significant for choosing treatment strategy during the postinfarction period. However, there are no universally acknowledged methods for such prediction [8].

We attempted to determine criteria for predicting unfavorable postinfarction remodeling of LV

by the results of tissue Doppler echocardiography (TDE) carried out during the acute period of infarction. Using this method we can evaluate local contractility and relaxation of LV [1], which provides unique information about the coronary patient. It should be noted that the potentialities of TDE as a method for predicting postinfarction remodeling were virtually never studied.

MATERIALS AND METHODS

The study was carried out in patients with a history of acute myocardial infarction with *ST* elevation and systemic thrombolysis. The exclusion criteria were complete blockade of the His bundle left pedicle on ECG; class IV according to Killip's classification on admission; unsatisfactory echographic image. Dead patients, patients with a history of coronary revascularization, and patients lost for contact during 12 months of observation period were excluded from the study.

Echocardiography (EchoCG) was carried out on a Vivid-3 Expert device (GE HC). The first EchoCG was carried out before discharge (days 8.8 ± 0.2 after admission), control EchoCG 12 months after in-

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farction (EchoCG-1 and EchoCG-12, respectively). Standard measurements in the unidimensional mode were carried out. The end-diastolic and end-systolic volumes of LV were evaluated in the B mode by Simpson's method [9] and by dividing these values by the body surface area value, the end-diastolic and end-systolic indexes were calculated. Unfavorable remodeling was estimated as LV end-systolic index >35 ml/m² in EchoCG-12 [6].

The LV dysfunction zone was located in accordance with the common 16-segment model [7], regional contractility was evaluated visually semi-quantitatively by a 4-point system (1: normal status; 2: hypokinesia; 3: akinesia; and 4: dyskinesia).

Pulsed TDE was carried out during EchoCG-1. Longitudinal rates of LV myocardial movement were evaluated. Because of technological peculiarities of the method [1], analysis was carried out only for 12 basal and median LV segments from three orthogonal apical sections: four-compartment, two-compartment, and longitudinal. The control volume was placed in the center of the studied segment. The range of measured rate of the segment was ± 22 cm/sec. The resultant rate curves were saved in the digital format, after which measurements were carried out on saved images. The maximum systolic (Sm) and diastolic (Em) rates of the segment were measured. The next step was calculation of summary Sm and Em rates for normally contracting segments and segments in the contractile dysfunction zone and the indexes of these summary rates (their proportion in total number of segments with disordered contractility). Segments with disordered contractility with a positive peak of isovolumetric contractions (IVC) were counted and the isovolumetric contraction index was calculated similarly.

This calculation of rate indexes in TDE is not standard; we propose this approach for comparing patients with different numbers of non-functioning segments.

RESULTS

A total of 68 patients were included in the study; EchoCG-12 was carried out in 55, 28 of these formed the unfavorable remodeling group (R₁) and 27 the favorable remodeling group (R₀).

Patient age and the incidence of disorders in the contractility of basal and median segments differed negligibly between the groups. The number of patients with involvement of the lower segments was significantly lower in R₁ group: 0 vs. 6 (22%; $p=0.010$), while the number of patients with combined disorders in the lower and posterolateral segments was higher: 13 (46%) vs. 1 (4%); $p<0.01$.

The following parameters were lower in the group with unfavorable dynamics of the end-systolic index in comparison with R₀ group: Sm summary rate index in the dysfunction zone (4.48 ± 0.28 vs. 5.37 ± 0.31 cm/sec; $p=0.036$); Em summary rates index in the dysfunction zone (6.01 ± 0.34 vs. 7.33 ± 0.39 cm/sec; $p=0.014$); Em summary rate index for normal segments (13.94 ± 1.89 vs. 26.79 ± 3.08 cm/sec; $p<0.001$); and the IVC index (0.25 ± 0.06 vs. 0.45 ± 0.07 cm/sec; $p=0.044$). Independent effects of IVC and index of summary rates of normal segments Em on the dynamics of end-systolic index of LV was demonstrated. Discriminative points were found for them and their diagnostic value was estimated. The sensitivity of index of summary rates Em for normal segments <16.00 cm/sec for prediction of unfavorable time course of the end systolic index was 75%, specificity 78%. For the IVC index <0.31 both values were 68%.

Since remodeling depends on changes in not only infarction zone, but also normal segments, we evaluated the impact of basal rates of normal myocardium movements for remodeling. This analysis, undertaken for the first time (as far as we know), showed that the end-systolic index of rate for normal segments was more significant for the prognosis than that for the dysfunction zone.

The course of remodeling was more often unfavorable in cases without positive IVC peak (marker of viable myocardium) in the dysfunction zone [4].

Involvement of the posterolateral segments, normally characterized by high rates, and of lower ones (low-rate) was more incident in the R₁ group, which presumably leveled the impact of the site of damage for the measured rates. However, additional studies of the prognostic role of the studied TDE parameters should be carried out in groups not differing by the location of the dysfunction zone.

REFERENCES

1. M. N. Alekhin, *Ultrazvuk. Funkts. Diagnost.*, No. 3, 115-125 (2002).
2. Yu. N. Belenkov, *Rus. Med. Zh.*, No. 17, 685-694 (2000).
3. Yu. V. Belov and V. A. Varaksin, *Kardiologiya*, No. 1, 19-23 (2003).
4. C. Coletta, A. Sestili, F. Seccareccia, *et al.*, *Heart*, **89**, No. 10, 1138-1143 (2003).
5. A. W. Hamer, M. Takayama, K. A. Abraham, *et al.*, *Circulation*, **90**, No. 6, 2899-2904 (1994).
6. K. E. Hammermeister, T. A. DeRouen, and H. T. Dodge, *Ibid.*, **59**, No. 3, 421-430 (1979).
7. S. C. Smart, T. Knickelbine, T. R. Stoiber, *et al.*, *Ibid.*, **95**, No. 6, 1394-1401 (1997).
8. M. St. John Sutton and C. H. Scott, *Eur. Heart J.*, **23**, No. 7, 509-511 (2002).
9. D. W. Wahr, Y. S. Wang, and N. B. Schiller, *J. Am. Col. Cardiol.*, **1**, No. 3, 863-868 (1983).