Predictor of High-Risk Patients after Acute Myocardial Infarction by Serial Echocardiography

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ZHANG ET AL.: ***Predictor of High-Risk Patients after Acute Myocardial Infarction by Serial Echocardiography.*** *In order to distinguish between the low and high-risk patients for ventricular remodeling after acute myocardial infarction (AMI) and to determine whether an early predictor of progressive ventricular dilatation and chronic dysfunction could be identified, 70 consecutive patients undertook serial echocardiography on the entry, 3 days, 2 weeks, 3 and 6 months after admission. The results showed that variables influencing the pattern of remodeling are the location of AMI, initial endocardial surface area index and abnormal wall motion area and reperfusion of infarcted-related artery. Increased endocardial surface area index within 48 hours of onset of AMI can be considered as an echocardiographic manifestation of infarct expansion, and was proposed an early predictor for high risk patients after AMI. (J HK Coll Cardiol 2002;10:100-104)*

Echocardiography, Myocardial infarction, Ventricular remodeling

摘要

探討急性心肌梗塞後心室重構的高低危患者和決定梗塞區膨展是高危患者的早期預測指標。7 0 例連續患者均在入院後 24 小時內、72 小時、2 周、3 個月和 6 個月分別進行超聲心動圖檢查。結果顯示：梗塞部位、入院時心臟大小和梗塞區面積及梗塞相關血管再通是決定心室重構類型的因素。入院後 48 小時內心臟進行性增大是梗塞區膨展的臨床特徵，也是高危患者的早期預測指標。

關鍵詞：超聲心動圖　心肌梗塞　心室重構

Introduction

Two dimensional echocardiography has become established as an ideal noninvasive method to identify and quantitate regional left ventricular morphology that accompanies acute myocardial infarction (AMI).[1,2] A number of studies have emphasized the acute structure and functional changes were dynamic and varied with the size and location of the infarction. In addition, dilatation of the left ventricle might play an important and active role in the development of chronic heart failure, and left ventricular volume was the most powerful predictor of survival in patients with coronary artery disease.[3] Deterioration of cardiac performance correlated with the degree of dilatation in patients with AMI. However, the time course and interaction of regional function of noninfarcted and infarcted myocardium and global left ventricular dysfunction had

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not been evaluated in detail. The purpose of this study was to use computerized echocardiography to prospectively determine the natural history of left ventricular size, area of abnormal wall motion (AWM), i.e. area of functional infarct size, and ventricular function, to distinguish between the low and high risk patients for ventricular remodeling from entry until 6 months after AMI, and to determine whether an early predictor of progressive ventricular dilatation and chronic dysfunction could be identified.

Materials and Methods

Study Group

Patients with a first Q wave AMI were prospectively screened for inclusion into the study. Q wave AMI was defined by the presence of chest pain lasting at least 30 minutes, new ST segment elevation in ECG, appearance of new Q waves on serial electrocardiograms, and a significant rise in creatine kinase (CK) and cardiac isoenzyme fraction (CK-MB). Patients with previous myocardial infarction, primary valvular heart disease, cardiomyopathy, the onset of symptoms more than 48 hours before entry were excluded from the study. In addition, patients with echocardiogram of inadequate quality for quantitative analysis were also excluded. Seventy of the 73 patients completed the study, including 3 patients not being followed up (follow-up rate 95.4%), Eight patients (7 anterior and 1 inferior; mean age 64±6.2 years) died during follow-up (11%). Six months follow-up were completed in 62 patients, 42 of that had anterior infarction, 20 had inferior infarction (male 50 and female 12, mean age 59±11.6 years). Patients were in hospital within 12 hours after disease onset and treated with thrombolytic therapy. Sixteen (11 anterior and 5 inferior, mean age 59.8±10.1 years) of 62 patients had patency of infarct-related coronary artery, which was assessed by uniform clinical criteria (receiving thrombolytic therapy) or by coronary angiography (receiving coronary angioplasty or/and coronary stents). Thirty normal volunteers served as normal controls. This group consisted of 22 men and 8 women (mean age 60.9±15.3 years). Twenty-four of the 70 patients had cardiac events such as cardiogenic death, persistent ventricular tachycardia, ventricular fibrillation, symptomatic heart failure and post-infarction angina pectoris.

Data Acquisition of Echocardiography

Two dimensional echocardiography was performed prospectively with a 2.5 mHZ transducer using a phased array system (HP1000 or HP2500) by one sonographer at entry, 3 days, two weeks, 3 and 6 months after first AMI. Echocardiographic images of the left ventricle were obtained from five standard imaging planes-parasternal short axis views at mitral valve, papillary muscles and apical level as well as apical four chamber and two chamber views.

Measurement of Endocardial Surface Area Index of Left Ventricle and AWM

Suitable images from the five recorded planes (three short axis views, two and four chamber views) were used to derive the cardiac dimensions including long axis and endocardial length of left ventricle, and the endocardial length of abnormal wall motion segment. The endocardial surface area of left ventricle and AWM area were calculated with a computerized data process system reported and validated previously.[4,5] Because body surface area has been identified as a primary determinate of normal left ventricular dimensions, the endocardial surface area of left ventricle was corrected for the body surface area. This corrected endocardial surface area, i.e., ESAi, and AWM area were used in analyses for between-patients comparisons.

Standards of Ventricular Size

Thirty normal volunteers without coronary artery disease were used as controls. This group, mean age 60.9±15.3 years, consisted of 22 man and 8 women. The mapping technique was then applied to generate a normal range for endocardial surface area. The range of normal ESAi was 63.7±5.1 cm^2/m^2. An ESAi of more than 2 standard deviation above the control group were defined as ventricular enlargement.

Standards of Infarct Expansion

Infarct expansion was defined as left ventricular enlargement, wide-mouthed, thinned-walled myocardial segments that display dyskinetic expansion during systole.

Statistic Analysis

Data were expressed as mean±SD. Statistical comparisons between groups were made by analysis of

variance (ANOVA). Comparisons of frequency of events were performed by x^2 analysis.

Results

Serial Echocardiographic Quantitative Analysis

After AMI, the changes of left ventricle and the infarct area had different trends during 6 months. The effects on left ventricular ejection fraction (EF) value (measured by Simpson's equation) were also different. Based on the data shown in Table 1, patients were divided into four subgroups: (1) 20 had a normal ESAi on admission, no changes during 3 months after AMI, a decrease in left ventricular size at 6 months ($P<0.05$). Decrease in infarct area was noted from entry to 6 months after AMI ($P<0.01$), without EF reduced; (2) 27 had progressive left ventricular dilatation from entry to 3 months after AMI ($P<0.05$), decreased in left ventricular size and infarct area from 3 to 6 months ($P<0.01$), and reduction in EF value ($P<0.01$); (3) 15 had limited dilatation with subsequent reduction. Whether left ventricular dilatation or not at entry, no change in ESAi at two weeks, but decrease in the left ventricular size noted from 2 weeks to 6 months after AMI, and progressive decrease in infarct area from entry to 6 months, without significant reduction in EF value; (4) 7 had an enlarged ESAi at entry, accelerated left ventricular dilatation within 72 hours after AMI. All the patients had infarct area extended greatly with typical manifestation of infarct expansion, and large reduction in EF value. Those 8 patients died in hospital except one were all in this pattern.

Influencing Factors of Ventricular Remodeling after AMI

Based on the size of infarct area, ESAi within 48 hours after AMI and early reperfusion, various remodeling process could be analyzed as follows: In the patients with no dilatation of left ventricle, AWM area was the smallest. Most of them had inferior AMI. Left ventricular size was unchanged except in two patients (ESAi=74 cm^2/m^2). Six of the 20 patients had early reperfusion.

In the group with progressive dilatation of left ventricle, most patients had anterior AMI, 12 of whom

Table 1. Natural history of ESAi, area of AWM, and EF value

Time	Group 1 n=20	Group 2 n=27	Group 3 n=15	Group 4 n=7
ESAi				
24 hours	66.5±5.5	73.8±8.2	72.5±11.2	79.4±9.2
72 hours	67.7±6.3	76.6±9.2	74.3±11.0	91.1±7.7*
2 weeks	69.5±8.6	77.3±8.4	71.1±8.7	
3 months	65.5±9.0	79.6±9.4*	65.2±7.9*	
6 months	62.1±7.5*	69.9±7.9##	61.8±6.7**	
AWM area				
24 hours	24.7±3.2	55.3±12.6	55.1±9.0	66.4±11.3
72 hours	24.6±3.1	56.0±13.7	54.1±9.6	79.7±10.3*
2 weeks	22.6±4.2	56.8±14.4	46.0±7.0**	
3 months	20.4±4.9**	61.0±15.5	42.9±7.0	
6 months	17.0±4.8#	43.2±9.9 **##	38.5±7.1**	
EF				
24 hours	0.47±0.05	0.45±0.07	0.47±0.05	0.42±0.01
72 hours	/	/	/	0.34±0.08*
2 weeks	0.48±0.06	0.44±0.08	0.47±0.08	
3 months	0.47±0.07	0.44±0.05	0.48±0.06	
6 months	0.52±0.06	0.39±0.07**##	0.51±0.06	

Group 1: LV no changes; group 2: progressive dilatation; group 3: progressive decreased; group 4: accelerated ventricular dilatation

*compared with at entry $P<0.05$, **compared with at entry $P<0.01$; #compared with at 3 months $P<0.05$, ##compared with at 3 months $P<0.01$

had initial left ventricular dilatation at entry, with AWM area larger than those without initial dilatation (P<0.001).

In patients with progressive decrease in size of left ventricles in the presence of anterior AMI, 7 patients had initial dilatation, with AWM area larger than those without initial dilatation (P<0.001). All patients had early reperfusion.

All seven patients with accelerated left ventricular dilatation within 72 hours after AMI had initial dilatation and anterior AMI. None had early reperfusion, but they had the most significant left ventricular dilatation and the largest AWM area. The initial left ventricular size and AWM area between progressive dilatation group and progressive decrease group had no significant difference.

Influence on Cardiac Function and Prognosis by Ventricular Remodeling

Patients with accelerated ventricular dilatation in 72 hours after AMI had the lowest initial EF value at entry (P<0.05), which continued to further decrease within 72 hours of onset of AMI. Seven of those patients were dead in hospital. Six months after AMI, EF value in progressive dilatation group was the lowest and differed significantly from the other two groups (P<0.05). EF value was much lower in patients with initial dilatation than without initial dilatation (P<0.01).

Twenty-four of the 70 patients had cardiac events, including 7 patients with accelerated ventricular dilatation within 72 hours after AMI, 14 patients with progressive ventricular dilatation , 2 patients with no ventricular dilatation, and 1 patient with progressive ventricular size reduction. There were 19 patients with anterior AMI with initial dilatation but without early repefusion at entry (12 from ventricular progressive dilatation group, 7 from accelerated ventricular dilatation group), 15 of which had cardiac events (7 died in hospital, 3 had persistent ventricular tachycardia, 6 had symptomatic heart failure, 1 had post-infarction angina pectoris).

Discussion

Significance of Discrimination of Left Ventricular Remodeling by Echocardiography

Our study demonstrated that there were various manifestations of ventricular remodeling, such as no significant changes in ventricular size, progressive dilatation, limited dilatation with subsequent reduction and accelerated ventricular dilatation in 72 hours, which had different effects on left ventricular function and prognosis. The infarct size was critical to ventricular remodeling. Patients with larger anterior infarct area tended to have the higher risk of infarct expansion and progressive dilatation, while those who had smaller infarct areas of inferior infarction had the lower tendency. Measurement of ESAi and AWMA area by echocardiography could serially assess the process of ventricular remodeling accurately, and distinguish between the low and high risk patients for ventricular remodeling for intervention. Variables influencing the pattern of remodeling were the location of AMI, initial ESAi and AWMA size and reperfusion of infarct-related artery.

Assessment of Infarct Expansion by Echocardiography

Infarct expansion may be responsible for left ventricular enlargement in the early stages after AMI, and contribute to the ventricular remodeling in all stages. This occurred in patients with large infarct area. Ventricular remodeling after AMI is an important process affecting ventricular function and survival. Therefore, infarct expansion was the most powerful predictor of high risk patients with ventricular remodeling after AMI. Gaudron et al demonstrated that infarct expansion was present in 50% of patients with anterior infarctions,[6] but only 7 of 44 anterior infarctions had infarct expansion in our study. There were 26 patients of anterior AMI with initial dilatation but without early reperfusion at entry, most of whom had cardiac events. Thus, we believed that increased ESAi within 48 hours of onset of AMI can be considered an echocardiographic manifestation of infarct expansion, was suggested to be used as an early predictor for high risk after AMI.

The relationship between reperfusion and prognosis at the early stage after AMI. Early reperfusion after AMI can reduce infarct area, prevent infarct expansion and subsequent left ventricular dilatation. This study demonstrated that there were 15 anterior AMI with early reperfusion, 7 of whom had infarct expansion at entry, infarct area decreased significantly at 2 weeks after AMI, left ventricular size decreased and EF value

increased markedly at 3 months (0.47±0.06 VS 0.53± 0.04, P<0.05). Thus, echocardiography can serially and accurately assess dynamic changes of infarct expansion and ventricular remodeling after AMI.

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