Right Ventricular Functions After Anterior and Anteroseptal Myocardial Infarction

ÖN YÜZ VE ANTEROSEPTAL MİYOKARD İNFARKTÜSÜ SONRASI SAĞ VENTRİKÜL FONKSİYONLARI

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. Summary

Several investigators have evaluated right ventricular function in patients with acute right ventricle (RIO and acute inferior infarctions by echocardiography but studies of changes in right ventricular function during anterior and anteroseptal myocardial infarction are few. The purpose of this study was to investigate the extent of right ventricular dysfunction and to determine the importance of interventricular septum (IVS) involvement on RV functions after early phase of anterior-antemseptal myocardial infarction (A-ASP MI). We studied 24patients (23 men, I woman; mean age 55+10) with A and ASP MI and 14 control subjects (13 men. I woman; mean age 51+10). They had no historical or electrocardiographic evidence of a previous MI. Twelve patients had A MI and 12 patients had ASP MI. They had no heart failure symptoms. Two-dimensional and doppler echocardiography was performed in all patients in the first 72 hours. We used the ejection fraction (EF) , sizes of RV, preejection period/ejection time (PEP/ET) for evaluating the systolic functions ofRV Among the 24 patients with A-MI and ASP-MI, global left ventricular EF within the fust 72 hrs was 43+9.2%. The global EF of the LV averaged 62.2+4.1% in the normal subjects (p<0.05). The septal contraction was decreased in all patients. The RV EF was 6().9±6.7%j in the patient population and 61.8+4.6%) in the control group (p=non-signiftcant). There were no differences in systolic and diastolic diameters between the study group and normal subjects (1.75+0.9 cm vs 1.7+1.1 cm in systolic diameter, 2.56+0.26 cm vs 2.35+0.29 cm in diastolic diameter, respectively. P>0.05). The PEP/ET was same in both groups (p>0.05). We used the E/A ratio, deceleration time of tricuspid flow and vena caval index (VC1 index) for evaluating the diastolic functions of RV. There were no differences in E/A ratio (1.1+0.8 vs 1.04+0.09), in deceleration time of tricuspidalflow (136+43 sec vs 129+20 sec) and in VC1 index (49+7% vs 52+11%) between the study group and normal subjects (p>0.05). It is concluded that the right ventricular functions are not affected in the early phase of anterior and anteroseptal myocardial infarction. The integrity of interventricular septum contraction is not important for the maintenance of right ventricular function in patients with impaired left ventricular function.

Key Words: Echocardiography, Myocardial infarction, Right ventricular function

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Özet

Bir çok araştırıcı akut sağ ventrikül (SV) ve akut inferior miyokarel infarktüsü (MI) sonrası ekokardiyografik olarak sağ ventrikül fonksiyonlarını çalışmıştır. Ancak ön yüz ve anteroseptal MI sonrası sağ ventrikül fonksiyonlarını inceleyen çalışına sayısı sınırlıdır. Bu çalışmanın amacı, ön yüz ve anteroseptal MI sonrası erken dönemde sağ ventrikül fonksiyonlarında değişiklik olup olmadığını, değişiklik varsa buna interventriküler septum tutuluşunun katkısını araştırmaktır. Çalışmaya 24 ön yüz ve anteroseptal MI'lü (23 erkek, 1 kadın, ortalama yaş 55+10) hasta ile 14 kontrol olgusu (13 erkek, 1 kadın, ortalama yaş 51+10) alındı. Hiç bir hastada kalp yetmezliği semptomları yoktu. Ml'nün il/c 72 saatinde tüm hastalara iki boyutlu ve doppler ekokardiyografik inceleme yapıldı. SV sistolik fonksiyonlarım değerlendirmede ejeksiyon fraksiyonu (EF), SV çapları, preejeksiyon period/ejeksiyon zamanı (PEP/ET) kullanıldı.Kontrol grubunda sol ventrikül EF %60.9+6.7 olmasına karşın MI'lü 24 hastada ilk 72 saat içindeki global sol ventrikül EF %43+9.2 idi (p<0.05). Tüm hastalarda septal kontraksiyon azalmıştı. Çalışma grubu ve kontrol grubu arasında SV sistolik ve diastolik çapları arasında bir fark saptanmadı (sistolik çaplar 1.75+0.9 cm'e 1.7+1.1 cm, diastolik çaplar 2.56+0.26 cm'e 2.35+0.29 cm. P>0.05) . PEP/ET her iki grupta da benzerdi. SV diastolik fonksiyonlarını değerlendirmek için trikuspid akını E/A oranı, deselerasyon zamanı ve vena caval index (VCI indeks) kullanıldı. E/A oranında (1.1+0.8 'e 1.04+0.09), trikuspid akım deselerasyon zamanında (136+43 sn'e 129+20 sn) ve VCÎ indeksinde (%49+7 'e %52+ll) çalışma grubu ve kontrol olgular arasında farklılık saptanmadı (p>0.05). Çalışmanın sonunda; ön yüz ve anteroseptal Ml'nün erken döneminde sağ ventrikül fonksiyonlarının etkilenmediği ve interventriküler septum kontraksiyon bütünlüğünün, bozulmuş sol ventrikül fonksiyonlu hastalarda sağ ventrikül fonksiyonlarının devamlılığı açısından önemli olmadığı sonucuna varıldı.

Anahtar Kelimeler: Ekokardiyografi, Miyokard infarktüsü, Sağ ventrikül fonksiyonu

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More precise characterization of right ventricular function should improve our understanding of pathophysiology and permit more rational manage-

nt of cardiac disorders. Several investigators
e evaluated right ventricular function in patients
h acute right ventricle and acute inferior infarc-
as (1,2) by echocardiography but studies of
nges in right ventricular function during anteri-
and anteroseptal myocardial infarction are few.
per interpretation of ventricular function re-
res knowledge about right and left ventricular
ction. Based on hemodynamic data (3,4), the
nt ventricle responds quite differently to inferior
n it does to anterior infarction. The purpose of
s study was to investigate the extent of right ven-
ular dysfunction and to determine the impor-
ce of interventricular septum (IVS) involvement
RV functions after early phase of anterior-an-
septal myocardial infarction (A-ASP MI) by
ocardiography.

Materials and Methods

Patients

Twelve patients with anterior myocardial in-
tion (A-MI) and 12 patients with anteroseptal
ocardial infarction (ASP-MI) and 14 normal sub-
ts with good echogenity were studied. The mean
of populations was 55±10 (23 men, 1 women)
of normal subjects was 51±10 (13 men, 1
man) (Table 1). The diagnosis of acute MI was
de when the following criteria were present:

1) prolonged chest pain consistent with my-
rdial ischemia (at least 15 minutes) 2) electro-
diographic changes including evolving ST-T
ve changes and pathologic Q waves and 3) at
st twice the normal elevation in serum creatinine
ase with MB isoenzyme ≥5. Patients with his-
ical or electrocardiographic evidence of prior
coexisting congenital or acquired valvular dis-
e, pulmonary hypertension due to chronic ob-
ctive pulmonary disease, Killip II-III-IV heart
ure and patients who have received beta block-
therapy were excluded. All patients received
nventional therapy in the coronary care unit
rombolytic therapy, nitrates, aspirin, heparin)
there were no substantial differences in therapy
ong patients.

Echocardiography

M-mode and two-dimensional echocardio-
ms were performed with the patient in the left lateral position with a Hewlett-Packard Sonos 2500 model echocardiograph (Hewlett-Packard Co., Andover, Mass.) in the parasternal long and short axis, apical four and two chamber views within the first 72 hours. Simultaneously with the echocardiogram, an electrocardiogram was recorded. The systolic and diastolic diameters of right ventricle {RV) and left ventricle (LV) were obtained in the parasternal long axis view. Pulsed wave doppler recordings of the RV inflow and pulmonary flow were recorded from the parasternal short axis view. The sample volume was superimposed on the two-dimensional image of the tips of the tricuspid leaflets and below the pulmonary valve. LV inflow was obtained from the apical four-chamber view when the sample volume was at the tips of the mitral valve leaflets. The preejection period (PEP), ejection time (ET) of the RV, which show the systolic functions of RV, were measured and PEP/ET were calculated (Figure 1). The E velocity and A velocity, E/A ratio and deceleration time of tricuspidal flow were measured (Figure 2). Vena cava inferior diameters before and after inspiration were measured about 2 cm away from the entrance to the right atrium. The respiratory caval index (VCI index) was defined as the percentage of change in inferior vena caval diameter after inspiration. Left ventricular ejection fraction (LV EF) was calculated with the biplane area-length method and right ventricular ejection fraction (RV EF) was calculated with the single plane area-length method in the apical four chamber views. All echocardiography measurements were perforated by the same experienced observer blinded to the clinical findings and results were averaged. The net results were the average of three measurements (all of the patients were in sinusal rhythm).

Statistical Analysis

Results are reported as mean±SD. Student's t test was used for quantitative data. A p value <0.05 was considered to be significant.

Results

The demographic features of the patients and control subjects are shown in Table 1.

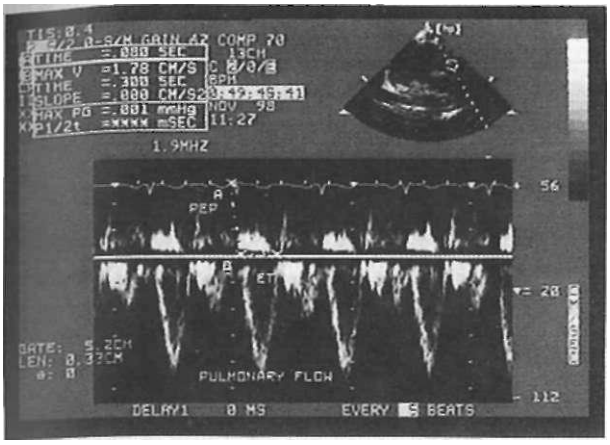

Figure 1. The pulmonary flow from the parasternal short axis echocardiographic view.
(PGP: Preejection period, ET: Ejection time)

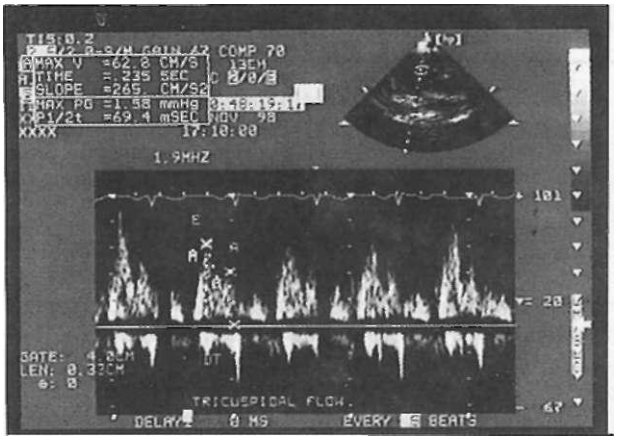

Figure 2. The tricuspidal flow from the parasternal short axis echocardiographic view.
(E: Early diastolic filling of the right ventricle, A: Atrial contraction, DT: Deceleration time of the right ventricle)

RV systolic functions

We used the ejection fraction , sizes of RV, PEP/ET for evaluating the systolic functions of RV. Among the 24 patients with A-MI and ASP-MI, global LV EF within the first 72 hrs was 43±9.2%. The global EF of the LV averaged 62.2±4.1% in the normal subjects ($p<0.05$). The septal contraction was decreased in all patients. The RV EF was 60.9±6.7% in the patient population and 61.8±4.6% in the control group (p=non-significant).There were no differences in systolic and diastolic diameters between the study group and normal subjects (1.75±0.9 cm vs 1.7±1.1 cm in systolic diameter, 2.56±0.26 cm vs 2.35±0.29 cm in diastolic diameter, respectively. $P>0.05$). The PEP/ET was same in both groups ($p>0.05$) (Table 2).

RV diastolic functions

We used the E/A ratio, deceleration time of tricuspid flow and vena caval index to evaluate the diastolic functions of RV. There were no differences in E/A ratio (1.1±0.8 vs 1.04±0.09), in deceleration time of tricuspidal flow (136±43 sec vs 129±20 sec) and in VCI index (49±7% vs 52±11%) between the study group and normal subjects ($p>0.05$). Echocardiographic parameters are also listed in Table 3.

Discussion

Factors that influence right ventricular function in coronary artery disease which include right ventricular ischemia, septal shifting and afterload of RV, are reported to be complex (5). Many inves-

Table 1. The demographic data of patients and normal subjects

	A and ASP-MI (n=24)	Control (n=14)	P
Age	55±10	51±10	ns
Gender (M/F)	23/1	13/1	
Killip <Il	24	14	
Systolic blood pressure (mmHg)	108±23	115±21	ns
Diastolic blood pressure (mmHg)	74±19	76±15	ns
Heart rate (/sec)	72±13	69±14	ns

(A: Anterior, ASP: Anteroseptal, MI: Myocardial Infarction)

Table **2.** The echocardiographic parameters which reflect systolic functions of right ventricle in patients with anterior and anteroseptal myocardial infarction and control subjects

	A and ASP-MI (n=24)	Control (n=14)	p	
LV EF (%)	43±9.2	62.2±4.1	<0.05	
RV EF (%)	60.9±6.7	61.8±4.6	ns	
RVes (cm)	1.75±0.9	1.71±1.1	ns	*
RVed (cm)	2.56±0.26	2.35±0.29	ns	
RV PEP/ET	0.39±0.08	0.35±0.05	ns	

(A: Anterior, ASP: Anteroseptal, MI: Myocardial Infarction, LV EF: Left Ventricular Ejection Fraction, RV EF: Right Ventricular Ejection Fraction, RVes: Right Ventricular end-systolic diameter, RVed: Right Ventricular end-diastolic diameter, RV PEP/ET: Right Ventricular Precjectionperiod / Ejection Time)

Table 3. The echocardiographic parameters which reflect diastolic functions of right ventricle in patients with anterior and anteroseptal myocardial infarction and control subjects

	A and ASP-MI (n=24)	Control (n=14)	p
Tricuspid flow E/A	1.1*0.18	1.04±0.09	ns
Tricuspid flow DT (sec)	136±43	129±20	ns
V C i index (%)	49±7	52±1	ns

(A: Anterior, ASP: Anteroseptal, MI: Myocardial Infarction, DT: Deceleration Time, VCPVena Cava Inferior)

tigators claim that RV functions might be depressed after anterior transmural myocardial infarction with septal involvement. Some reports showed that the IVS involvement in patients with acute myocardial ischemia was the major determinant of the RV function (6,7). But some investigators that RV functions were primarily related to pulmonary hypertension-associated LV dysfunction (8,9). In their necropsy series, Isner et al (10) emphasized the extent of septal infarction as a principal determinant of right ventricular decompensation. Nakamura et al (11) showed that the presence or absence of IVS involvement was significant determinant of RV response to exercise and IVS contraction might be important for the maintenance of RV function in patients with impaired LV function. Also, Fixer et al (12) showed that IVS ischemia induced deteriotation in RV function. Berger et al (8) reported that reduced RV reserve in patients with coronary artery disease is primarily related to pulmonary hypertension-associated LV dysfunction during exercise. Marmor et al (9) also showed that in patients with anterior infarction, there was persistant impairment of LV function but only transient impairment of the RV and they reported that it was likely that increased RV afterload might play a partial role in its transient dysfunction. But Marmor's results were different from those reported by Reduto et al (2). These investigators have found no significant impairment in RV functions in 12 of 13 patients with anterior MI. Both Marmor and Reduto did not obtaine pulmonary artery pressures by hemodynamic investigation like us. They used clinical findings to determine increased RV afterload. In the Reduto's series, only 3 of their 13 patients with anterior MI were in clinical class II and none were in class III, whereas in the Marmor's series 11 of their 22 patients were in class II and 7 were in class III. The greater hemodynamic impairment of the left ventricle in Marmor's patients may explain the difference in the results of the two studies. In our series, all patients had depressed interventricular septal contraction. Hemodynamic investigation was not performed in our patients as it is an invasive procedure. We used Killip classification like Reduto and Marmor and Killip >II patients were not included in this study to eliminate the patients with high pulmonary artery pressure and to evaluate the impor-

tance of the contribution of IVS to the RV functions. The possible of influence of drug therapy on ventricular function was also considered. No patients received digoxin, diuretic or beta blocker during the study and nitroglycerine infusion was decreased gradually and stopped before 3 hours of the study. Thus, it is unlikely that drug therapy accounted for the depression or improvement in RV function in our patients with anterior-anteroseptal Ml.

In conclusion, we showed that impairment of septal contraction does not effect the RV function. We found no significant impairment in RV functions in patients with early phase anterior and anteroseptal Ml who had no heart failure. Because of its thin free wall and large surface area, the RV cannot adapt as readily as the LV to the development of high intracavitary pressure under comparable loading conditions. These anatomic insights support the concept that the RV performance is highly afterload dependent.

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