

## The Value of Tissue Doppler Imaging in Prediction of Multivessel Disease in Patients with Acute Inferior Myocardial Infarction Treated by Thrombolytic Therapy

*Trombolitik Tedavi Uygulanan Akut Alt Duvar Miyokard İnfarktüsünde Doku Doppler*

*Görüntülemenin Çok Damar Hastalığını Belirlemedeki Yeri*

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### Özet

**Amaç:** Akut alt duvar miyokard infarktüsü (AAMI) ST elevasyonlu MI'nin %40-50'sini oluşturur ve genellikle mortalitesi ön duvar MI'ne göre daha düşüktür. Çalışmalarda AAMI'de çok damar hastalığı 40-45% oranında gösterilmiştir. Bu çalışma trombolitik tedavi uygulanan akut alt duvar miyokard infarktüslerinde çok damar hastalığını saptamada doku Doppler görüntüleme yönteminin kullanılıp kullanılmayacağını araştırmak için yapıldı.

**Yöntem:** Çalışmaya ST elevasyonlu AAMI tanısı ile servisi-mize yatırılan, primer perkütan girişimi kabul etmeyen ve streptokinaz uygulanan 49 hasta alındı. Tüm hastalara yatışlarının ilk gününde 2-D Eko ve PW doku Doppler incelemesi, ilk 10 gün içerisinde ise koroner anjiyografileri yapıldı. Hastalar anjiyografilerine göre tek damar lezyonu saptananlar Grup-I, 2 ve 3 damar lezyonu saptananlar ise Grup-II olarak belirlendi. Doku Doppler incelemesi ile Sm, SmVTİ, Em, Am, Em/Am oranı, DT, Q-Sm, CT, İVRT değerleri ölçüldü.

**Bulgular:** Çalışmaya alınan hastaların %45'de çok damar hastalığı saptandı. Çok damar hastalarında duvar hareket skor indeksi yüksek bulundu. Ancak geleneksel Doppler parametreleri açısından gruplar arasında anlamlı fark saptanmadı. Doku Doppler görüntülemeye ise sistolik ve diyastolik parametreler açısından istatistiksel olarak anlamlı fark bulundu. Grup II'de mitral anulus lateral, septal, posterior ve anterior bölgelerde Sm, Em, Em/Am'nin azaldığı, DT, Q-Sm, İVRT'nin uzadığı saptandı. Regresyon analizinde, çok damar hastalığının bağımsız belirleyicileri mitral lateral Sm(r=0.79, p

**Sonuç:** Trombolitik tedavi uygulanan AAMI'de çok damar hastalarının tespiti ve tedavilerinin planlamasında non invazif bir yöntem olarak doku Doppler görüntüleme yönteminin kullanılabileceği kanaatindeyiz.

**AnahtarKelimeler:** Akut Miyokard infarktüsü, çok damar hastalığı, doku Doppler ekokardiyografi

### Abstract

**Objective:** Acute inferior myocardial infarctions (AIMI) consists the 40-50% of all ST elevated MI. Studies have showed that the rate of multivessel disease (MVD) is 40-45% in AIMI. Our study was designed to investigate whether tissue Doppler imaging can be used to determine MVD in patients with AIMI who were treated by thrombolytic therapy.

**Method:** 49 patients with AIMI who were admitted to our hospital and refused primary percutaneous angioplasty were enrolled in this study. Patients were treated with streptokinase as a thrombolytic therapy. All patients underwent to two dimensional echocardiographic and pulse wave tissue Doppler imaging examination on the day of admission and evaluated by coronary angiography within 10 days of admission. Patients with one vessel disease consisted the group I and patients with 2 or 3 vessel disease consisted the group II. Tissue Doppler imaging was performed and Sm, SmVTİ, Em, Am, Em/Am, DT, Q-Sm, CT, İVRT values were estimated.

**Results:** In our study group, 45% of patients were found to have MVD. The differences between the groups according to traditional Doppler parameters and LVEF were not significant. Tissue Doppler imaging showed that in group II when compared to group I; Sm, Em, Em/Am values were reduced, whereas DT, Q-Sm, İVRT values were increased. In regression analyses mitral lateral and septal Sm were found to be independant indicators of MVD.

**Conclusion:** We argued that the tissue Doppler imaging can be a usefull tool as a non invasive method for determining of MVD in patients with AIMI

**Keywords:** Myocardial infarction, multivessel disease, tissue Doppler echocardiography

### Introduction

In Western developed countries the cardiovascular disease is the major cause of morbidity and premature death (1). Inferior wall acute myocardial infarction (MI) consists the 40-50% of all ST elevated myocardial infarction and

prognosis is better than anterior infarction (2,3). Electrocardiography (ECG) reflects ST segment depression on precordial derivations in approximately half of the patients with acute inferior MI (4,5).

Previous studies reported that the patients with concomitant precordial ST segment depression have higher multivessel disease and higher in-hospital morbidity and mortality (6). Patients with inferior MI who have multivessel disease have been found to have higher rate of heart failure, lower EF, more extensive wall motion abnormalities in echocardiographic evaluation and have worse complications in short and long terms (7). Echocardiographic evaluation in patients with inferior MI can estimate the number of diseased vessels, as it shows the wall motion abnormalities supplied by coronary arteries and evaluation for left and right ventricular functions (8). The sensitivity of 2D echo is low due to visual evaluation of wall motion score index and lack of quantitative knowledge by this method. Concurrently, multivessel disease is not always accompanied by wall motion abnormalities, it can also be present in patients with normal left ventricular systolic functions.

Tissue Doppler imaging (TDI) has been in widespread clinical use to assess myocardial wall motion and non-invasive hemodynamics for many years (9,10). By this technique, wall motions can be evaluated segmentally or globally and systolic and diastolic functions can be assessed (11).

It is important to determine multivessel disease and identify the jeopardized myocardium in patients with inferior MI for prognostic evaluation. There are a number of studies for prognostic evaluation of these patients by using coronary angiography or myocardial perfusion scintigraphy. However, to our knowledge, there isn't any study carried out by TDI methods, so we aimed to investigate the value of tissue Doppler imaging in prediction of multivessel disease in patients with acute inferior myocardial infarction.

## **Material and Methods**

We enrolled forty-nine patients with acute inferior wall MI attending cardiology department. All of the patients were treated with fibrinolytic therapy because of refusing primary percutaneous coronary intervention (PCI). Acute MI was diagnosed according to World

Health Organisation criteria (12). Streptokinase was the choice of fibrinolytic therapy in all patients. Exclusion criteria were as follows: history of previous MI, patients with right ventricular infarction, left or right bundle branch block, severe valvular disease, congenital heart diseases or cardiomyopathies, history of coronary angioplasty or coronary artery bypass graft surgery and rhythm other than sinus. Informed consent was taken from all the patients before enrollment and the study was designed to comply with the ethical principles of our institution.

### *Electrocardiographic Definition*

The 12 lead ECGs of patients were taken at admission to coronary care unit, at 9th hour of admission and daily for every patient at 25 mm/sec in velocity. Acute inferior MI definition was made as  $\geq 1$ mm ST segment elevation in at least two derivations of D2, D3 and aVF (13).

### *Coronary Angiography and Heart Catheterization*

Conventional coronary angiography was performed with Philips Integris 5000 equipment in patients within 10 days after admission (14). Coronary obstruction of  $\geq 70\%$  were accepted as occlusive coronary artery disease (15). Angiographically, patients with two or more involved coronary arteries, were accepted as multivessel disease. Patients were divided into two groups according to their angiographic findings. Group-I: patients with single vessel disease, Group-II: patients with multivessel disease (patients with two or three vessels were involved). All patients underwent left ventriculography to evaluate wall motion and ejection fraction and during this process left ventricular end-diastolic pressures were measured. Each coronary angiographic evaluation was assessed by two different cardiologists who were blinded to clinical data of patients and the decision was made together.

### *Echocardiographic Examination*

All patients underwent echocardiographic examination within 24 hours of admission with Vingmed System 5 Doppler echocardiographic



(GE, Norway) unit with a 2.5 MHz electronic transducer. The echocardiographic evaluation was performed at left lateral decubitus position from standard parasternal long and short axis views and apical two and four chamber views appropriate to American Echocardiography Association report (16). Left ventricular diastolic diameter (LVDD), left ventricular systolic diameter (LVSD), left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), left ventricular ejection fraction (LVEF), left atrium (LA) diameter was measured by 2D echocardiography. Ejection fraction was determined by modified Simpson method (17).

Doppler echocardiographic recording allowed analysis of the diastolic mitral flow velocities of the early diastolic peak flow velocity (E wave), the late diastolic peak flow velocity (A wave), the E/A ratio, the deceleration time of early diastolic wave (DT), and isovolumic relaxation time (IVRT). Left ventricular wall motion was examined qualitatively in 16 segments, appropriate to American Echocardiography Association recommendations (18). 50% or more thickening of left ventricular wall was accepted as normal myocardium. When compared to normal myocardial segments, 40% or less thickening was defined as hypokinesia, absence of systolic thickening was defined as akinesia and paradoxical excursion during systole was defined as dyskinesia. Each segment was given a score according to its contractility as follows: 1- Normal, 2- Hypokinesia, 3-Akinesia, 4- Dyskinesia, 5- Aneurysm. Then wall motion score index was estimated (19).

In apical 2 and 4 chamber views tissue Doppler pulsed wave (PWTD) sample volume was placed to points where left ventricular lateral, septum, inferior and anterior walls intersect with mitral annulus respectively (20). Sample volume was provided to be parallel to wall axis and the following measurements were made: Systolic myocardial velocity (Sm), systolic myocardial velocity square (SmVTI), peak velocity of early diastolic wave (Em), peak velocity of late diastolic wave (Am), Em deceleration time (EmDT), Em/Am ratio, precontraction time (Q-Sm), left ventricular contraction time (CT), isovolumic relaxation time (IVRT) (21). All echo-

cardiographic measurements were made in three consecutive cycles and the average values were estimated.

### Statistical Analyses

SPSS 10.0 was used for statistical analyses. Descriptive statistic results were expressed as mean, standard deviation, median, minimum and maximum or as number and percentage for numeric and categorical parameters, respectively. To compare qualitative values, Student's t test and chi square test were used. According to the distribution, the differences between the groups for numeric parameters were compared by Student's t-test or the Mann-Whitney U test. The significance level was assumed as  $P < 0.05$ .

### Results

There was not significant difference between groups according to age, gender, hypertension, family history of coronary artery disease, diabetes mellitus, smoking, total cholesterol, triglyceride, peak creatinin kinase (CK) and CK-MB values ( $p > 0.05$ ). But heart rate, pre-infarction angina existence and Killip class was found higher in group-II than group-I. ( $p < 0.01$ ,  $p < 0.001$ ,  $p < 0.001$  respectively) (Table 1).

### Electrocardiographic Findings

All patients had ST segment elevation in inferior leads on surface ECG. In 24 patients (48%) there was not precordial ST segment depression, 11 patients (23%) has precordial ST segment depression in V1-V4 leads and 14 patients (29%) in V4-V6 leads.

### Coronary Angiography Findings

27 patients ( 55% ) were found to have one vessel disease ( group- I ) and 22 patients (45%) were found to have multivessel disease (two or three vessel involved) (group-II). Distribution of lesions was as follows: Right coronary artery (RCA) lesion in 19 patient (38.7%), Circumflex (CX) lesion in 8 patient (16.3%), RCA and left anterior descending (LAD) lesion in 8 patient (16.3%), RCA and CX lesion in 8 patient (16.3%), RCA, CX and LAD lesion in 6 patient (12.2%).



**Table 1.** The basic characteristics of patients

	Grup I (n=27)	Grup II (n=22)	P value
Age(year)	52±10	58±8	NS
Male (%)	%96 (26)	%77 (17)	NS
Female (%)	%4 (1)	%23 (5)	NS
Systolic blood pressure(mmHg)	128±27	131±33	NS
Diastolic blood pressure(mmHg)	78±14	81±20	NS
Family history(%)	29 (8)	31(7)	NS
Smoking (%)	74 (20)	54(12)	NS
Diabetes Mellitus(%)	3(1)	13(3)	NS
Triglyceride (mg/dl)	168±54	205±73	NS
Total cholesterol(mg/dl)	213±39	225±34	NS
Preinfarction angina (%)	22.2	77.7	<b>p&lt;0.001</b>
In hospital mortality (%)	3	4	NS
Killip class	1.07±0.2	1.78±0.8	<b>p&lt;0.001</b>
Pulse(/dk)	69±18	82±18	<b>p&lt;0.01</b>
Peak CK (IU)	1666±1224	2464±1874	NS
Peak CK-MB (IU)	268±290	398±406	NS

CK: Creatinin kinase, CK-MB: Creatinin kinase muscle brain, Data are expressed as means±SD, NS: Non significant p>0.05

In our study left ventricular end-diastolic pressure was found statistically higher in patients with multivessel disease compared to patients with single vessel disease ( $21 \pm 8$  mmHg,  $12 \pm 4$  mmHg,  $p < 0.001$ , respectively).

### Echocardiographic Findings

Left ventricular systolic and diastolic volumes measured by 2D echocardiography were higher in group-II. ( $p < 0.05$ ,  $p < 0.01$ ; respectively). Left ventricular systolic and diastolic diameters were higher in group-II ( $p < 0.01$ ,  $p < 0.001$ ; respectively). Left ventricular EF was lower in group-II, but the difference was not significant. Wall motion score index, was found to be higher in group-II ( $p < 0.001$ ). Except E/A ratio there was not any statistically significant difference between two groups according to E, A, DT and IVRT (Table 2).

### Tissue doppler imaging (TDI) findings

Systolic and diastolic parameters evaluated by TDI were found to be deteriorated in all four regions of mitral annulus in patients with multivessel disease. In patients with multivessel disease peak systolic velocity Sm and SmVTI were found to be decreased in all four regions.

( $p < 0.001$ ,  $p < 0.001$ , respectively). There was not any significant difference between Group-I and group-II according to traditional PW Doppler diastolic parameters, but TDI parameters were found to be deteriorated markedly in group-II. In group-II Em was found to be significantly lower in all four regions ( $p < 0.001$ ). The Am at lateral and posterior annulus regions between two groups were significantly different ( $p < 0.05$ ) but there was not any difference at septal and anterior annulus regions. In group-II, Em/Am was smaller than group-I in all four regions and the difference between the two groups was statistically significant ( $p < 0.001$ ). In group II, DT, Q- Sm and IVRT was found to be markedly prolonged in all four regions and contraction time was found to be markedly shortened (Table 3).

In multiple logistic regression analyses in which mitral lateral Sm, mitral septal Sm, mitral inferior Sm, mitral anterior Sm, wall motion score index and EF were used as independent variables, mitral lateral Sm ( $r = 0.79$ ,  $p < 0.05$ ) and mitral septal Sm ( $r = 0.83$ ,  $p < 0.05$ ) were found as independent predictors of multivessel disease. Em, Em/Am were found lower in patients with multivessel disease compared to patients with single vessel disease.



**Table 2.** 2 D and PW Doppler echocardiographic parameters of patient

	<b>Grup-I (n=27)</b>	<b>Grup-II (n=22)</b>	<b>P value</b>
LVDD (mm)	47.2±4.3	52.5±6.8	<b>p&lt;0.01</b>
LVSD (mm)	33.2±7.1	41.5±6.8	<b>p&lt;0.001</b>
LVDV (ml)	59.7±14.2	78.6±24.0	<b>p&lt;0.01</b>
LVSV (ml)	28.6±12.0	43.7±24.2	<b>p&lt;0.05</b>
LVEF (%)	54±13	50±13	NS
LA (mm)	31.6±3	37.7±4	<b>p&lt;0.001</b>
Wall motion score index	0.40±0.28	0.74±0.46	<b>p&lt;0.001</b>
E (cm/sn)	5.75±15.2	5.29±16.2	NS
A (cm/sn)	6.15±14.0	6.51±16.7	NS
E/A	0.93±0.24	0.76 ±0.27	<b>p&lt;0.05</b>
DT (msn)	173±42	188±32	NS
IVRT (msn)	101±24	107±27	NS

LVDD: Left ventricular diastolic diameter, LVSD: Left ventricular systolic diameter, LVEF: Left ventricular ejection fraction, LVDV: Left ventricular diastolic volume, LVSV: Left ventricular systolic volume, LA: Left atrium diameter, IVRT: Izovolumetric relaxation time, DT: Deseleration time, E: Early diastolic peak flow velocity, A: Late diastolic peak flow velocity, NS: Non significant (p>0.05)

## Discussion

The studies that have been carried out show that precordial ST segment depression can be a predictor of multivessel disease in patients with inferior MI. In acute inferior MI, prognosis is related to accompanied multivessel disease, left ventricular systolic dysfunction and presence of right ventricular infarctions (4).

Coronary angiography is the gold standard to determine coronary artery disease existence and prevalence. But it is invasive and its complications restricts its routine use. M-Mode and 2D echocardiography have important role in evaluating left ventricular functions as they are non-invasive methods. Khattar et al have investigated the role of 2D echocardiography in determining multivessel disease and have found the sensitivity of 2D echo as 68% (22).

TDI method is useful in evaluating left ventricular wall motions segmentally and globally. Moreover it provides more data about left ventricular systolic and diastolic functions compared to traditional echocardiographic methods (9,10). Palmes et al have showed the decrease of systolic velocity in left ventricular lateral segments by TDI in patients with critical Cx artery lesions whose lateral wall motions were normal by 2D echo (23). Brunch et al. have evaluated phasic myocardial velocity determi-

ned by TDI in patients with critical LAD lesions and normal left ventricular systolic functions and have found that systolic myocardial velocity is decreased in these patients compared to normal subjects (24). Thus they have mentioned that TDI is predictive in diagnosing critical coronary artery disease. Edvardsen et al have evaluated left ventricular wall motions by TDI in patients with LAD lesions and they have found that early peak systolic velocity (Em) is markedly decreased especially in apical septum in patients with complete LAD occlusions (18). Furthermore they have found that myocardial systolic velocities are lower in ischemic regions compared to non ischemic regions.

In our study we found that Sm and SmVTI are lower in patients with multivessel disease compared to patients with single vessel disease. (p<0.001, p<0.001 respectively). Our results were consistent with other studies carried out before.

Recent study have showed that Sm and Em is decreased and precontraction time is prolonged in infarct regions in patients with myocardial infarctions (25). As a novel echocardiographic technique, real-time 3D echocardiography and TDI derived strain rate echocardi-



**Table 3.** TDI parameters of patients

	<b>Grup-I (n=27)</b>	<b>Grup-II (n=22)</b>	<b>P value</b>
<b>Mitral lateral</b>			
$E_m$ (cm/sn)	10.9±3.2	6.7±2.0	<b>p&lt;0.001</b>
$A_m$ (cm/sn)	8.9±2.6	10.9±2.5	<b>p&lt;0.05</b>
$E_m/A_m$	1.30±0.5	0.64±0.2	<b>p&lt;0.001</b>
DT (msn)	133±31	187±36	<b>p&lt;0.001</b>
Sm (cm/sn)	7.6±1.7	5.0±0.9	<b>p&lt;0.001</b>
SmVTİ (cm)	1.6±0.3	1.1±0.3	<b>p&lt;0.001</b>
Q-Sm (msn)	147±32	184±39	<b>p&lt;0.001</b>
CT (msn)	343±36	291±35	<b>p&lt;0.001</b>
IVRT (msn)	43±12	85±34	<b>p&lt;0.001</b>
<b>Mitral Septal</b>			
$E_m$ (cm/sn)	8.6±2.0	6.7±2.0	<b>p&lt;0.001</b>
$A_m$ (cm/sn)	9.0±2.4	9.1±1.9	NS
$E_m/A_m$	1.02±0.3	0.66±0.8	<b>p&lt;0.001</b>
DT (msn)	154±29	180±42	<b>p&lt;0.05</b>
Sm (cm/sn)	7.1±1.0	5.3±0.9	<b>p&lt;0.001</b>
SmVTİ (cm)	1.6±0.3	1.1±0.3	<b>p&lt;0.001</b>
Q-Sm (msn)	29±29	153±27	<b>p&lt;0.01</b>
CT (msn)	355±38	293±45	<b>p&lt;0.001</b>
IVRT (msn)	52±13	79±24	<b>p&lt;0.001</b>
<b>Mitral Anterior</b>			
$E_m$ (cm/sn)	8.9±2.0	6.1±2.0	<b>p&lt;0.001</b>
$A_m$ (cm/sn)	7.8±1.8	9.0±2.2	NS
$E_m/A_m$	1.2±0.3	0.71±0.2	<b>p&lt;0.001</b>
DT (msn)	158±22	187±40	<b>p&lt;0.01</b>
Sm (cm/sn)	7.0±1.0	5.7±1.0	<b>p&lt;0.001</b>
SmVTİ (cm)	22±32	153±31	<b>p&lt;0.01</b>
CT (msn)	361±38	299±45	<b>p&lt;0.001</b>
IVRT (msn)	50±16	86±33	<b>p&lt;0.001</b>
<b>Mitral Posterior</b>			
$E_m$ (cm/sn)	9.6±2.7	6.1±1.8	<b>p&lt;0.001</b>
$A_m$ (cm/sn)	9.3±2.1	1.5±1.7	<b>p&lt;0.05</b>
$E_m/A_m$	1.0±0.3	0.57±0.6	<b>p&lt;0.001</b>
DT (msn)	141±24	193±34	<b>p&lt;0.001</b>
Sm (cm/sn)	7.4±1.7	5.3±1.4	<b>p&lt;0.001</b>
SmVTİ (cm)	1.7±0.4	1.1±0.3	<b>p&lt;0.001</b>
Q-Sm (msn)	141±34	176±41	<b>p&lt;0.01</b>
CT (msn)	342±67	290±51	<b>p&lt;0.001</b>
IVRT (msn)	51±16	82±31	<b>p&lt;0.001</b>

Em: Early diastolic peak velocity; Am: Late diastolic peak velocity; CT: Contraction time; DT: Deceleration time; IVRT: İzovolumetric relaxation time; Sm: Systolic myocardial velocity; SmVTİ: Systolic myocardial velocity square; Q-Sm: Precontraction time; NS: Non significant ( $p>0.05$ )

graphy was used for evaluation of patients with myocardial infarction (26,27). M. Kidawa et al. showed that Sm and Em decreased in infarct regions and isovolumetric contraction time was

prolonged in patients with right ventricular MI and 3-D echocardiography was found a useful method especially in patients with threshold of  $EF < 51\%$  (26). Strain rate was found as a



powerful measurement of contractility which is less influenced by changes in cardiac load and structure. Thus, peak systolic strain rate is the more relevant parameter to assess myocardial contractile function noninvasively. In our study we showed that contraction time is shortened and precontraction time is prolonged in patients with multivessel disease. In patients with multivessel disease, this can be explained by total ischemic load leading to reduction in contractile reserve and myocardial cell damage. As a result myocardial contraction capacity is decreased. Left ventricular diastolic functions have been shown to deteriorate globally and segmentally before systolic functions declines (28,29).

In our study we observed that, left ventricular filling pressure of patients with multivessel disease was higher and the diastolic functions deteriorates as left ventricular filling pressure increases. Em, Em/Am were found lower in patients with multivessel disease. This result can be thought as an evidence of relaxation impairment in patients with multivessel disease.

The limitations of our study are the small number of patients and the lack of strain rate echocardiography studies.

As a result we established that, diastolic and systolic parameters which cannot be obtained by traditional echocardiographic methods are impaired in patients with multivessel disease with acute inferior MI. For these reasons we believe that TDI can be useful as a non invasive method in determining patients with multivessel disease and risk stratification in patients with acute MI. Further studies with larger number of patients using 3D and strain rate echocardiography may provide further information.

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