Assessment of the Diastolic Dysfunction in Patients with Acute Myocardial Infarction by Analyzing LVIF Pattern on Pulsed Doppler Echocardiography

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Summary:

To assess diastolic filling after acute myocardial infarction (AMI), 82 patients underwent Doppler Echocardiographic examination within 72 hours of AMI. Left Ventricular inflow pattern (LVIF) was carefully analyzed to study the diastolic function. 20 subjects served as control group. In 67 patients (81.7%) LVIF pattern revealed findings of increased diastolic dysfunction (i.e., small "E" wave with prolonged deceleration time and a normal or increased amplitude of "A" wave. 10 patients (12%) had big early diastolic filling wave (E wave) and a normal or decreased amplitude at atrial contraction (A wave). 5 patients (4%) had a relatively normal looking LVIF pattern. In conclusion, LVIF pattern is a good indicator of diastolic function and there is impairment of diastolic function after AMI.

Introduction:

Acute Myocardial infarction (AMI) is well known to produce impaired systolic function, whereas reperfusion may attenuate the measured decline in systolic functions[1,2]. The concept of myocardial stunning has been proposed to explain the delayed recovery in systolic myocardial performance after AMI. The impact of AMI and reperfusion on left ventricular diastolic performance and the possibility of diastolic stunning is less clear. In this regard, it has been shown that transient myocardial ischaemia can result in diastole filling abnormalities in addition to systolic dysfunction[3,4,5]. Furthermore, experimental studies has indicated that AMI leads to abnormal left ventricle stiffness or relaxation that may improve with time[6,7]. Doppler Echocardiographic-derived indexes of left ventricular inflow velocity may provide a clinically useful measure of left ventricular diastolic function[7-9].

So, the aim of the study was to determine the influence of AMI on left ventricular chamber stiffness and relaxation by analyzing left ventricular inflow (LVIF) pattern on Doppler Echocardiography.

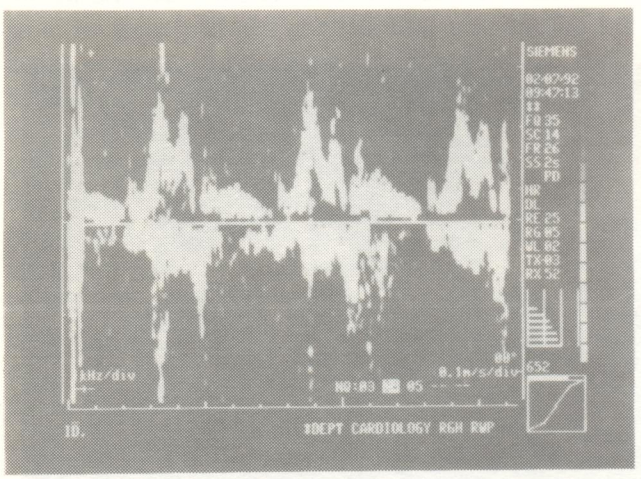

Figure 1.
LVIF pattern in a young man of 38. The peak early mitral flow velocity (E wave) is much taller than the flow velocity at atrial contraction (A wave). Mitral deceleration time is measured from the peak of the "E wave" to its terminal end.

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Methods:

I. Study Group:

Pulsed Doppler Echocardiogram were performed in 82 patients (62 men & 20 women) within 72 hours of admission with AMI as determined by typical history, standard electrocardiographic changes and an increased in cardiac enzymes. They were all in sinus rhythm and had a technically adequate study. The patients ranged in age from 38 to 78 years (mean 51±8) .12 Patients with MI had a history of systemic hypertension and 22 were known to have Diabetes Mellitus. Functional status was defined as follows:

i) Class I : asymptomatic,
ii) Class II : shortness of breath with moderate exertion,
iii) Class III : shortness of breath with mild exertion,
iv) Class IV : shortness of breath with minimal exertion.

II Control Group:

Doppler studies were also performed in 20 patients with the history of atypical chest pain or palpitation (men 14, women 6). These patients ranged in age from 30 to 65 years (mean 41±9). No patient had MI as determined by history, electrocardiogram and wall motion analysis. The patients were divided into two groups, i.e., patients above and below the age of 40.

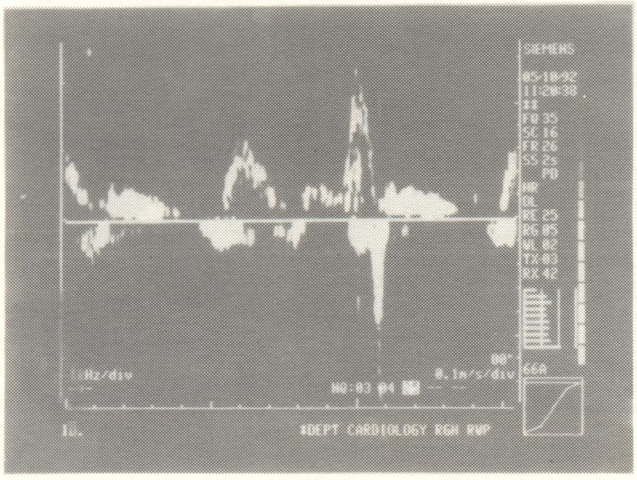

Figure 2.
LVIF in a 55 years old male with AMI showing increases in mitral deceleration time and flow velocity at atrial contraction (A) with decreases in peak early mitral flow (E) and ratio of peak early flow (E) to flow at atrial contraction (A).

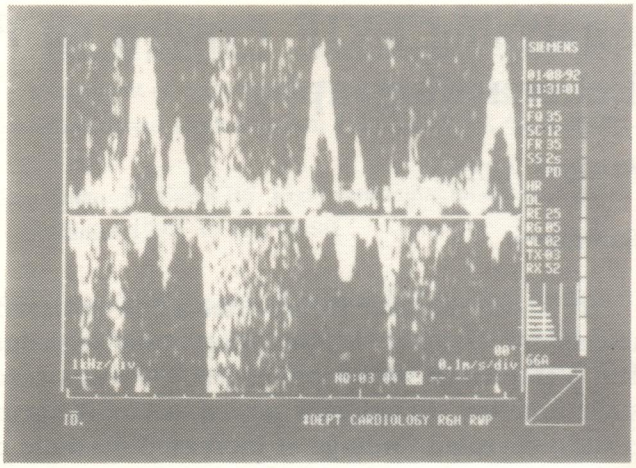

Figure 3.
LVIF in a 67 years old male with extensive anterolateral myocardial infarction and LVF. This pattern (II) is characterized by a decrease in deceleration time and flow velocity at atrial contraction (A) and increase in peak early flow velocity (E) and E/A ratio.

Doppler Studies:

Doppler examination of LV inflow were performed with a 3.5-MHz transducer using the apical 4 chamber view, the doppler sample volume was placed in the mitral valve funnel on the LV side of the mitral annulus. The sample volume was placed as parallel to flow as possible to maximize recorded velocities[10]. Peak filling velocity of the early rapid filling wave (E) and the atrial contraction wave (A wave) as well as E/A ratio were determined. Since we lacked the facilities of a digitizing pad and a micro computer, we could not measure the exact amplitude of these waves (time velocity integral interval could not be determined because of the same reasons). Left atrial size and LV dimensions were also measured from parasternal long axis view on trans thoracic echocardiography.

Results:

I. **Normal Control:**

In this group of patients, the "E" wave was bigger than "A" and in most of the patients below 50 years of age. In patients above 50 years "E/A" ratio was either 1 or less than 1 (i.e., E wave is smaller than A). (Fig. 1).

II. **Study Group:**

The patient in this study could be separated into two groups that showed distinctly different mitral flow velocity patterns compared with normal subjects.

i) **Group I:**

Most of the patients (i.e. 67 patients) in this group had a LVIF pattern with the following findings; a decreased peak early mitral inflow velocities but normal or increased mitral flow velocities at atrial contraction, a reduced ratio of peak early mitral flow velocity to that atrial contraction and a prolonged mitral deceleration time. Most of these patients were asymptomatic and only 19 patients had clinical evidence of impaired LV function in the form of basal crepitation or a third heart sound. Almost all of them were found to have sustained extensive anterior and anterolateral myocardial infarction. (Fig. 2).

ii) **Group II:**

In this group, patients were symptomatic, i.e., they complained of dyspnoea on mild exertion or rest (NYHA) and clinically most of them were in class II to IV of NYHA. Their physical examination revealed signs of high filling pressure (i.e., engorged neck veins, Gallop rhythm and basal crepitation). Their LVIF had one or the following findings; a normal or increased peak mitral flow velocity atrial systolic function. The long mitral deceleration time frequently seen in these patients may reflect a prolonged fall in left ventricular pressure associated with the impaired relaxation and the low early diastolic filling rates[12]. LVIF pattern of groups II patients (those who were symptomatic and were in functional class II to IV

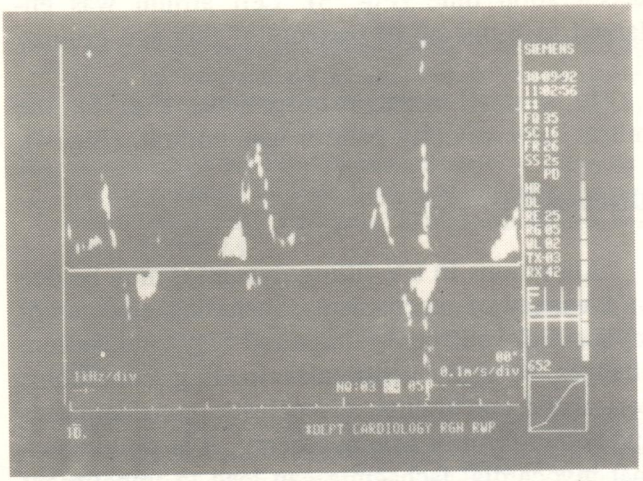

Figure 4.
This normal looking (normalized) LVIF pattern was seen in a 47 years old male with acute anteroseptal infarction.

NYHA) revealed increased amplitude of "E" wave and decreased or normal "A" wave. This has been termed "pattern II" (Appleton et al[12].

This type of pattern was seen in patients who had higher filling pressure on cardiac catheterization. These patients were also found to have impaired LV relaxation. However, the normal or increased peak mitral flow velocity in early diastole in these patients suggest that an elevated left atrial pressure normalized or increased the early diastolic transmitral pressure gradient and masked the expected effect of the relaxation abnormality on this gradient and Doppler variable[14]. It was discovered by the some group of investigators (i.e., Appleton et al) that short mitral deceleration time was inversely correlated with the left ventricular rapid filling wave (E wave). This pattern likely reflects a marked decrease in LV chamber compliance, which is characteristic of advanced disease of various origins[13].

In the present study, most patients with this pattern had left atrial enlargement. The decrease in velocity at atrial contraction may be related to systolic atrial dysfunctions perhaps as a result of a long standing increase in atrial after load from an elevated LV pressure or increased viscous forces[16] or to an elevated LV pressure present at the time of atrial contraction.

In early diastole, a normal or decreased mitral velocity at atrial contractions and a short mitral deceleration time (Fig. 3). Left atrium was enlarged in 8 out of 10 patients of this age group.

In a small number of patients (i.e. 5), the mitral flow velocity pattern was normal. (Fig. 4).

Discussion:

Alterations in LV filling dynamics may be due to a variety of mechanisms, including changes in myocardial stiffness, impaired LV relaxation, changes in right ventricular loading conditions, pericardial constraints, viscoelastic considerations and nonuniformity among others[11]. It is well known that myocardial ischaemia can lead to impairment in systolic function, with regional wall motion abnormalities resulting decreased ejection fraction and increased ventricular volumes[13]. The impacts of acute ischaemia are further reflected by impairment in early LV filling after transient coronary occlusion by angioplasty[4]. The patients in group I, who were either asymptomatic or were in functional class I (NYHA) exhibited E/A reversal pattern, which has been named "Pattern I" by Appleton et al[12]. Most of these patients had extensive anterior/anterolateral infarction. Since we did not have the facilities for measuring the haemodynamic parameters so the correlation of doppler pattern was only clinical.

It has been demonstrated by Appleton et al that such patients had lower pulmonary wedge pressure, on cardiac catheterization. This pattern appears to occur when there is impaired LV relaxation. With less filling in early diastole (deflected as a small "E" wave on doppler pattern of LVIF), the per cent of left ventricular filling with atrial contraction would likely be increased as a compensatory mechanism, perhaps aided by atrial hypertrophy or enhanced. In this study, a small number of patients had relatively normal appearing mitral flow velocity pattern (although may had extensive infarct as determined by ECG and serum enzyme levels). In previous studies[14,15] most workers have correlated these Doppler patterns with data obtained on cardiac catheterization and they found a moderate increase in pulmonary wedge pressure in patients with, "normalized" pattern. They believed that the modest increase in left atrial pressure in these patients "masked" relaxation abnormality by "normalizing" the early diastolic transmitted pressure gradient. In such patients analysis of pulmonary venous flow (PVF) may be of help in differentiating normal from the pseudonormal LVIF pattern. These patients are found to have a big "A" wave, as compared to the normal PVF[16].

Conclusion:

The conclusion of the study was that LV filling is impaired during acute infarction with the predominant impairment in early diastole. Careful analysis of LVIF pattern may give us some clue about the filling pressure and this non-invasive assessment of haemodynamic is extremely useful in a place like our hospital where we lack the facility of cardiac catheterization.

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