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Assessment of Right Ventricular Function in St Segment Elevation Anterior wall Myocardial Infarction by Tissue Doppler Imaging

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Abstract
Objective: Strain and strain rate imaging is currently the most popular echocardiographic technique that reveals subclinical myocardial damage. There are currently no available data on this imaging method with regard to assessing right ventricular involvement in Anterior wall myocardial infarction after reperfusion therapy. Therefore, we aimed to evaluate right ventricular regional functions using a derived strain and strain rate imaging tissue Doppler method in patients who were successfully treated after revascularization therapy at the end of 60-72 hours(at discharge) by Tissue Doppler Imaging.
Methods: Clinical findings on admission were documented, along with biochemical tests. 12 lead ECG was done at admission, after 90 minutes and at 60-72 hours which included right sided leads. Echocardiography was performed at admission and after revascularization at 60-72 hours, in left lateral decubitus position with parasternal and apical views by conventional echocardiography along with TDI of RV and LV walls. Coronary angiogram findings were recorded whenever it was done. There were 35 subjects in AWMI group and 20 subjects in control group. In AWMI group 28 were male and 7 were female . In control group 14 were male and 6 were female.
Results: The RV systolic function in our study was well quantified by using the tissue velocities at the tricuspid annulus and was found to be significantly decreased in AWMI group compared to control. The diastolic velocities and the ratio E' /A' were also decreased significantly in AWMI group.
Conclusion: RV systolic and diastolic functions are affected in AWMI patients and is well quantifiable by TDI.TDI is a simple, quick, and inexpensive bedside tool and can be used to assess RV function. RV systolic function improves as early as within 72 hours with early reperfusion procedures and is related to LV systolic function. RV functional recovery is best related to LV systolic function improvement, suggesting that septal contraction alone has an important role in this setting.

I. Introduction

ST segment elevation myocardial infarction (STEMI) continues to be a major public health problem in the industrialized world and is an increasingly important problem in developing countries [1].If a significant quantity of myocardium undergoes ischemic injury the left ventricle (LV) pump function becomes depressed and end systolic volume increases.[2] The degree to which LV end systolic volume, a widely used parameter in echocardiography, increases is perhaps the most powerful hemodynamic predictor of mortality following STEMI.[3] On the other hand assessment of right ventricle (RV) function has always been a challenge in view of its complex geometry and this is more so in acute myocardial infarction (AMI).Most research on RV function after AMI involving various walls has concentrated on the prognostic role of RV dysfunction on the long term outcome.The extent of involvement of RV in anterior wall myocardial infarction(AWMI) affecting LV function would be all the more important as it involves a larger area of myocardium with higher adverse events and outcomes. Though conventional echocardiography fulfills the criteria of a simple bedside tool,there are limitations to image the partly retrosternal RV and poor demarcation of the heavily trabeculated RV endocardial borders[4]. But recently a new means of quantitatively assessing ventricular function,with tissue doppler imaging (TDI) has become available. TDI allows quantification of regional myocardial velocities, by colour TDI technique[5]. Very few studies have been done on RV function in anterior wall MI with TDI and hence this study was taken to quantify the RV function at initial presentation and after reperfusion therapy.

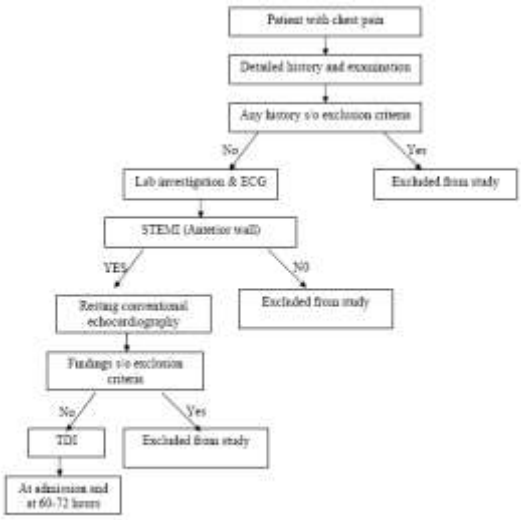
II. Aim Of The Study

1) This study is undertaken to assess the RV function quantitatively in patients presenting with ST segment elevation anterior wall myocardial infarction with a newer echocardiographic modality TDI.
2) Assessment of RV function after reperfusion therapy at the end of 60 hours(at discharge) by TDI.

III. Materials And Methods

Study design: Prospective Study, to compare two groups
Group A – ST elevation Anterior wall MI; Group B – Controls
Study population: Patients admitted to Institute with ST elevation acute anterior wall myocardial infarction were recruited to the study.
Inclusion Criteria: Patients presenting with ST elevation anterior wall myocardial infarction who meet the WHO/ACC criteria[9] for ST elevation myocardial infarction were consecutively recruited to the study after due consent.
Exclusion Criteria: Inferior Wall Myocardial Infarction, LV ejection fraction < 40% Non ST elevation MI, Sustained atrial fibrillation, Significant valvular heart disease (more than mild), Congenital heart disease, Cardiomyopathies, Severe systemic illness, Significant pericardial disease, Pregnancy, Anemia (Hb<10 gm %) Pulmonary Embolism.Pulmonary Hypertension, Patients on Pacemakers.

Algorithm Of The Study



Statistical Analysis

The following methods of statistical analysis have been used in this study. The results for each parameter averaged (mean + standard deviation) for continuous data and are presented in Tables and Figures.
1) The student 't' test was used to determine whether there was a statistical difference between control and study groups in the parameters measured.
Student's t test is as follows:

$$t=\frac{\bar{x}_1-\bar{x}_2}{s\sqrt{\frac{1}{n_1}+\frac{1}{n_2}}} \sim\sim\sim t_{n1+n2-2} \quad \text{Where } s^2=\frac{(n_1-1)s_1^{\,2}+(n_2-1)s_2^{\,2}}{(n_1+n_2-2)}$$

In the above test the "p" value of less than 0.05 was accepted as indicating statistical significance. Data analysis was carried out using Statistical Package for Social Science (SPSS) package.

IV. Results

There were 35 subjects in AWMI group and 20 subjects in control group. In AWMI group 28 were male and 7 were female . In control group 14 were male and 6 were female. Mean age(years) of subjects in AWMI group was 52.6±12.5 and in control group it was 44±8.1 (P=0.007) while that of BMI (kg/ m2) was 25.4±2.59 v/s 25.46±2.7 in AWMI group and controls.(Table 1) In AWMI group 11.4% (4) were having DM, 11.4% (4) were having hypertension and 25.7% (9) were having both DM and hypertension. 51.4% (18) did not

have any co-morbidities. 42.9% (15) had history of ethanol and tobacco use and 57.1% (20) without in AWMI group.

Biochemical analysis showed hemoglobin (gm%) within normal range, with no significant changes. The fasting glucose (mg/dl) was higher in AWMI group 155.29±82.09 versus 91.57±20.97 (P=0.001) (Refer Table 1). Serum cholesterol (mg/dl) and triglycerides (mg/dl)were higher in AWMI group in comparision to controls 204±97 v/s 189.81±31.17 (P=0.238) and 190.97±100.5, v/s 163.29±45.92 (P=0.241) and were not significant while Serum LDL levels (mg/dl) were significantly higher in AWMI group compared to controls.136.43±38.6 v/s 107.53±14.5(P=0.002)(Refer Table 1)

The average time interval between the chest pain and arrival to hospital was ≤4 hrs in the AWMI group.

Demographic & Biochemical Data:

	AWMI	**CONTROLS**	**'p' value**
Age (years)	52.63 ± 12.59	44± 8.08	<0.007
BMI (kg/m^2)	25.40 ± 2.59	25.46 ± 2.71	0.939
Hb (gm%)	14.79 ± 2.24	13.84 ± 1.47	0.092
S.Creatinine (mg/dl)	1.15 ± 0.23	1.00 ± 0.20	0.026
Sodium (meq/L)	138.40±4.32	138.81±2.13	0.687
Potassium (meq/L)	4.07±0.54	4.31 ±0.42	0.092
FBS (mg/dl)	155.29±82.09	91.57 ± 20.97	<0.001
PPBS (mg/dl)	195.31±75.64	133.14±19.46	<0.001
S. Cholesterol (mg/dl)	204.97±52.80	189.81±31.17	0.238
HDL (mg/dl)	35.43±7.28	37.86 ± 11.19	0.329
LDL (mg/dl)	136.43±38.60	107.53±14.59	<0.002
S. Triglycerides (mg/dl)	190.97±100.57	163.29±45.92	0.241

Table 1: Demographic and biochemical data.

Awmi: Anterior wall myocardial infarction, BMI: Body mass index, Hb: Haemoglobin, FBS: Fasting blood sugar, PPBS: Post prandial blood sugar, HDL: High density lipoprotein, LDL: Low density lipoprotein, S:serum; P<0.05 is significant

	AWMI	**CONTROLS**	**p value**
LVID d (mm)	42.83 ± 5.28	44.10 ± 4.47	0.363
IVS d (mm)	12.31 ± 1.84	10.20 ± 1.00	<0.0001
LVPWD d (mm)	11.44 ± 1.78	8.14 ± 1.01	<0.0001
EDV (mm)	82.03 ± 27.61	62.50 ±8.38	<0.003
ESV (mm)	40.66 ±18.62	31.50 ± 8.10	0.042
LVEF (%)	44.91 ± 2.46	64.20 ± 3.88	<0.0001
LV Mass (gm)	175.59 ±51.28	138.70 ± 26.77	<0.004
LV-E (cm/s)	58.82 ± 21.85	81.45 ± 12.29	<0.0001
LV-A (cm/s)	62.41 ± 24.71	62.55 ± 13.14	0.982
LV-E/A	0.942 ± 0.88	1.35 ± 0.32	<0.001
FS (%)	22.60 ± 2.63	34.90 ± 2.90	<0.0001
LV-DT (ms)	252.47±54.14	188.60 ±1.60	<0.011
LV septal – PSV (cm/s)	5.26 ± 1.26	11.50 ± 1.60	<0.0001
LV E/E'	11.45 ± 6.25	11.47 ± 3.61	0.99

Table 2: Conventional Echocardiographic and Tissue Doppler imaging data of Left ventricle

LVIDd: Left ventricular internal diameter in diastole, IVSd: Interventricular septum in diastole, LVPWD d: Left ventricular posterior wall diameter in diastole, LVEF: Left ventricular ejection fraction, LV Mass: Left ventricular mass, LV-DT: Left ventricular deceleration time, LV-E: Left ventricle diastolic early filling velocity, LV-A: Left ventricle diastolic late filling velocity, LV-E': Early diastolic velocity of the mitral septal annulus,PSV: Peak systolic velocity, FS: Fractional shortening,EDV-End diastolic volume,ESV-End systolic volume, E/ E': Early diastolic filling velocity by Pulsed wave Doppler to Early diastolic velocity by Tissue Doppler Imaging, ***LV:*** *Left ventricle,AWMI:Anterior wall myocardial infarction*
P<0.05 is significant

	AWMI	CONTROLS	p value
RV TAPSE (mm)	16.97±2.05	20.90±2.04	<0.0001
RV-E (cm/s)	42.68±11.28	40.35±8.74	0.429
RV-A (cm/s)	45.58±11.85	38.00±16.16	0.053
RV-DT (ms)	188.12±60.95	162.65±37.83	0.098
RV-E/A	0.95±0.24	1.16 ±0.30	<0.008
RV-PSV (cm/s)	10.66±1.57	12.25±1.33	<0.0001
RV- E/E'	6.11±3.20	5.09±2.08	0.20

Table 3: Conventional Echocardiographic and Tissue Doppler imaging data of Right ventricle

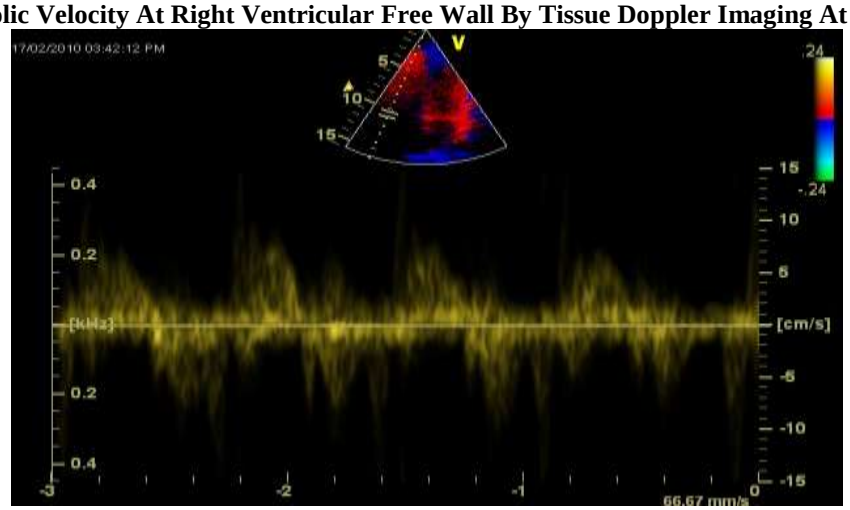
RVTAPSE: Right ventricle tricuspid annular plane systolic excursion, RV-E: Right ventricular diastolic early filling velocity, RV-A: Right ventricular diastolic late filling velocity,RV-DT: Right ventricular deceleration time, RV-PSV: Right ventricular tricuspid annular peak systolic velocity, RV-E': Early diastolic velocity of the tricuspid annulus, E/ E': Early diastolic filling velocity by Pulsed wave Doppler to Early diastolic velocity by Tissue Doppler Imaging, RV: Right ventricle. P<0.05 is significant

	Pre treatment AWMI (At admission)	Post treatment AWMI (After 60 hours)	p value
RVTAPSE (mm)	16.97 ± 2.05	18.69 ± 2.47	<0.002
RV-E (cm/s)	42.68 ± 11.28	44.11 ± 6.88	0.525
RV-A (cm/s)	45.58 ±11.85	46.64 ± 10.50	0.698
RV-DT (ms)	188.12 ± 60.95	193.65 ± 66.04	0.721
RV-E/A	0.95 ±0.24	0.98 ± 0.21	0.626
RV-E' (cm/s)	7.77 ± 2.18	9.06 ± 1.87	<0.010
RV-A' (cm/s)	11.91 ± 2.41	12.23 ± 2.00	0.556
RV-PSV (cm/s)	10.66 ± 1.57	11.69 ± 1.64	<0.009
RV-E'/A'	0.67 ±0.19	0.75 ± 0.16	0.053
RV-E/E'	6.11 ± 3.20	5.15 ± 1.76	0.125
LV-septal PSV (cm/s)	5.26 ± 1.26	6.0 ± 1.84	0.054
LV-E/E'	11.48 ± 6.25	12.12 ± 4.93	0.620

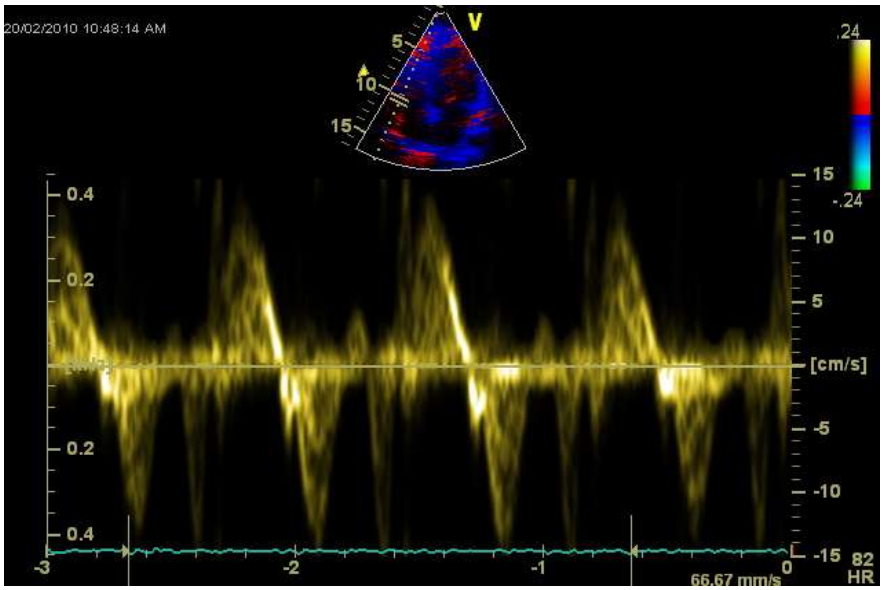
Table 4: Conventional echocardiographic and Tissue Doppler imaging data-Pre and post treatment

RVTAPSE: Right ventricle tricuspid annular plane systolic excursion, RV-E: Right ventricular diastolic early filling velocity, RV-A: Right ventricular diastolic late filling velocity, RV-DT: Right ventricular deceleration time, RV-E': Early diastolic velocity of the tricuspid annulus, RV-A': Late diastolic velocity of the tricuspid annulus, RV-PSV: Right ventricle tricuspid annular peak systolic velocity, LV-PSV: Left ventricular septal peak systolic velocity,LV:Left ventricle, RV:Right ventricle,E/ E':Early diastolic filling velocity by Pulsed wave Doppler to Early diastolic velocity by Tissue Doppler Imaging, E'/A':Early diastolic filling velocity to late early diastolic filling velocity by TDI, AWMI:Anterior wall myocardial infarction. P<0.05 is significant

Peak Systolic Velocity At Right Ventricular Free Wall By Tissue Doppler Imaging At Admission



Peak Systolic Velocity At Right Ventricular Free Wall By Tissue Doppler Imaging After Reperfusion



V. Discussion

Our study had 35 AWMI and 20 controls, AWMI group had higher fasting and post prandial blood sugar levels, serum cholesterol and LDL levels. LV systolic function as reflected by LVEF by conventional echocardiography was significantly reduced in AWMI compared to controls subjects.AWMI group also had significant diastolic dysfunction as evident by low E and E/A ratio.RV systolic function by TAPSE and TDI were also reduced significantly in AWMI group compared to control subjects. This impairment of RV systolic function was compared in the same AWMI group at admission and after reperfusion at 60 hours and was found to have improved significantly as evidenced by TAPSE and TDI.The RV diastolic dysfunction as assessed by pulse wave Doppler and diastolic tissue velocities by TDI also showed improvement after reperfusion though statistically not significant. In this study we have shown that AWMI affects right ventricular systolic function and is influenced by early reperfusion.

Tissue Doppler Imaging Of Right Ventricle

Assessment of RV function by TAPSE has been well correlated with radionuclide angiography and appears reproducible . [19]TDI is an ultrasound technique that provides assessment of contracting myocardium by measuring endocardial velocities. [24]Typically, TDI recording is composed of a systolic (Sa) wave an early diastolic wave E ' and an atrial wave A' . [24,25] When TDI is performed on tricuspid annulus it allows evaluation of the RV systolic and diastolic functions.[22,26] A few reports demonstrated that the systolic myocardial velocity of the tricuspid annulus is a sensitive indicator of global RV contractility, which is decreased in patients with RV dysfunction compared with normal subjects.[27,28] TDI has the potential to become an alternative tool in the assessment of both systolic and diastolic RV function and has demonstrated good reproducibility in measuring tricuspid annular velocities.[29] This corresponds well to our results, especially for the peak systolic velocity, which appeared to be the most clinically useful parameter in quantifying the RV function before and after reperfusion and is well correlated with the earlier studies. Systolic myocardial velocity derived from the tricuspid annulus has been shown to have strong correlation with +dP/d*t* for the RV, implying that the systolic myocardial velocity provides accurate information on RV contractility. [30] In our study, there was significant difference in tricuspid annulus peak systolic myocardial velocities between patients with AWMI and healthy subjects.

RV Systolic and Diastolic function

Most research on right ventricular (RV) function after acute myocardial infarction (AMI) has concentrated on the prognostic role of RV dysfunction in the long term outcome. This was mainly determined by assessing RV function in the acute phase of myocardial infarction and by clinical follow up thereafter.[31, 32]Our study mainly assessed the RV function in the acute phase of MI and supports the previous data suggesting that RV function using TDI derived tricuspid annulus motion provides functional information on longitudinally oriented RV myocardial fibers. [33] The evaluation of RV systolic function is useful in a variety of clinical situations .[31] The accurate and most often applied methods -radionuclide ventriculography and MRI [32] are time consuming and expensive methods and practically difficult in the setting of acute MI.

Pulsed TDI is a unique method of measuring systolic and diastolic velocities of motion of the tricuspid annulus. Very few studies have assessed the RV function using this modality. [17]

Meluzin et al [21] have well documented TDI as simple, quick and inexpensive in assessing RV function. The RV systolic function in our study was well quantified by using the tissue velocities at the tricuspid annulus and was found to be significantly decreased in AWMI group compared to control.The diastolic velocities and the ratio E' /A' were also decreased significantly in AWMI group. Similarly the RV systolic function and the diastolic function were also quantified using TDI at 60 hours after the reperfusion either by primary angioplasty or thrombolysis and was found to have improved significantly, although the improvement in diastolic function was not statistically significant.Factors that influence right ventricular function in coronary artery disease like right ventricular ischemia, septal shifting and RV afterload , are reported to be complex.[34] Some reports showed that the septal involvement in patients with acute myocardial ischemia was major determinant of the RV function.[35,36] But some investigators claim that RV function were primarily related to pulmonary hypertension associated with LV dysfunction. [37,38]

their necropsy series Isner et al [39] emphasized the extent of septal infarction as a principal determinant of right ventricular decompensation. Nakamura et al [40] showed that the presence or absence of IVS involvement was a 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 [41] showed that IVS ischemia induced deterioration in RV Function In our study only four patients were in killip class III and none of them had pulmonary hypertension and the influence of increase in RV afterload secondary to LV systolic dysfunction was nil. However the decrease in the septal contraction as evidenced by decreased peak systolic velocities at basal septum by TDI and a significant improvement after early reperfusion leading to improvement in RV function in these patients supports the theory that RV performance is dependent on LV septal contractile contributions transmitted through systolic ventricular interaction [42,43]and is similar to earlier studies quoted ,[39,40]suggesting that septal contraction may be important for the maintenance of RV function in patients after infarction with impaired LV function. Early reperfusion facilitates recovery of RV function and minimizes the extent of infarction even after prolonged ischaemia.[44] These findings also suggests RV function correlates significantly with LV function as shown by Moller and colleagues in their study.[45] Although the Interventricular Septum(IVS) has long been regarded as a functional part of the LV, it has lately been shown that the IVS actually contributes to the systolic function of both ventricles and thereby improving both RV and LV functions as in our study which supports these findings. An experimental study showed that RV function was impaired as much with septal ligation as with right coronary artery ligation, [46] showing that ischemic injury to either the IVS or the RV free wall alters RV function and suggest that both regions play a significant part in maintaining RV performance. The important role of the IVS in RV performance was later confirmed in patients by means of biventricular angiography, with thickening of the IVS being shown to contribute to the systolic contraction of both ventricles. [47]

VI. Conclusions

RV systolic and diastolic functions are affected in AWMI patients and is well quantifiable by TDI. RV systolic function improves as early as within 72 hours with early reperfusion procedures and is related to LV systolic function. TDI is a simple, quick, and inexpensive bedside tool and can be used to assess RV function.RV functional recovery is best related to LV systolic function improvement, suggesting that septal contraction alone has an important role in this setting.

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