Abstract:
Background: The aim of this study was to assess left ventricular dyssynchrony after acute ST elevated myocardial infarction (STEMI) in patients with normal QRS duration. Real time 3D echocardiography (RT3DE) with triplane tissue synchronization imaging (TSI) used to identify segmental left ventricular systolic velocity in ejection phase to evaluate LV dyssynchrony in patients with STEMI and the findings were compared with control.

Materials and methods: RT3DE with triplane TSI was performed within 4 days of AMI after thrombolysis or primary PCI in 31 patients and compared with 31 age-matched controls. Regional myocardial velocities were assessed in 12 segments in ejection phase, and the corresponding time to peak systolic velocity (Ts) was measured. To assess LV dyssynchrony Ts-4, Ts-6, Ts-SD-6, Ts-12 and Ts-SD-12 were computed by offline dedicated software semi-automatically.

Results: The dyssynchrony parameters were significantly prolonged in patients with AMI. Among the dyssynchrony parameters TS-SD-12 was better indicator of LV dyssynchrony. The Ts-SD-12 was significantly prolonged in the STEMI group when compared with controls. In patients with acute STEMI mean Ts-SD-12 was 43.2±19.1 milliseconds whereas in control group it was 23.0 ±6.5 milliseconds (p<0.05). The Ts-SD-12 was prolonged in patients with Anterior than Inferior STEMI as follows respectively 45.9± 17.6 and 40.0± 21 milliseconds.

Conclusions: Triplane TSI by RT3DE is useful in evaluating LV dyssynchrony in patients with acute STEMI and even in those with normal QRS duration there is significant left ventricular dyssynchrony early after STEMI.

Key words: STEMI, LV dyssynchrony, RT3DE, time to peak systolic velocity in ejection phase (Ts).

Introduction:
In normal heart, the left ventricle (LV) contracts and relaxes in a synchronous manner. Dyssynchrony refers to the uncoordinated mechanical contraction of the heart. Systolic dyssynchrony can be defined as uncoordinated timing of contraction in different segments of the myocardium. Conventionally dyssynchrony is related with heart failure, cardiomyopathy, conduction defect and bundle branch block and is used as predictor of cardiac resynchronisation therapy (CRT) response. But recently LV dyssynchrony is assessed in patients with AMI as a predictor of LV remodeling and is a non-invasive indirect procedure for detection of infarct size. Dyssynchrony early after AMI causes severe LV dysfunction as a consequence of early cardiac remodeling, elevation of filling pressure, reduced diastolic filling, reduced LV stroke volume and reduced CO.

Acute myocardial infarction causes remodeling of LV due to complex alterations in ventricular architecture involving both infarcted and noninfarcted zones. Left ventricular remodeling develops in considerable proportion of patients after AMI despite successful treatment with thrombolysis and primary PCI and even in the presence
of patent infarct related artery and associated with increased cardiovascular events and mortality.\(^5\) Therefore, early identification of patients at risk for developing LV remodeling after AMI has important prognostic and therapeutic implications. In addition, it was demonstrated that dysynchrony associated with AMI was mainly determined by the infarct size.\(^6\)

Advances in echocardiographic techniques, i.e. tissue Doppler echocardiography, speckle-tracking echocardiography, and real-time 3-dimensional echocardiography have demonstrated an impaired left ventricular synchronicity in patients with acute myocardial infarction.\(^7\) Myocardial Tissue Doppler Imaging (TDI) is an ultrasound technique that provides assessment of the contracting myocardium by measuring myocardial velocities.\(^8\)

Among the echocardiographic tools used to evaluate mechanical dysynchrony, TDI analysis occupies a prominent position and the use of colour-coded TDI for dysynchrony assessment is presently advocated.\(^9\) The major advantage of colour-coded TDI is the simultaneous comparison of various LV segments. Tissue Synchronization Imaging (TSI) can be used to assess intra-ventricular dysynchrony in the longitudinal plane (apical views). Using a triplane imaging probe (3D probe) from the apical window, simultaneous grey-scale images can be acquired in apical four, two and three chamber views. Simply by activating colour-coded TDI during the triplane acquisition, information on the myocardial velocities of various LV segments in these three planes can be obtained during the same heartbeat. TSI can also be applied to a triplane dataset. The TSI tool will automatically calculate the time to peak myocardial systolic velocities in several LV segments and generate a report and a colour scheme with the different activation times of those LV segments.\(^10\)

RT3DE provides optimal information of LV volumes, functions and sphericity.\(^11\) Although role of LV dysynchrony as a predictor of remodeling after AMI is well established but its relation with traditional echocardiographic prognostic parameter of LV function and other variables remain imprecise. So this study was designed to compare the dysynchrony parameters between case and control groups and to assess correlation with LV volumes and functions.

Materials and methods:
This case control study was done during the period of June 2012 to May 2013 at National Heart Foundation Hospital & Research Institute (NHF&RI). In group I, 31 patients with Acute STEMI with normal QRS duration admitted in CCU of NHF&RI treated either by thrombolysis or primary PCI were included and 31 age-matched healthy controls were enrolled in group II. Samples were collected on the basis of inclusion and exclusion criteria without any randomization. Patients with previous MI, wide QRS complex (>120ms), heart failure, history of previous PTCA or CABG, Pacemaker, CRT or ICD implantation and with poor echo window with inadequate Doppler signal and suboptimal two dimensional images were excluded.

Meticulous history was taken and detailed clinical examination performed in both groups. Demographic data and risk factors were recorded, 12 lead ECG and blood biochemistry done. Echocardiography was done within four day of acute STEMI.

Echocardiography:
Comprehensive echocardiography was performed in each patient with standard echocardiography machine Vivid E9 (Vingmed- General Electric, Horten, Norway).

2D M-mode
All case and controls were imaged in the left lateral decubitus position. Standard images were obtained using a 3.5-MHz transducer, at a depth of 16 cm in the parasternal long axis (PLAX) and short-axis (SAX); apical four chamber (A4C), apical two chamber (A2C) and apical long axis (ALAX) views. The LV was divided into 17 segments including true apex for quantifying wall motion score index (WMSI). A semi-quantitative scoring system e.g. 1-normal, 2-hypokinesia, 3-akinesia and 4-dyskinesia was used to analyze both case and control groups. Global WMSI was calculated by the standard formula: sum of the segment score divided by number of segment scored.\(^12\)

Real time 3 Dimensional Echocardiography (RT3DE)
RT3DE allows intra-ventricular volumes and dyssynchrony to be evaluated by analyzing LV wall motion in multiple apical planes during the same cardiac cycle and provide better spatial resolution than a single plane.

RT3DE was performed at the same time to assess global LV systolic function and LV dyssynchrony with 3D phased array transducer (2.5 MHz). Apical full volume 3D data sets were acquired in harmonic mode, integrating during a brief breath-hold (3-4 Seconds), R wave-triggered sub-volumes into a larger pyramidal volume (90° by 90°) with a complete capture of the LV.\(^13\) Dedicated algorithm was used by specific software to calculate LV end-diastolic volume (EDV), LV end-systolic volume (ESV)
and left ventricular ejection fraction (LVEF). A semi-
automated method by software Echopac BT12, GE
Vingmed Ultrasound for the detection of the apical 4-
chamber view and the 60° and 120° incremental views
and for the tracing of the endocardial border in the entire
3D dataset including LV trabeculations and papillary
muscles within the LV volume was used. Subsequently,
a final reconstruction of the LV model was generated
and LV volumes and LVEF were obtained. This software
also enables tissue tracking by colour coding the
displacement in the myocardium and allowed quantitative
analysis of multiple tissue velocity traces.14

To measure the time to peak systolic velocity in the
ejection phase (Ts) by RT3DE based TSI of individual
segments, the following rules were used:

• Aortic valve opening and closure was marked and
superimposed on TDI tracings to guide the
identification of the ejection phase.
• Time was measured from the onset of the QRS
complex to the highest systolic peak during the
ejection phase (between aortic valve opening and
closure).
• If there were multiple peaks in the ejection phase, the
highest peak was used.
• If the segment had only a negative peak in the ejection
phase or the velocity was noisy with very low and
inconsistent velocities, those particular segments
were neglected, and
• Ts were not measured on the isovolumic contraction
phase or isovolumic relaxation phase or during
postsystolic shortening.1

The RT3DE approach acquires three standard apical
views during the same heart beating (triplane). The
images were acquired in TSI modality, thus allowing 3-D
“surface rendering” visualization of the extent of
dyssynchrony. The method is fast (a few seconds) and
easy to perform.15 During acquiring an apical 4C view,
the multi-dimension button was pressed followed by the
tri-plane button. A full sector of the desired imaging plane
was acquired for simultaneous visualization and to
compare all the walls within the left ventricle. Accurate
TSI analysis achieved with frame rates around 100fps or
higher. TVI was acquired by entering TVI mode. Optimize
2D gain required for a clean chamber that is free of noise
by simply depressing the gain button and ECG trace,
free of noise and with a consistent heart rate was used
and image was acquired in cine loop. Then TSI mode
selected and the image was colorized according to the
time-to positive peak systolic velocity. Regions reaching
peak velocity early in systole were marked in green. Regions reaching peak velocity late in systole or in
diastole were marked yellowish to red. The loop was
freezed in a late diastolic frame and a tri-plane TSI image
displayed. For measurement TSI myocardium surface
selected by tracking curser at basal and mid LV in 4
opposing segments of myocardium starting on the left
side of the A4C view and ending on right side. Process
automatically selects segments on A2C and ALAX views
of triplane image. TSI software automatically calculates
the time to peak myocardial systolic velocities in 12 LV
segments and generate a report and a colour scheme
with the different activation times of these LV segments.
The colour schemes can be projected on a surface
rendered endocardial volume and a bulls-eye view TSI
report which was automatically generated with
parameters of LV dyssynchrony.16

For detection of LV dyssynchrony the following
parameters with cut off values were used: 17

• Opposing segment delay (Ts-4) >60 ms
• Maximal difference in Ts between any of the 6 basal
LV segments (Ts-6) >110 ms
• Standard deviation of Ts of the 6 basal LV segments
(Ts-SD-6) >34.5 ms
• Maximal difference in Ts of the 12 LV segments (Ts-
12) >100 ms
• Standard deviation of Ts of the 12 LV segments (Ts-
SD-12) >34.4ms

Statistical Methods:
The collected data were checked and coded manually
and then entered into a computer database. All analyses
were performed using the SPSS (SPSS for Windows
17.0) software package. Continuous variables were
presented as mean ± standard deviation. Categorical
variables were presented as the percentage. Chi-square
test, Fisher exact test and continuity correction were used
for categorical variables and unpaired t-test was used
for continuous variables if appropriate. Pearson’s and
Spearman correlation exponents were used to force of
relationship between continuous variables. A value of p <
0.05 was considered statistically significant.

Results:
The mean age of group I patients was 48.6±11.6 years
and 45.6±10.4 years for group II patients (Table I). The
commonest age group of study patients was 41-50 years
in both the groups (29 % and 30 % in group I and group
II respectively). Male patients were predominant in both groups; there were 6.5 percent female in group I and 6.7 percent female in group II. Smoking, hypertension, obesity and dyslipidaemia were the most common risk factors in both groups. Family history of IHD and diabetes were significant risk factors in group I (Table II). In 87.1% (n=27) patients in Group I was treated by thrombolysis with Streptokinase and 12.9% (n=4) by Primary PCI.

### Table-I

**Age distribution of the study population (n=62)**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Group I (Case: n=31)</th>
<th>Group II (control: n=31)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;30</td>
<td>2 6.5</td>
<td>3 10.0</td>
<td></td>
</tr>
<tr>
<td>31-40</td>
<td>7 22.6</td>
<td>7 23.3</td>
<td></td>
</tr>
<tr>
<td>41-50</td>
<td>9 29.0</td>
<td>10 30.0</td>
<td></td>
</tr>
<tr>
<td>51-60</td>
<td>8 25.8</td>
<td>8 26.7</td>
<td></td>
</tr>
<tr>
<td>&gt;60</td>
<td>5 16.1</td>
<td>3 10.0</td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>48.6±11.6</td>
<td>45.6±10.4</td>
<td>0.286NS</td>
</tr>
</tbody>
</table>

### Table-II

**Risk factors of the study population (n=62)**

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Group I (n=31)</th>
<th>Group II (n=31)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td>17 54.8</td>
<td>16 53.3</td>
<td>0.309NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>20 64.5</td>
<td>19 63.3</td>
<td>0.923NS</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>22 71.0</td>
<td>17 56.7</td>
<td>0.249NS</td>
</tr>
<tr>
<td>Family H/O IHD</td>
<td>16 51.6</td>
<td>9 30.0</td>
<td>0.021S</td>
</tr>
<tr>
<td>Diabetes</td>
<td>14 45.2</td>
<td>7 23.3</td>
<td>0.034S</td>
</tr>
<tr>
<td>Obese &amp; over weight</td>
<td>14 45.1</td>
<td>16 53.2</td>
<td>0.644NS</td>
</tr>
</tbody>
</table>

Among the patients with myocardial infarction 17 (54.8%) suffered from AMI anterior and 14(45.2%) from AMI inferior. Baseline investigation revels significantly high cardiac markers (Table III).

### Table-III

**Baseline clinical examination and investigation findings of study population (n=62)**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group I (n=31)</th>
<th>Group II (n=31)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>72.2±12.3</td>
<td>71.2±10.4</td>
<td>0.836NS</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>117.4±18.9</td>
<td>125.6±14.5</td>
<td>0.093NS</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>75.8±9.1</td>
<td>79.1±7.9</td>
<td>0.418NS</td>
</tr>
<tr>
<td>BMI</td>
<td>25.0±2.3</td>
<td>25.2±3.8</td>
<td>0.220NS</td>
</tr>
<tr>
<td>QRS duration</td>
<td>93.6±11.9</td>
<td>84.3±6.3</td>
<td>0.001S</td>
</tr>
<tr>
<td>RBS(mg/dl)</td>
<td>8.8±3.8</td>
<td>5.6±0.7</td>
<td>0.0001S</td>
</tr>
<tr>
<td>Serum Creatinine(mg/dl)</td>
<td>1.2±0.24</td>
<td>1.0±0.09</td>
<td>0.001S</td>
</tr>
<tr>
<td>CK MB (u/L)</td>
<td>227.3±125.6</td>
<td>28.4±8.2</td>
<td>0.0001S</td>
</tr>
<tr>
<td>Serum Troponin (ng/ml)</td>
<td>43.9±8.2</td>
<td>0.0±0.0</td>
<td>0.0001S</td>
</tr>
<tr>
<td>Total Cholesterol(mg/dl)</td>
<td>200.7±46.5</td>
<td>201.8±36.2</td>
<td>0.920NS</td>
</tr>
<tr>
<td>HDL(mg/dl)</td>
<td>39.7±9.1</td>
<td>39.4±6.4</td>
<td>0.071NS</td>
</tr>
<tr>
<td>LDL(mg/dl)</td>
<td>129.4±36.1</td>
<td>147.6±12.7</td>
<td>0.086NS</td>
</tr>
<tr>
<td>Triglycerides(mg/dl)</td>
<td>164.0±54.8</td>
<td>199.0±36.4</td>
<td>0.057NS</td>
</tr>
</tbody>
</table>

The patients with STEMI had reduced ejection fraction, increased wall motion score index, greater left ventricular volumes evident by measurement of RT3DE end diastolic and end systolic volumes than the control group (Table IV). End systolic volume and WMSI index was significantly higher in anterior MI than inferior MI (Table-V).

### Table-IV

**Distribution of the groups by RT3DE parameters (n=62)**

<table>
<thead>
<tr>
<th>RT3DE</th>
<th>Group I (n=31)</th>
<th>Group II (n=31)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDV (ml/m²)</td>
<td>87.0±16.2</td>
<td>78.2±13.6</td>
<td>0.542NS</td>
</tr>
<tr>
<td>ESV (ml/m²)</td>
<td>47.5±13.2</td>
<td>31.2±5.2</td>
<td>0.001S</td>
</tr>
<tr>
<td>EF (%)</td>
<td>45.2±6.2</td>
<td>60.1±1.4</td>
<td>0.0001S</td>
</tr>
<tr>
<td>WMSI</td>
<td>1.5±0.25</td>
<td>1.0±0.0</td>
<td>0.0001S</td>
</tr>
</tbody>
</table>

In the normal controls RT3D echocardiography derived TSI results revealed, there was no significant variance in time to peak myocardial systolic velocity (Ts) among LV segments. However, Ts of the patients with STEMI in group I was significantly prolonged when compared with controls in group II (Table VI). In contrast, in patients with anterior MI Ts-12 and Ts-Sd-12 parameters were high and in inferior MI Ts-4, Ts-6, Ts-SD-6 were higher (Table – VII). LV dyssynchrony was identified in 54.8% (n=17) by Ts=SD-12 in Group I and was better than other parameters (Table-VIII).

### Table-V

**Distribution of the patients in group I by RT3DE parameters (n=31)**

<table>
<thead>
<tr>
<th>RT3DE</th>
<th>AMI Anterior AMI (n=17)</th>
<th>Inferior (n=14)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDV (ml/m²)</td>
<td>91.0±19.1</td>
<td>82.2±10.7</td>
<td>0.123NS</td>
</tr>
<tr>
<td>ESV (ml/m²)</td>
<td>52.6±14.5</td>
<td>41.3±8.4</td>
<td>0.012S</td>
</tr>
<tr>
<td>EF (%)</td>
<td>42.7±6.2</td>
<td>48.4±4.8</td>
<td>0.08NS</td>
</tr>
<tr>
<td>WMSI</td>
<td>1.6±0.3</td>
<td>1.3±0.1</td>
<td>0.011S</td>
</tr>
</tbody>
</table>

There was significant positive correlation between Ts-SD-12 and WMSI (Pearson correlation =.478, p (2 - 0.007) but correlation with EDV, ESV and EF was not significant although both these values were significantly higher in patients with STEMI (Figure-3).
Table VI

**Distribution of the groups by RT3DE time to peak systolic velocity in ejection phase parameters (n=62)**

<table>
<thead>
<tr>
<th>Time to peak systolic velocity in ejection phase ( (T_s) )</th>
<th>Group I ( (n=31) )</th>
<th>Group II ( (n=31) )</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>( T_s-4 ) (Opposing segment delay)</td>
<td>55.8±47.4</td>
<td>23.2±18.8</td>
<td>0.001*</td>
</tr>
<tr>
<td>( T_s-6 ) (Maximum difference of ( T_s )-6 basal segments)</td>
<td>73.1±54.1</td>
<td>45.1±25.3</td>
<td>0.003*</td>
</tr>
<tr>
<td>( T_s-SD-6 ) (Standard deviation of ( T_s ) of 6 basal segments)</td>
<td>36.2±19.5</td>
<td>21.4±8.1</td>
<td>0.0001*</td>
</tr>
<tr>
<td>( T_s-12 ) (Maximum difference of ( T_s )-12 LV segments)</td>
<td>132.8±51.9</td>
<td>70.9±24.5</td>
<td>0.001*</td>
</tr>
<tr>
<td>( T_s-SD-12 ) (Standard deviation of ( T_s )-12 LV segments)</td>
<td>43.2±19.1</td>
<td>23.0±6.5</td>
<td>0.0001*</td>
</tr>
</tbody>
</table>

Table VII

**Distribution of the group I patients by RT3DE time to peak systolic velocity in ejection phase (n=31)**

<table>
<thead>
<tr>
<th>Time to peak systolic velocity in ejection phase ( (T_s) )</th>
<th>AMI Anterior ( (n=17) )</th>
<th>AMI Inferior ( (n=14) )</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>( T_s-4 ) (Opposing segment delay)</td>
<td>48.1±32.0</td>
<td>65.2±61.2</td>
<td>0.356*</td>
</tr>
<tr>
<td>( T_s-6 ) (Maximum difference of ( T_s )-6 basal segments)</td>
<td>56.5±39.2</td>
<td>64.2±41.2</td>
<td>0.292*</td>
</tr>
<tr>
<td>( T_s-SD-6 ) (Standard deviation of ( T_s ) of 6 basal segments)</td>
<td>35.4±15.3</td>
<td>37.3±24.3</td>
<td>0.798*</td>
</tr>
<tr>
<td>( T_s-12 ) (Maximum difference of ( T_s )-12 LV segments)</td>
<td>140.2±52.3</td>
<td>124.0±51.8</td>
<td>0.397*</td>
</tr>
<tr>
<td>( T_s-SD-12 ) (Standard deviation of ( T_s )-12 LV segments)</td>
<td>45.9±17.6</td>
<td>40.0±21.0</td>
<td>0.414*</td>
</tr>
</tbody>
</table>

Table VIII

**Distribution of LV Dyssynchrony in group I by time to peak systolic velocity in ejection phase \( - T_s \). (n=31)**

<table>
<thead>
<tr>
<th>( T_s )</th>
<th>LV Dyssynchrony</th>
<th>No LV Dyssynchrony</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>TS-4</td>
<td>13</td>
<td>41.9</td>
</tr>
<tr>
<td>Ts-6</td>
<td>14</td>
<td>45.2</td>
</tr>
<tr>
<td>Ts-SD-6</td>
<td>14</td>
<td>45.2</td>
</tr>
<tr>
<td>Ts-12</td>
<td>15</td>
<td>48.3</td>
</tr>
<tr>
<td>Ts-SD-12</td>
<td>17</td>
<td>54.8</td>
</tr>
</tbody>
</table>

Fig. 1: RT3DE: A. apical full volume 3D data, B. reconstructed 3D model with LVEDV, LVESV and LVEF (RT3DE – real time 3D echocardiography, LVEDV – LV end diastolic volume, LVESV – LV end systolic volume, LVEF – LV ejection fraction)
Fig.-2: RT3DE triplane TSI: a. Event timing (aortic valve opening and closure); b. full volume 3D data; c. TVI; d. TSI; e. bull's eye TSI report (TVI- tissue velocity imaging, TSI – tissue synchronization imaging)

Fig.-2: Scatter plots showing correlation of Ts-SD-12 with A. EDV, B. ESV, C. EF, D. WMSI (Ts-SD-12 – time to peak systolic velocity in ejection phase, EDV – end diastolic volume, ESV – end systolic volume, EF – ejection fraction, WMSI – wall motion score index) there was significant correlation between Ts-SD-12 and WMSI (Pearson correlation -0.478, p<0.007)
Discussion:
Mean age difference was not statistically significant. Nearly similar pattern of age distribution were reported in different studies in Bangladesh by Wahab and Alam. But there was difference in mean age with different studies done in abroad; Nucifora et al 57±11 and Ko SK et al 61±13 years. Most probably this was due to the late onset of atherosclerotic coronary artery disease in developed countries than that of a third world country population. In Bangladesh, the various studies showed, the female patients formed a small percentage. Kabiruzzaman 4% and Selim 11.5% female patients in their respective studies. Wahab and Alam showed that smoking was the commonest risk factor in their study population. Smoking, hypertension and family history of CAD were the commonest risk factors in the study done by Nuciforae et al.

Previously in a study by Zhang et al for the assessment of LV dysynchrony by 2D TSI, all the patients were treated with thrombolysis. In their study 24(53%) patients had anterior AMI and 21(47%) had inferior AMI. In another study by Mollema et al they recruited patients with AMI who underwent primary PCI. Delgado et al in their study by RT3DE echocardiography included patients with AMI after primary PCI. In this study patients treated either with thrombolysis or primary PCI were included.

The mean ejection fraction by RT3D echo was 45±6% in patients with acute STEMI, EDV 87±16ml, ESV 47±13 ml and WMSI 1.5±0.25. Zhang et al in their study found that Patients with acute MI had reduced LV systolic function, dilated LV end-systolic and end-diastolic dimensions and greater LV mass when compared with controls. Findings of the present study is consistent with the findings of Delgado et al by RT3DE in patients with AMI, where mean ejection fraction was 44±7%, mean end diastolic volume was 108±23 ml and end systolic volume was 60±15 ml. Mollema et al in their study showed baseline WMSI in acute myocardial infarction group was 1.5±0.23, which is consistent with this study.

Zhang et al in their study by 2D TSI echocardiography in AMI patients all Ts values were significantly prolonged than the normal healthy controls. They had used Ts-SD-12 segment delays to identify dysynchrony and the mean Ts-SD in AMI patients was 42.2±13.7 ms and in control group it was 18.0±7.0 ms. In anterior AMI it was 46.8±13.9 ms and in inferior AMI 34.6±8.5 ms. So these results were similar in the present study although the mean TS-SD in control group was higher but within reference level. Antoni ML et al in their study by speckle-tracking mean difference in activation time was 61±79 ms and 14% patients with STEMI showed significant dysynchrony.

In this study 54.8% acute STEMI patients had LV dyssynchrony identified by Ts-SD-12. Zhang et al Ts-SD of the MI group was significantly higher than the normal controls, and the prevalence of systolic asynchrony in the MI group was 69.8%, Yu CM found the standard deviation of Ts of 12 LV segments was the most powerful predictor of remodeling of LV.  Montazeri et al in their study found Ts-Sd-12 and Ts-SD-6 were more prevalent in patients with wide QRS duration then other predictors of dysynchrony. Turan B et al in their study using Ts-6 maximum delay as marker of dysynchrony found in 23% of STEMI patients had dysynchrony, Delgado et al in their study by RT3D echocardiography used SDI (systolic dyssynchrony index) as marker of dysynchrony and SDI was higher in 45% of patients with AMI.

There was significant correlation between Ts-SD-12 and WMSI (Pearson correlation -.478, p (2 tailed) -.007 but correlation with EDV, ESV and LVEF was not significant although both these values were significantly higher in patients with AMI. Mollama et al in their study had similar correlation with WMSI early after AMI but in their study EDV and ESV was significantly correlated with dysynchrony and these findings were termed as a predictor of severe LV dyssynchrony and late LV remodeling. In line of these findings LV dyssynchrony in acute STEMI was significantly associated with LV functions specially WMSI and related with changes in LV volumes.

Limitations:
Although the results of this study support the hypothesis, there were some limiting factors which might influence the results:

- Sample size was small.
- Disparity of gender.
- Difference in time of echocardiography after AMI
- Inter and intra observer variability was not done.

Conclusion:
The present study illustrates that LV systolic dyssynchrony is a relatively common feature in patients with acute STEMI. This occurs early in STEMI even in the absence of wide QRS complexes. In addition, patients with anterior myocardial infarction have more severe LV systolic dyssynchrony than those with inferior myocardial infarction.

Recommendation:
LV dyssynchrony soon after AMI may cause severe detrimental impact on LV performance and it may potentiate to the vicious cycle of progressive LV dysfunction. Larger studies including clinical end points and long term follow-up and angiographic correlations are required to examine the significance of LV dyssynchrony in acute STEMI.
References:


