ASSESSMENT OF RIGHT VENTRICULAR FUNCTION IN ACUTE ST-ELEVATION MYOCARDIAL INFARCTION BY ECHOCARDIOGRAPHY

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ABSTRACT

BACKGROUND
Right Ventricular (RV) function has been shown to be a major determinant of clinical outcome and consequently should be considered during clinical management and treatment. Thus, the need for diagnosis of RV dysfunction is evident.

MATERIALS AND METHODS
200 consecutive patients admitted with Acute ST-Elevation Myocardial Infarction to the ICCU of Osmania General Hospital, Hyderabad during the period of Jan. 2013 to Dec. 2013. All patients who presented with ST Elevation Myocardial Infarction for the first time within the 5 days of onset of symptoms, irrespective of age, gender and treatment status were included.

RESULTS
Incidence of RV dysfunction in study population by TAPSE< 1.5 cm was 44 (22%). 23 (29.1%) patients with Inferior wall MI had RV dysfunction and 21 (17.3%) patients with anterior wall MI had RV dysfunction. Out of 200 patients with acute myocardial infarction, 22% (44/200) had RV dysfunction. Mean age of patients in RV dysfunction group was 57.4 ± 11.3 years and of patients in no-RV dysfunction (Normal RV function) group was 52.3 ± 11.2 years (p=0.008). 15.9% (7/44) patients in RV dysfunction group had past history of Coronary artery disease compared to 5.8% (9/156) in normal RV function group (p=0.02).

CONCLUSION
Nearly one fourth of patients with Acute ST-Elevation Myocardial Infarction had right ventricular dysfunction. LV dysfunction contributes significantly to RV dysfunction in acute myocardial infarction more so in anterior wall MI.

KEYWORDS
Right Ventricular Function, Acute ST-Elevation Myocardial Infarction, Echocardiography, TAPSE, RV FAC.


BACKGROUND
Right Ventricular (RV) function may be impaired either by primary right-sided heart disease, or secondary to left-sided cardiomyopathy or valvular heart disease. In addition, it should be considered that RV dysfunction may affect left ventricular (LV) function, not only by limiting LV preload, but also by adverse systolic and diastolic interaction via the interventricular septum and the pericardium (ventricular interdependence). Moreover, RV function has been shown to be a major determinant of clinical outcome and consequently should be considered during clinical management and treatment. Thus, the need for diagnosis of RV dysfunction is evident.

MATERIALS AND METHODS
Study population: 200 consecutive patients admitted with Acute ST-Elevation Myocardial Infarction to the ICCU of Osmania General Hospital, Hyderabad during the period of Jan. 2013 to Dec. 2013.

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Study Design
Hospital-based descriptive study.

Inclusion Criteria
All patients who presented with ST Elevation Myocardial Infarction for the first time within the 5 days of onset of symptoms, irrespective of age, gender and treatment status.

Exclusion Criteria
Patients with past history of Q-wave myocardial Infarction, Chronic obstructive pulmonary disease, Pulmonary arterial hypertension, Chronic Rheumatic heart disease, Cardiomyopathies, Congenital heart diseases, Poor echo window for assessment of Right Ventricle and those unwilling for the study.

All patients were clinically evaluated. Detailed history noted from patients, attendants and from first clinical clerking from the documents available. Detailed general examination done. Vitals recorded. JVP evaluated, a thorough cardiovascular examination done. All the details were entered in a proforma.

ECGs recorded at admission with BPL Cardiart machine, a 12-lead single channel ECG, with additional leads of V3R, V4R in case of inferior wall MI, at 25 mm/second speed and at 10 mm/mV standardisation.

All the patients were subjected to two-dimensional echocardiography, M-mode and Doppler echocardiography within one day of admission. Echocardiographic examinations were performed on Philips iE33 machine with a system equipped with Doppler tissue imaging.
Assessment of Right Ventricular Function

Tricuspid Annular Plane Systolic Excursion (TAPSE)\[1,2\]

In real time, two-dimensional apical four chamber view M-mode cursor was placed through the junction of tricuspid valve plane and RV free wall in such a way that annulus moved along the M-mode cursor. The systolic displacement of tricuspid annulus towards the cardiac apex was recorded in M-mode. The total displacement was the vertical distance from the crest to trough of the leading edge obtained in M-mode due to the movement of annulus. The distance was measured using the calliper inbuilt in echo machine. Units were expressed in centimetres. A cut-off value of <1.5 centimetres was taken for identification of patients with RV dysfunction.

Right Ventricular Fractional Area Change (RV FAC)\[3,4\]

RV function was assessed quantitatively using echocardiographic analysis as percentage of change in cavity area from end diastole to end systole in apical four chamber view. End diastole was considered as the frame at the onset of R-wave from simultaneously recorded electrocardiogram on echo machine; end systole was regarded as smallest ventricular cavity before tricuspid valve opening. Using apical four chamber view, endocardial border of right ventricular free wall and septum were traced from base to apex.

Right Ventricular Fractional Area Change was Calculated by the Formula Given Below:

$$\text{END DIASTOLIC AREA} - \text{END SYSTOLIC AREA} \times 100$$

$$\text{END DIASTOLIC AREA}$$

Doppler Tissue Imaging for Assessment of Peak Systolic Velocity of Tricuspid Lateral Annulus (Sa)\[2,5\]

In two dimensional apical four chamber view, in a tissue Doppler imaging mode, sample volume placed on tricuspid annulus at the place of attachment of anterior leaflet of tricuspid valve. Ultrasound beam was placed parallel to the direction of tricuspid annular motion. Peak systolic annular velocity recorded at speed of 75 mm/s for subsequent analysis. The minimal gain was used to assure clear and well-defined pulse Doppler tissue imaging in left lateral decubitus position in end expiratory phase. Three consecutive cardiac cycles were measured for peak tissue velocity and mean was taken. For measuring peak tissue velocities calliper present in echo machine was used. Units expressed in centimetres per second (cm/s).

Statistical Methods

Data was analysed using SPSS 16.0 statistical package. All categorical variables were expressed as numbers and percentages and the significant differences were analysed by Chi-square test. All continuous variables were expressed in mean ± Standard deviation (SD) and the differences between the groups were analysed by Student t-test. The difference in the means in between multiple groups was analysed by analysis of variance (ANOVA). A value of p <0.05 was considered statistically significant.

RESULTS

200 consecutive patients with Acute ST-Elevation Myocardial Infarction were evaluated in this study. Among 200 patients, 157 (78.5%) were male. Mean age was 53.35 ± 11.48 years.

Presenting Complaints

195 (97.5%) patients presented with chest pain, 91 (45.5%) had shortness of breath, 31 (15.5%) had orthopnoea, 10 (5%) had syncope, 57 (28.5%) had palpitations, 57 (28.5%) had restlessness during presentation.

Location of MI

121 (60.5%) had anterior wall myocardial infarction (AWMI), 79 (39.5%) had inferior wall myocardial infarction (IWMI) out of which 56 (28%) had inferior wall MI without RV MI and 23 (11.5%) had inferior wall MI with RV MI.

113 (56.5%) were treated with thrombolytic therapy, 87(43.5%) did not receive thrombolytic therapy. 8 (4%) patients died.

Incidence of RV Dysfunction in Study Population

RV dysfunction by TAPSE<1.5 cm

Total number of patients with RV dysfunction was 44 (22%). 23 (29.1%) patients with Inferior wall MI had RV dysfunction and 21 (17.3%) patients with anterior wall MI had RV dysfunction.

Incidence of LV Dysfunction in the Study Population

In the study population, 32.5% (65/200) had no LV dysfunction, while 37.5% (75/200) had mild LV dysfunction, 26% (52/200) had moderate LV dysfunction and 4% (8/200) had severe LV dysfunction.

RV Function in Relation to LV Function

LV dysfunction contributes significantly to RV dysfunction in acute myocardial infarction more so in anterior wall MI. As the severity of LV dysfunction increases RV function worsens which was statistically significant (p<0.05).

### LV DYSFUNCTION

<table>
<thead>
<tr>
<th>No LV Dysfunction</th>
<th>Mild (n=75)</th>
<th>Moderate (n=52)</th>
<th>Severe (n=8)</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAPSE (cm)</td>
<td>1.88±0.3</td>
<td>1.78±0.2</td>
<td>1.66±0.3</td>
<td>1.45±0.3</td>
</tr>
<tr>
<td>Sa (cm/s)</td>
<td>11.6±2.3</td>
<td>11.6±1.7</td>
<td>10.9±2.6</td>
<td>8.7±1.8</td>
</tr>
<tr>
<td>RVFAC (%)</td>
<td>44.9±12.2</td>
<td>44.5±12.2</td>
<td>40.8±9.9</td>
<td>38.8±11.3</td>
</tr>
<tr>
<td>No. of pts. With RV dysfunction</td>
<td>10 (15.4%)</td>
<td>12 (16%)</td>
<td>17 (32.7%)</td>
<td>5 (62.5%)</td>
</tr>
</tbody>
</table>

*ANOVA test applied, ** Chi square test applied.
RV Function in Relation to Thrombolytic Therapy

The mean TAPSE was similar in both the groups of patients (1.77 ± 0.3 cm). Mean tricuspid annular peak systolic velocity (Sa) was 11.4 ± 2.3 cm/s in patients who were given thrombolytic therapy compared to 11.2 ± 2.2 cm/s in whom thrombolytics were not given. Mean RV FAC in patients in whom thrombolytics were given was 42.7 ± 11% compared to 43.5 ± 12% in patients in whom thrombolytics were not given. These RV functional parameters in relation to thrombolytic therapy was statistically not significant (p=0.05).

<table>
<thead>
<tr>
<th>Thrombolytic Therapy</th>
<th>Given (n=113)</th>
<th>Not given (n=87)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAPSE (cm)</td>
<td>1.77 ± 0.3</td>
<td>1.77 ± 0.3</td>
<td>0.993</td>
</tr>
<tr>
<td>Sa (cm/s)</td>
<td>11.4 ± 2.3</td>
<td>11.2 ± 2.2</td>
<td>0.528</td>
</tr>
<tr>
<td>RV FAC (%)</td>
<td>42.7 ± 11</td>
<td>43.5 ± 12</td>
<td>0.653</td>
</tr>
<tr>
<td>Patients with RV dysfunction</td>
<td>25 (22.1%)</td>
<td>19 (21.8%)</td>
<td>0.962</td>
</tr>
</tbody>
</table>

Table 3. RV Functional Parameters and Patients with RV Dysfunction with Respect to Thrombolytic Therapy

Comparison of Demographic Features between Patients with Normal RV Function and RV Dysfunction

Out of 200 patients with acute myocardial infarction, 22% (44/200) had RV dysfunction.

Mean age of patients in RV dysfunction group was 57.4 ± 11.3 years and of patients in no RV dysfunction (normal RV function) group was 52.3 ± 11.2 years (p =0.008). 15.9% (7/44) patients in RV dysfunction group had past history of Coronary artery disease compared to 5.8% (9/156) in normal RV function group (p=0.02).

<table>
<thead>
<tr>
<th></th>
<th>Normal RV Function (n=156)</th>
<th>RV Dysfunction (n=44)</th>
<th>p-value**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>52.3 ± 11.2</td>
<td>57.4 ± 11.3</td>
<td>0.008*</td>
</tr>
<tr>
<td>Male (%)</td>
<td>125 (80.1%)</td>
<td>32 (72.7%)</td>
<td>0.29</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>53 (34.0%)</td>
<td>19 (43.2%)</td>
<td>0.26</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>46 (29.5%)</td>
<td>10 (22.7%)</td>
<td>0.37</td>
</tr>
<tr>
<td>Smoking (%)</td>
<td>100 (64.1%)</td>
<td>28 (63.6%)</td>
<td>0.95</td>
</tr>
<tr>
<td>Tobacco chewing</td>
<td>28 (17.9%)</td>
<td>8 (18.2%)</td>
<td>0.97</td>
</tr>
<tr>
<td>Past H/O CAD (%)</td>
<td>9 (5.8%)</td>
<td>7 (15.9%)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Table 4. Comparison of Demographic Features in Patients with Normal RV Function to those with RV Dysfunction

*Student t test applied, **for rest 6 variables, chi square test applied.

DISCUSSION

In the present study, RV dysfunction was present in 22% (44) of the patients, which was close to that of the study by Zornoff et al[3] in which 19% of patients had RV dysfunction in early AMI.

Patients who had RV dysfunction were significantly older compared to those who had normal RV function. This finding is concordant with the study by Gloria Tamborini et al.[6] which concluded TAPSE was negatively related with age, and also with the study of Zornoff et al.[3]

No significant difference noted with regards to gender distribution, frequency of hypertension, diabetes, smoking or other forms of tobacco usage in patients with or without RV dysfunction. These observations were similar to those of Zornoff et al[3] in their study of 416 patients with acute MI.

Significantly higher number of patients in RV dysfunction group had past history of coronary artery disease. This observation was consistent with that of the study by Zornoff et al[3] in which previous coronary artery disease was significantly high in the patients with RV dysfunction.

All the echocardiographic measures of RV functions (TAPSE, Sa and RV FAC) decreased as the LV ejection fraction decreased signifying that RV function was significantly dependent on LV function. These findings were consistent to the findings of the study by J L Caplin et al,[7] PflistererM et al[8] and A Marmor et al.[9] In these studies also RV function was dependent on LV ejection fraction in patients with AWMI, but it is independent of LV EF in patients with IWMI.

These findings suggest the pathophysiological mechanisms of RV dysfunction in acute myocardial infarction. In AWMI the RV dysfunction is predominantly due to LV dysfunction which causes acute increase in afterload of RV leading to contractile dysfunction of RV. In contrast, in IWMI the RV dysfunction was probably due to direct involvement of RV myocardium due to ischaemia.

There was no significant difference in echocardiographic RV functional parameters between patients who were given thrombolytic therapy and those who were not given thrombolytic therapy. These findings were similar to the studies of Roth A et al[10] and Tabone X et al[11] which suggested thrombolytic therapy was of little benefit in improving RV function early after MI. In another study by Kinn et al,[12] there was rapid improvement in haemodynamics only in patients with successful reperfusion of the infarct related artery after thrombolytic therapy. Probably failed reperfusion or early stunning of RV might have contributed to these findings in our study.

CONCLUSION

Nearly one fourth of patients with Acute ST-Elevation Myocardial Infarction had right ventricular dysfunction. LV dysfunction contributes significantly to RV dysfunction in acute myocardial infarction more so in anterior wall MI. As the severity of LV dysfunction increases RV function worsens.

REFERENCES


