Assessment of Right Ventricular function by Echocardiography in Inferior wall Myocardial Infarction and Angiographic correlation to Proximal Right Coronary Artery Stenosis

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Abstract: Right ventricular (RV) infarction imposes a higher risk of adverse events in inferior wall myocardial infarction (IWMI). In this study, we have attempted to correlate various indices of RV function assessed by echocardiography with presence of proximal right coronary artery (RCA) stenosis in patients with first episode of acute IWMI.

Methods: 102 patients with first episode of acute IWMI underwent echocardiographic assessment within 24 h of symptom onset and RV function was assessed by RV fractional area change (RVFAC), tricuspid annular plane systolic excursion (TAPSE), myocardial performance index (MPI) and tissue Doppler velocities from RV free wall were measured. Patients underwent coronary angiogram (CAG) subsequently and they were classified into group 1 and group 2 based on the presence or absence, respectively, of a significant proximal RCA stenosis.

Results: There were 102 patients with first episode of IWMI of which 78 patients underwent CAG. There was significant difference between group 1 (n = 26) and group 2 (n=52) in TAPSE, MPI by tissue Doppler and in tissue Doppler systolic velocity from RV free wall. There was a good inter observer correlation for TAPSE, MPI by TDI, and S0 velocity. RV function indices like TAPSE, MPI-TDI and S0 velocity are useful in predicting proximal RCA stenosis in first episode of acute IWMI.

Keywords: Right ventricular function, Right coronary artery stenosis, Right ventricular infarction

I. Introduction

In inferior wall MI, Presence of RVMI imposes an increased risk of complications like shock, arrhythmia and death.[1] Occurrence of proximal right coronary artery is usually responsible for RV infarction in inferior wall MI (relative risk 3.0).[1][2] ECG changes are transient and disappear in 48% of cases within 10 hours making it a less dependable tool in late presentations.[3] There are only limited studies validating usefulness of various echocardiographic (Echo) parameters of RV function in assessing RV infarction and predicting proximal RCA stenosis. Most of them assessed only a single parameter of RV function and many lacked angiographic correlation. In this study, we tried to assess whether echocardiographic assessment of RV function was useful to predict proximal RCA stenosis to help in arriving at a subset of inferior wall MI patients at higher risk of adverse clinical events Tissue Doppler indices like MPI have proven role in assessing global RV function[4] Our study attempts to assess various parameters of RV function within 24 h of presentation in first episode of acute inferior wall MI and to correlate them with a proximal RCA lesion in coronary angiogram.

II. Methods

All Consecutive patients admitted to coronary care unit of the Dept. of Cardiology, Villupuram Medical College, from July 2015 to December 2015 with first episode of acute inferior wall myocardial infarction presenting within 24 hours of symptom onset were recruited for the study. Informed consent was taken from the patients. The study was approved by the Institutional Ethics Committee. Following definitions were used for the study. Inferior wall myocardial infarction was defined as ischemic cardiac pain lasting more than 30 min., characteristic ST-segment elevation of _0.1 mV in two or more inferior leads (II, III, aVF) and CK-MB elevation more than twice the upper reference limit. ECG evidence of RV infarction was defined as ST segment elevation of _0.1 mV in V4R in ECG taken within 6 hours of onset of symptoms. Significant proximal RCA stenosis was defined in the coronary angiogram by the presence of occlusion, _70% stenosis, acute thrombosis or dissected plaque in RCA before the origin of first major RV branch. Exclusion criteria were previous documented abnormal ventricular function, left bundle branch block, atrial fibrillation, paced rhythm, valvular heart disease more than mild as per ACC/AHA criteria, pulmonary hypertension with RV systolic...
pressure by Echo >40 mmHg, pulmonary embolism, poor Echo window and ECG with lead V4R not taken within 6 hours of symptom onset. Echocardiographic assessment of RV function was performed as early as possible, within 24 hours of symptom onset in the selected cases after exclusions and coronary angiogram performed as a part of primary PCI or within one month of index event as an elective procedure to assess presence of a significant proximal RCA stenosis. Patients were divided into two groups according to angiographic localization of lesion, group I with significant proximal RCA stenosis, and group 2 without significant proximal RCA stenosis.

2.1 Assessment of RV function

For assessment of RV function the following parameters were used.

**RVFAC**

RVFAC was defined as (RV end diastolic area _ RV end systolic area)/end diastolic area _ 100. Right ventricular area in diastole and systole were obtained by tracing the RV endocardium in both phases from the annulus along free wall to apex and then back to annulus along inter ventricular septum in apical 4 chamber view.

**TAPSE**

In apical 4-chamber view, M-mode cursor was placed through tricuspid annulus at lateral RV free wall in such a way that the annulus moved along M-mode cursor. From M-mode tracing the amount of longitudinal motion of annulus at peak systole was measured. Total displacement was measured by leading edge of echoes and expressed in millimeter.

**MPI by pulsed-wave Doppler method (MPI-PW)**

In apical 4-chamber view, pulsed wave Doppler trans tricuspid flow velocities are recorded by placing the sample volume between the leaflet tips in the center of the flow stream. Doppler beam was aligned parallel to RV inflow and measurements were taken at end expiration. Trans-tricuspid early rapid filling velocity (E), peak atrial filling velocity (A), E/A ratio and E wave deceleration time were measured. Tricuspid valve closure opening time (TCO) was measured as the time interval from tricuspid valve closure marked at the end of A wave to tricuspid valve opening marked at the beginning of E wave in the next cardiac cycle in the pulse wave Doppler tracing. Pulsed Doppler of RV outflow was taken by placing the sample volume in RV outflow tract. Ejection time (ET) was calculated as time from onset to cessation of flow. Beats with less than 5% variation in ReR interval were taken to allow accurate measurement of myocardial performance index (MPI). MPI was calculated as TCO-ET divided by ET.

**Pulsed wave tissue Doppler imaging**

Pulsed TDI images were acquired by placing TDI cursor on the right ventricular free wall at the level of tricuspid annulus.3.5 mm sample volume was used. A major positive velocity (S0) was recorded with the movement of annulus towards apex during systole. With the movement of annulus towards base during diastole, two major negative waves were recorded-one during early diastole (E0) and one during late diastole (A0). S0 duration was measured as ejection time (ET), the time between the end of S0 and the beginning of E0 as isovolumic relaxation time (IRT), time between end of A0 and beginning of S0 as isovolumic contraction time (ICT). Right ventricular MPI is calculated as (IRT + ICT)/ET.

**RV wall motion abnormality**

The presence or absence of RV wall motion abnormality was assessed qualitatively from different views. In the parasternal view of RV inflow, wall motion of anterior and inferior wall of RV was assessed. Parasternal short axis view of RVOT was used to assess RWMA of RVOT and parasternal short axis view at papillary muscle level used to assess anterior, lateral and inferior walls of RV. From apical 4-chamber view wall motion abnormality of lateral wall of RV was assessed.

**RV diastolic dysfunction**

Trans-tricuspid E/A ratio, E/E0 ratio, E deceleration time were taken for grading RV diastolic dysfunction. Impaired relaxation was defined as E/A ratio <0.8, pseudo normalization as E/A ratio 0.8 to 2.1 with E/E0 ratio >6 and restrictive filling as E/A ratio >2.1 with deceleration time <120ms.

2.2. Statistical analysis

Comparison of categorical variables was performed by chi-square test. Continuous variables were expressed as means and standard deviations and analysis was done using two tailed t-test for equality of means. A p value of <0.05 accepted as statistically significant. Sensitivity and specificity
was calculated using standard formulas. ROC curves were constructed to obtain optimal cut off value for S0 velocity and MPI by tissue Doppler to predict proximal RCA stenosis.

### III. Results

During study period 102 patients were admitted with first episode of acute IWMI. After exclusions, 90 patients underwent echocardiographic assessment of RV function. 12 patients did not turn up for coronary angiogram. Study group comprised of 78 patients, 67 male and 11 female. Group 1 had 26 patients in whom there was a proximal RCA stenosis and group 2 had 52 patients who were having no stenosis in proximal RCA. There was no statistically significant difference between groups with regard to baseline variables like age, sex, body mass index (BMI), type 2 diabetes mellitus or hypertension. Mean time to ECG and echocardiographic assessment were also comparable. Systolic and diastolic blood pressures were significantly lower in patients with proximal RCA stenosis (Table 1).

#### Table 1: Clinical Parameters In Patients With And Without Proximal RCA Artery Stenosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group 1(N=26)</th>
<th>Group 2(N=52)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>56</td>
<td>55.2</td>
<td></td>
</tr>
<tr>
<td>Sex (Male%)</td>
<td>81</td>
<td>88</td>
<td></td>
</tr>
<tr>
<td>Type 2 Dm (%)</td>
<td>48</td>
<td>39</td>
<td></td>
</tr>
<tr>
<td>Htn (%)</td>
<td>54</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>Time To Echo</td>
<td>13.1</td>
<td>14.5</td>
<td></td>
</tr>
<tr>
<td>Systolic Bp</td>
<td>106.3</td>
<td>131.2</td>
<td></td>
</tr>
<tr>
<td>Diastolic Bp</td>
<td>71.5</td>
<td>82.3</td>
<td></td>
</tr>
<tr>
<td>Primary Pci (%)</td>
<td>11</td>
<td>9.4</td>
<td></td>
</tr>
<tr>
<td>Thrombolytic Therapy (%)</td>
<td>76</td>
<td>77</td>
<td></td>
</tr>
</tbody>
</table>

Left ventricular ejection fraction was significantly lower in first group. There was no significant difference in left ventricular diastolic dysfunction between the groups. Presence of right ventricular wall motion abnormalities and right ventricular diastolic dysfunction were higher in the first group depicting study design. Various parameters of RV function showed significant difference between the two groups. RV fractional area change, TAPSE and tissue Doppler velocities from RV free wall were significantly lower and MPI by pulsed Doppler and tissue Doppler were higher in proximal RCA group (Table 2).

#### Table 2: Echo Assessment in Patients with and without Proximal RCA Artery Stenosis

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>GROUP1(N=26)</th>
<th>GROUP 2(N=52)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVFAC</td>
<td>32</td>
<td>44</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td>TAPSE</td>
<td>13.4</td>
<td>21.1</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td>MPI-PW</td>
<td>0.41</td>
<td>0.31</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td>S0</td>
<td>9.6</td>
<td>14.9</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>EO</td>
<td>8.4</td>
<td>12.5</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td>A0</td>
<td>12.6</td>
<td>17.6</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>ICT</td>
<td>83</td>
<td>71</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>IRT</td>
<td>111</td>
<td>84.6</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>ET</td>
<td>224</td>
<td>291</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>MPI-TDI</td>
<td>0.85</td>
<td>0.64</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>LV/E</td>
<td>56</td>
<td>63</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td>EM/AM</td>
<td>1.09</td>
<td>1.21</td>
<td>P&lt;0.09</td>
</tr>
<tr>
<td>RV WMA (%)</td>
<td>74</td>
<td>2.5</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>RV DIASTOLIC DYSFUNCTION (%)</td>
<td>38.4</td>
<td>7.4</td>
<td></td>
</tr>
</tbody>
</table>

ASE guidelines suggest a tissue Doppler systolic velocity _10 cm/s, MPI-TDI _ 0.55 and RVFAC _ 35 as indicative of RV systolic dysfunction. For TAPSE cut off value according to guideline predicted proximal RCA lesion with good sensitivity and specificity, but for tissue Doppler indices new cut off values had to be determined by constructing ROC curve. We constructed ROC curves for tissue Doppler systolic velocity (S0) and MPI to obtain optimal cut off values to predict proximal RCA lesion. S0 _ 12.3 predicted proximal RCA lesion with sensitivity of 90.3% and specificity of 94.3%. MPI-TDI _ 0.69 also predicted proximal RCA lesion with good sensitivity and specificity (94.7% and 93.5% respectively) RVFAC and presence RV wall motion abnormalities also predicted proximal RCA lesion with good sensitivity and specificity. LV ejection fraction and presence of RV diastolic dysfunction was not found to be useful to predict proximal RCA lesion.

### IV. Discussion

The incidence of RVMI in inferior wall MI varies from 20% to 50% in various studies, and in less than 10% of patients RVMI is hemodynamically significant. In-hospital mortality rate for IWMI with RV
infarction is 31% compared to 6% in IWMI without RVMI. Mortality of cardiogenic shock due to right ventricular infarction (55%) was comparable to that due to left ventricular infarction (59%) in spite of patients being younger and a greater incidence of single vessel disease. Hence the diagnosis of RV infarction and assessment of RV function is of great importance in IWMI. Observational studies have suggested that early reperfusion in inferior wall MI with RV infarction is beneficial. In patients with IWMI with RVMI, in whom PCI was successful, persistent hypotension and mortality were less compared to patients in whom PCI was unsuccessful. Diagnosis of RV infarction by physical examination depends on the triad of hypotension, venous distension and clear lung fields in the setting of inferior wall myocardial infarction but it is only 25% sensitive. JVP elevation greater than 8 cm and a Kussmaul’s sign predict RVMI with greater sensitivity but less specificity. Hypovolemia or Hypervolemia also creates problems in the diagnosis by physical examination. ECG is also less sensitive for diagnosis of RV infarction. ST segment elevation 0.1 mV in lead V4R had a sensitivity of 83% in diagnosing RVMI confirmed by post-mortem examination. Sensitivity of lead V4R in diagnosing RVMI was 83% and specificity 77% in another study. ST segment elevation in lead III more than II is 97% sensitive but only 70% specific for right ventricular infarction. In our study, the sensitivity of ECG to predict proximal RCA lesion was 76%. Variable RF as function as shown in this study can be used to diagnose RV infarction and predict proximal RCA lesion with a higher sensitivity and specificity. LV ejection fraction as well as systolic and diastolic blood pressure measured significantly lower in proximal RCA group proving the hemodynamic significance of right ventricular infarction in IWMI. Right ventricular wall motion abnormalities could be detected in 73% of patients in group 1 in this study and found to correlate with proximal RCA stenosis. Distal right coronary artery lesions can also produce posterior right ventricular wall motion abnormalities as it is the most distal RCA territory and hence the specificity to predict proximal RCA lesion will be less. Combined right ventricular lateral wall and posterior wall motion abnormalities are indicative of more proximal RCA occlusions. Presence of right ventricular wall motion abnormalities in the setting of IWMI was predictive of right ventricular infarction with a sensitivity of 83% and specificity of 93% in earlier studies but there was no angiographic correlation. Assessment of RV wall motion abnormalities is subjective and often difficult when echocardiographic windows are poor. RV wall motion abnormalities can be present in other disease states also like pulmonary embolism and pulmonary hypertension.

Right ventricular diastolic dysfunction was also significantly higher in group 1. Mukhaini M et al had earlier demonstrated significant RV diastolic dysfunction in RVMI associated with IWMI. RV diastolic dysfunction may have contributed to the increase in MPI which is a measure of global RV function. Coming to other indices of RV function in the study, RVFAC was significantly lower in group 1 (32 _ 5.2) compared to group 2 (44 _ 5.2). RVFAC has been found to have a good correlation with MRI derived RVEF and is also found to have prognostic significance in patients with myocardial infarction. No studies are available correlating RVFAC with proximal RCA lesion. TAPSE was significantly lower in patients with proximal RCA lesion (mean 13.5 _ 1.3 Vs 21.3 _ 1.7, p < 0.05). Earlier studies had shown good correlation of TAPSE with ECG evidence of RV infarction, but the number of patients was less and there was no angiographic correlation. TAPSE was also an independent predictor of mortality in inferior wall MI. TAPSE also found to have a good correlation with radionuclide derived EF. TAPSE < 16 indicates RV systolic dysfunction according to ASE guidelines. In our study, TAPSE _ 16 predicted proximal RCA lesion with a sensitivity of 92.3% and specificity of 100% (p < 0.001). There was a good correlation between observers in the study. TAPSE has some limitations in that measurement is restricted to longitudinal function of RV free wall and functional status of LV may have an influence on it. Myocardial performance index by pulsed wave Doppler (MPI-PW) was also found to correlate with a proximal RCA lesion being significantly higher in group 1 (0.42 _ 0.2 Vs 0.29 _ 0.2, p < 0.05). MPI _ 0.30 by pulsed wave Doppler was correlated with the presence of RVMI in earlier studies but angiographic correlation was not studied. Calculated in this method is less reliable as it utilizes two different cardiac cycles for measurement of time intervals and inter-observer correlation is also less compared to other measures. Tissue Doppler indices namely velocities from RV free wall at the level of tricuspid annulus and MPI-TDI showed good correlation with proximal RCA lesion. Good inter-observer correlation was observed. These indices were easy to measure even when the echocardiographic windows were poor. ASE guidelines suggest a tissue Doppler systolic velocity _ 10 cm/s and MPI-TDI _ 0.55 as indicative of RV systolic dysfunction. But in this study these cut off values failed to predict proximal RCA lesion with good sensitivity and specificity. So we constructed ROC curves to determine optimal cut off values for S0 and MPI-TDI. It was found that S0 _ 12.3 predicted proximal RCA lesion with sensitivity of 90.3% and specificity of 94.3%, MPI-TDI _ 0.69 with a sensitivity of 94.7% and specificity of 93.5%. Ozdemir and colleagues had a similar result in a study of 60 cases of inferior wall MI but echocardiographic assessment was performed within 2 days. Right ventricular function is known to recover earlier after IWMI. So we had done echo assessment in each group of patients within 24 hours of symptom onset and may have a better assessment of RV function. Tissue Doppler systolic annular velocity has also been shown to correlate with prognosis in IWMI. MPI was found to correlate with radionuclide derived RVEF in earlier studies. Mezulin et
eral found out that systolic that velocity <11.5 cm/s will predict RVEF < 45%.[23] Oguzhan et al. had also demonstrated depressed RV free wall velocities detected by color tissue Doppler imaging in RVM associated with IWMI.[24] Our findings in the study are concordant with earlier studies and provides optimal cut off values to predict proximal RCA lesion.

V. Conclusion

Echocardiographic assessment of various parameters of RV function showed significant difference between groups with or without proximal RCA lesion. Tissue Doppler systolic annular velocity, myocardial performance index and TAPSE are easy to perform and useful in predicting proximal RCA as infarct related artery.

Conflicts of interest: none to declare.

References