Assessment of diastolic dysfunction using multidetector computerised tomography in normotensive noncoronary artery disease diabetic patients

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Aims and objectives

Diabetes mellitus is one of the most common chronic diseases. Several epidemiological studies have consistently reported diabetes as a strong risk factor for the development of cardiovascular diseases. Diabetes inflicts a direct insult to the myocardium, with cellular, structural and functional changes manifest as the diabetic myocardial phenotype.

Diastolic heart failure was traditionally considered a comparatively rare and benign disorder when compared to systolic heart failure. Large observational studies have recently shown that diastolic heart failure accounts for approximately 50% of all heart failure cases and its prevalence is growing. Up to 37% of individuals with diastolic heart failure have diabetes. Diabetic cardiomyopathy is defined as ventricular dysfunction occurring independently of coronary artery disease (CAD) and hypertension.

Echocardiography and Doppler imaging have been the traditional and most commonly performed non-invasive investigations for left ventricular function assessment. Recently Cardiac multidetector computed tomography (MDCT) has emerged as a potent non-invasive imaging modality for the evaluation of coronary atherosclerosis. So far, multiphase CT studies have been mostly restricted to cardiac morphology and LV systolic function analysis, and very little information is available on the feasibility of cardiac MDCT imaging to assess diastolic LV function.

Accordingly, the present study aimed to evaluate the feasibility of cardiac MDCT for assessment of diastolic function when compared with echocardiography. The evaluation of LV diastolic function was based on the assessment of transmitral velocity and mitral septal tissue velocity.
Methods and materials

Study design: Prospective analytical case control study.

The study was conducted from July 2013 to August 2015 at our institution. Asymptomatic normotensive non CAD diabetic patients were compared to age matched asymptomatic normotensive non CAD non-diabetic individuals for the evaluation of diastolic dysfunction by echocardiography and MDCT.

Cases and controls:

15 diabetic patients were enrolled for the study as cases. Written informed consent was obtained from all the participants prior to their enrolment in this study for undergoing 2D echocardiography and MDCT. All the participants were informed about the protocol.

Cases were included in the study protocol if they met the following criteria: (i) Diabetic patients, (ii) age range of 25-60 years, (iii) asymptomatic patients without diabetic complications, hypertension, hyperlipidemia or features of CAD.

Exclusion criteria set for the study were: (i) Hypertension, (ii) dyslipidemia, (iii) obesity, (iv) female patients planning pregnancy, (v) valvular heart disease, (vi) congestive heart failure, (vii) history of CAD. The purpose was to exclude other parameters which can cause diastolic dysfunction independent of diabetes and may act as confounding factors.

15 age and sex- matched healthy non-diabetic & normotensive individuals were selected as the controls, whose blood tests, 2D echo and MDCT were done with their consent for comparison with the cases.

Imaging:

Echocardiography

All the subjects underwent 2D echocardiography first; LV diastolic function assessment being done by conventional Doppler along with the Valsalva maneuver, Tissue Doppler imaging (TDI), and color M-mode flow propagation velocity. The Modified Simpson's or Bullet method techniques were used for the calculation of LV ejection fraction and LV mass was determined by the area#length method.

The following velocities and times were recorded with conventional Doppler at the mitral inflow as well as at the entrance of the pulmonary veins into the left atrium: Early ventricular filling (E), flow related to atrial contraction (A), Deceleration Time (DT),
Isovolumic Relaxation Time (IVRT) and E/A ratio. Measurements were averaged from 3 end-expiratory cycles.

Subjects were also made to perform Valsalva maneuver while echocardiography. Mitral inflow measurements (E and A) were obtained during phase II (straining) of the Valsalva maneuver. The maneuver was considered valid only if there was a 10% decrease in E velocity compared with baseline. Diastolic dysfunction was considered to be present if the change in the E/A ratio was ≥40% from baseline after the Valsalva maneuver.

Early diastolic mitral annular velocity was assessed by using pulsed wave TDI of both the septal and lateral walls in the apical 4-chamber view (averaged from 3 cardiac cycles). Both septal and lateral walls ≥8 cm/s demonstrated diastolic LV dysfunction in our study.

Diastolic dysfunction was considered to be present if diastolic abnormality was found with any of the echocardiographic approaches viz. conventional Doppler, the effects of the Valsalva maneuver on transmitral flow velocities and TDI.

**MDCT**

Next, MDCT imaging was performed with a 64#slice helical scanner. Before the scan, patients were monitored for blood pressure and heart rate. Patients with heart rate >65 beats/min were given metoprolol 50 to 100 mg orally, unless contraindicated. The renal function test of all the subjects was done as we had to use contrast for MDCT. Scan parameters used were- Scan type Cardiac helical, Pitch 0.160:1, Table speed row 6.4, Rotation time 0.35 sec, Slice thickness 0.625, Beam collimation 40 mm, Voltage 120 kVp, Current 500mA, Recon type Standard SSB, Scan length 10 cm.

**MDCT Post processing and analysis:**

CT Post processing was performed with advantage workstation. By retrospective ECG# gating our MDCT divided each cardiac cycle into ten phases and LV volume and LV length were calculated for each phase. This LV volume was plotted against time. Then, we calculated the difference in LV volume for each consecutive phase per second, which was again plotted against time. This graph gave the values of Early (E) and Late (A) peak transmitral velocities. These values were divided with respective transmitral area in E phase and A phase which gave us the values of E and A comparable to 2D Echo. By the ratio of E and A (E/A) we can calculate the diastolic dysfunction. Likewise, we plotted a LV length v/s time graph. We, then, plotted change in LV length per second v/s time graph. This graph gave us the value of Ea (peak mitral septal tissue velocity in early diastole). By calculating E/Ea (LV filling pressures) we can estimate diastolic dysfunction.

A. Transmitral flow:
Fig. 1: Transmirtal flow: Left ventricular (LV) volumes were measured for ten phases per cardiac cycle, using short-axis images by outlining endocardial contours in each phase. They were then plotted in a volume versus time curve (right upper). Changes in LV volumes between two consecutive phases were plotted against time (transmitral flow vs. time curve) (right lower). Subsequently, early and late peak transmitral flows (ml/s) were derived.

References: Department of Radiodiagnosis and Imaging, Institute Of Medical Sciences, Banaras Hindu University, Varanasi, UP, INDIA

B. Mitral valve area:
Fig. 2: Mitral valve area: Measurements were taken at the most distal level of the mitral valve leaflets (smallest mitral valve area) using reconstructed images at peak early and late transmural velocities. The mitral valve area was measured at the tip of the leaflets on short-axis views.

References: Department of Radiodiagnosis and Imaging, Institute Of Medical Sciences, Banaras Hindu University, Varanasi, UP, INDIA

C. Mitral septal tissue velocity:
Fig. 3: Mitral septal tissue velocity: Anatomic markers were positioned at the mitral septal annulus and cardiac apex. The LV length (cm) was calculated for each phase and plotted in a LV length versus time curve (right upper). Next, changes in LV length between 2 consecutive phases were calculated. Based on these numbers, mitral septal tissue velocities (cm/s) were calculated for each phase (velocity vs. time curve) (right lower).

References: Department of Radiodiagnosis and Imaging, Institute Of Medical Sciences, Banaras Hindu University, Varanasi, UP, INDIA

Transmitral velocity = \( \frac{\text{Transmitral flow (ml/s)}}{\text{Mitral valve area (mm}^2)\right)} = \frac{\text{cm}^3/\text{s}}{\text{cm}^2} = \frac{\text{cm}}{\text{s}} \)
Fig. 4: Transmitral velocity (cm/s) was calculated using the above formula.

References: Department of Radiodiagnosis and Imaging, Institute Of Medical Sciences, Banaras Hindu University, Varanasi, UP, INDIA

Diastolic function was graded in four categories using the following criteria:

1. Normal diastolic function (1 ≤ E/A < 2 and E/Ea ≤ 8);
2. Impaired relaxation pattern (diastolic dysfunction grade I; E/A < 1 and E/Ea ≤ 8);
3. Pseudo normal pattern (diastolic dysfunction grade II; 1 ≤ E/A < 2 and 9 ≤ E/Ea ≤ 12); and
4. Restrictive filling pattern (diastolic dysfunction grade III; E/A ≥ 2 and E/Ea ≥ 13).

Statistical analysis:

The intergroup and intermodality comparisons of variables obtained was done by applying t-test and chi-square tests by using the SPSS software (SPSS version 17.0; SPSS, Chicago, IL, USA). P-value < 0.05 was used as an indicator of significance.
Fig. 1: Transmural flow: Left ventricular (LV) volumes were measured for ten phases per cardiac cycle, using short-axis images by outlining endocardial contours in each phase. They were then plotted in a volume versus time curve (right upper). Changes in LV volumes between two consecutive phases were plotted against time (transmural flow vs. time curve) (right lower). Subsequently, early and late peak transmural flows (ml/s) were derived.

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Fig. 2: Mitral valve area: Measurements were taken at the most distal level of the mitral valve leaflets (smallest mitral valve area) using reconstructed images at peak early and late transmitral velocities. The mitral valve area was measured at the tip of the leaflets on short-axis views.

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Fig. 3: Mitral septal tissue velocity: Anatomic markers were positioned at the mitral septal annulus and cardiac apex. The LV length (cm) was calculated for each phase and plotted in a LV length versus time curve (right upper). Next, changes in LV length between 2 consecutive phases were calculated. Based on these numbers, mitral septal tissue velocities (cm/s) were calculated for each phase (velocity vs. time curve) (right lower).

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\text{Transmitral velocity} = \frac{\text{Transmitral flow (ml/s)}}{\text{Mitral valve area (mm}^2\text{)}} = \frac{\text{cm}^3/\text{s}}{\text{cm}^2} = \frac{\text{cm}}{\text{s}}
\]

Fig. 4: Transmitral velocity (cm/s) was calculated using the above formula.
Results

A total of 15 normotensive diabetic patients were included in the study as cases. The mean age was 48.20 ± 9.283 years.

Table 1

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Diabetics (Mean±SD)</th>
<th>Non-diabetics (Mean±SD)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48.20±9.283</td>
<td>50.87±5.630</td>
<td>.350</td>
</tr>
<tr>
<td>Pulse Rate (/min)</td>
<td>71.33±5.473</td>
<td>68.27±4.935</td>
<td>.118</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>116.53±11.963</td>
<td>116.67±8.837</td>
<td>.973</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>70.53±5.041</td>
<td>67.87±4.307</td>
<td>.131</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.1200±1.06382</td>
<td>20.9867±86344</td>
<td>.709</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>9.5373±1.98740</td>
<td>5.1400±.43556</td>
<td>.0001</td>
</tr>
<tr>
<td>FBS (mg/dL)</td>
<td>124.733±38.54381</td>
<td>92.333±9.4855</td>
<td>.004</td>
</tr>
<tr>
<td>Cholesterol (mg/dL)</td>
<td>39.27±31.183</td>
<td>41.40±28.830</td>
<td>.016</td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>120.13±28.468</td>
<td>122.40±13.076</td>
<td>.781</td>
</tr>
<tr>
<td>HDL (mg/dL)</td>
<td>57.00±10.981</td>
<td>60.80±10.101</td>
<td>.332</td>
</tr>
<tr>
<td>LDL (mg/dL)</td>
<td>71.67±29.796</td>
<td>56.33±10.761</td>
<td>.071</td>
</tr>
<tr>
<td>VLDL (mg/dL)</td>
<td>22.1600±6.63204</td>
<td>20.5000±3.54482</td>
<td>.04</td>
</tr>
</tbody>
</table>

Table 1: Baseline Characteristics of the Cases (diabetics) and Controls (non-diabetics) [SBP: systolic blood pressure; DBP: diastolic BP; BMI: basal metabolic rate; FBS: fasting blood sugar; TG: triglycerides; HDL: high density lipoprotein; LDL: low density lipoprotein; VLDL: very low density lipoprotein]

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Table 1 shows the baseline characteristics of the study population for both Diabetics and non-diabetics. None of the values show significant difference except for fasting blood sugar and HbA1c depicting that our cases and controls were comparable for all characteristics except for Diabetes. Above table also signifies that our study population is normotensive and none of them are obese or hyperlipidemic. Thus, we successfully excluded any confounding factor which may contribute to diastolic dysfunction by itself.
**Table 2**: Diastolic Function Parameters for 2D Echocardiography and MDCT in diabetic patients and non-diabetics.

**References**: Department of Radiodiagnosis and Imaging, Institute Of Medical Sciences, Banaras Hindu University, Varanasi, UP, INDIA

On statistical analysis we found that there is a significant difference between diabetic subjects and non-diabetic normal population in the values of E (p=0.039), E/A ratio (p=0.017) and Ea (p=0.000) as measured on 2D Echocardiography and E (p=0.036), E/A ratio (p=0.033) and Ea (p=0.000) as measured on MDCT [Table 2]. Hence, it can be said that these parameters were comparable between 2D echocardiography and MDCT for the study groups.

**Table 3**: Diastolic Dysfunction (DD) in cases as detected by 2D Echocardiography and MDCT

**References**: Department of Radiodiagnosis and Imaging, Institute Of Medical Sciences, Banaras Hindu University, Varanasi, UP, INDIA
In our study, total 73.3% people were observed to have grade I to grade II diastolic dysfunction on echocardiography and MDCT as compared to only 13.3% of non-diabetics. None of them had grade III (Restrictive filling pattern) diastolic dysfunction [Table 3]. The difference between the two groups was statistically significant (p=0.001). In non-diabetics (controls), only 2 subjects had Grade 1 diastolic dysfunction. Significantly high prevalence of LV diastolic dysfunction in normotensive patients with type 2 diabetes mellitus without coronary heart disease was observed in our study.

Further, correlation was done between diastolic parameters of 2D Echocardiography and MDCT in both the study groups. All the diastolic parameters had good correlation between echocardiography and MDCT in the study population [Figure 5]. Similar findings were observed in controls [Figure 6] as well. So, cardiac MDCT and 2D Echocardiography can be said to be comparable for assessment of diastolic dysfunction.

Fig. 5: Correlation between cardiac MDCT and 2D Echocardiography for assessment of diastolic parameters [E (r = 0.992; p < 0.001), A (r = 0.974, p < 0.001), E/A (r = 0.979; p < 0.01), Ea (r = 0.977; p < 0.001), and E/Ea (r = 0.994; p < 0.001)] in Diabetic patients.

References: Department of Radiodiagnosis and Imaging, Institute Of Medical Sciences, Banaras Hindu University, Varanasi, UP, INDIA
Fig. 6: Correlation between cardiac CT and 2D Echocardiography for assessment of diastolic parameters [E (r = 0.954; p < 0.001), A (r = 0.900, p < 0.001), E/A (r = 0.904; p < 0.01), Ea (r = 0.923; p < 0.001), and E/Ea (r = 0.973; p < 0.001)] in non-diabetic subjects.

References: Department of Radiodiagnosis and Imaging, Institute Of Medical Sciences, Banaras Hindu University, Varanasi, UP, INDIA

Thus this study showed that normotensive, asymptomatic patients with diabetes and normal LV systolic function may exhibit diastolic dysfunction. Accordingly, additional post processing for diastolic dysfunction may have the potential to enhance the clinical evaluation derived from cardiac CT. The information that is needed for evaluation of diastolic function can be derived from multiphase CT without additional image acquisition or radiation dose.

Limitations:

- Small sample size
- Tube current modulation was not done for acquisition of MDCT dataset.
- Several parameters may affect the transmitral velocity measurements including filling pressures, degree of LV relaxation, and myocardial elastic recoil and stiffness. For overcoming these limitations, additional measurements have been proposed, including the evaluation of pulmonary venous velocity and M-mode echocardiography flow velocity curves. However, these measurements were not taken in our study.
Table 1: Baseline Characteristics of the Cases (diabetics) and Controls (non-diabetics) [SBP: systolic blood pressure; DBP: diastolic BP; BMI: basal metabolic rate; FBS: fasting blood sugar; TG: triglycerides; HDL: high density lipoprotein; LDL: low density lipoprotein; VLDL: very low density lipoprotein]

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Table 2: Diastolic Function Parameters for 2D Echocardiography and MDCT in diabetic patients and non-diabetics.
<table>
<thead>
<tr>
<th>Diastolic Dysfunction (DD)</th>
<th>Echocardiography (n=15) No. (%)</th>
<th>MDCT (n=15) No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>4 (26.7)</td>
<td>4 (26.7)</td>
</tr>
<tr>
<td>Grade 1 DD</td>
<td>10 (66.7)</td>
<td>10 (66.67)</td>
</tr>
<tr>
<td>Grade 2 DD</td>
<td>1 (6.67)</td>
<td>1 (6.67)</td>
</tr>
<tr>
<td>DD</td>
<td>11 (73.3)</td>
<td>11 (73.3)</td>
</tr>
<tr>
<td>Total</td>
<td>15 (100)</td>
<td>15 (100)</td>
</tr>
</tbody>
</table>

**Table 3:** Diastolic Dysfunction (DD) in cases as detected by 2D Echocardiography and MDCT

Fig. 5: Correlation between cardiac MDCT and 2D Echocardiography for assessment of diastolic parameters[E (r = 0.992; p < 0.001), A (r = 0.974, p < 0.001), E/A (r = 0.979; p < 0.01), Ea (r = 0.977; p < 0.001), and E/Ea (r = 0.994; p < 0.001)] in Diabetic patients.

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Fig. 6: Correlation between cardiac CT and 2D Echocardiography for assessment of diastolic parameters [E (r = 0.954; p < 0.001), A (r = 0.900, p < 0.001), E/A (r = 0.904; p < 0.01), Ea (r = 0.923; p < 0.001), and E/Ea (r = 0.973; p < 0.001)] in non-diabetic subjects.

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Conclusion

There is a high prevalence of LV diastolic dysfunction with preserved systolic function in asymptomatic diabetic patients. MDCT has the potential to meet the present need for a robust tool that can establish diagnostic efficacy in future studies and thus, should be used to identify (pre-) diabetic individuals with diastolic dysfunction and to optimize their clinical management.
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