Mechanical dyssynchrony in dilated cardiomyopathy and relation to severity, etiology, and ECG

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Abstract

Background: Cardiac impairment is a maincause of mortality throughout the worldand despite optimal medical treatment, it still carries high mortality. The aim of current study was to evaluate the intraventricular dyssynchrony prevalence in patients with dilated cardiomyopathy patients and its relation to underlying etiology, QRS with and severity of systolic dysfunction.

Methods: This cross-sectional study included 58 patients with cardiomyopathy of ischemic (ICM) and non-ischemic cause (NCM). Mechanical dyssynchrony (intraventricular dyssynchrony) assessed by m-mode, pulsed Doppler, TDI and 2D speckle tracking. Assessment of left ventricular systolic function was done by ejection fraction and stroke volume. Prevalence of mechanical indices in DCM and their relation to underlying etiology, QRS duration and severity of LV systolic dysfunction were done.

Results: Prevalence of intraventricular dyssynchrony indices were more in NCM than ICM except PSI was more in ICM, and there was significant association of these indices with QRS width. There was non-significant negative correlation between these indices and LV systolic indices.

Conclusion: Mechanical dyssynchrony indices are affected by the underlying etiology and QRS width as well as severity of LV systolic dysfunction associated with existence of the mechanical dyssynchrony.

Keywords: ECG, dilated cardiomyopathy, left ventricular dysfunction, mechanical dyssynchrony

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Introduction

One of the major problems that affect about 10% of population (with age more than 65 years) is heart failure (HF) (¹). HF can be defined as the incapability of the heart to provide oxygen to meet the metabolic requests of the body, due to defect in the cardiac structure or function (²). In spite of considerable progress in medical treatment of HF, morbidity and mortality are still high with a bad prognosis in presence of conduction abnormality (³). This abnormality is considered a common finding in patients with severe HF which causes dyssynchrony (⁴,⁵,⁶). Ventricular dyssynchrony among patients with HF portends poor prognosis (⁷). Ventricular dyssynchrony in these patients with HF, as a result of...
left bundle branch block or interventricular conduction delays, leads to irregular ventricular activation which causes irregular wall motion with filling reduction (8) and reduction in cardiac output (9).

Left ventricular (LV) dyssynchrony may present in patients with HF in the absence of regionally delayed electrical activation (10), e.g. in ICM, the scar leads to inhomogeneity in dyskinetic and a kinetic muscle. The aim of current study was to evaluate the intraventricular dyssynchrony prevalence in patients with dilated cardiomyopathy and its relation to underlying etiology, QRS and severity of systolic dysfunction.

**Material and method**

This cross-sectional study was performed in Mirjan Teaching Hospital in Hilla city–Babylon province, Iraq from 12\(^{th}\) of December 2017 to the 25\(^{th}\) of April 2018. In current study, 58 patients with ICM and NCM, with mean age of 63±6 and 52±10.2, respectively, were recruited from Shaheed Al-Mihrab for cardiac catheterization and Mirjan Teaching Hospital, after having their signed consent and approval from ethical committee at Babylon College of medicine. Inclusion criteria included ICM and NCM patients. Ischemic DCM patients were categorized according to history, ECG findings, echocardiographic study (distinguishing features of an ICM include a relatively greater degree of regional heterogeneity of systolic function often with areas of frank scar or aneurysm formation, when either area of scar conforming to a well-defined coronary territory, or LV aneurysm is noted, the likelihood of an ischemic etiology is high) (11). Angiographic finding depends on the presence of stenosis more than 50% or 70% of the left main coronary artery or one of the major epicardial arteries, respectively (12).

**Echocardiography**: by VIVID 9 GE, with a transducer of 3.5MHz, all the measures were evaluated in accordance to ASE guidelines (13). ECG-guided acquisition: the patients underwent resting conventional echocardiography by measuring:

- **The ejection fraction (EF)** by modified Simpson method.
- **The stroke volume (SV)** was done by continuity equation (14). By measuring the diameter of LVOT by parasternal long axis view and tracing aortic flow (apical 5chamber view with applying pulse Doppler on LVOT) to get VTI.

**Evaluation of Intraventricular mechanical dysynchrony**

1. M-Mode method: assessed by M-mode cursor positioned at mid-ventricular level of parasternal long axis view, then measured the time between septal and posterior inward movement (SPWD). SPWD > 130ms means significant dyssynchrony (13).

2. Tissue Doppler imaging (TDI): This modality allows regional myocardial velocity measurements. Color-DTI can evaluate time-velocity measures offline and this is major benefit of it. It requires a high
frame rate (100-140 frame/sec)\(^{(15)}\). The time-to-peak myocardial systolic velocity (Ts) in six basal and six mid-LV segments (from three apical views) were estimated manually \(^{(13,16,17)}\), this was done by the following steps:

a. LV ejection timing: determining the opening and closure of aortic valve (AV) by LVOT pulsed Doppler.

b. Regions of interest are positioned at the base and mid region of LV walls (opposed walls) for time-velocity plots determination.

c. Measuring the time from the QRS complex to the systolic peak velocity for each region (Ts), so a total of 12 segments for the 3 views (Figure 1).

The following DTI-derived measures were evaluated according to \(^{(13,18,19)}\): Opposite wall difference in Ts in each 4 segments/view is with cutoff value <65msec, maximum difference in Ts (Differences between the longest and shortest Ts) is with a cutoff value <110 and Mechanical dysynchrony index (Ts-SD or Yu index), which is the SD of Ts 12-site, is with cutoff value <33ms.

![Figure (1A) Color coded tissue Doppler image of apical 4 chamber view shows dysynchrony between lateral and septal walls.](image1.png)

![Figure (2B) Color coded tissue Doppler image of apical 2chamber view shows dysynchrony between anterior and inferior wall.](image2.png)
Figure (1C) **Color coded tissue Doppler image** of apical 3 chamber view shows dyssynchrony between septal and posterior wall.

**Strain**

2D speckle tracking acquisition includes adjustment of frame rate from 40-80 frame/sec through applying AFI study for apical 3, 4 and 2 views. Analyzing of apical 3 chamber view should be done at first for timing of end systole by aortic closure. So, the GLS was evaluated automatically (Figure 2). For assessment of percentage of abnormally delayed or ineffective LV contractions [i.e. myocardial contraction after aortic closure], the post-systolic index (PSI) was measured. PSI = 100 × (peak strain after AVC-end systolic strain)/peak strain after AVC] \(^{(20,21)}\). PSI was automatically assessed \(^{(22)}\). When DLC is more than 30% of 12 LV could be considered with significant mechanical dyssynchrony \(^{(3)}\) (Figure 3).

Figure (2) 2D speckle tracking image demonstrating reduced global longitudinal strain.
Statistical analysis

Independent $t$-test and Chi-squared tests were used for assessment of continuous variables and categorical variables, respectively. Relation between different variables was done by Pearson correlation coefficient ($r$).

Results

Fifty eight cardiomyopathy patients were included in this cross-sectional observational study, 32 of them were ischemic in etiology while the rest 26 patients were non ischemic CMP. They were with no statistically significant difference in their base line demographic, ECG and echocardiographic characteristics apart from those ischemic patients were significantly older than non-ischemic group (Table 1).

Table (1) The demographic data, ECG, conventional echocardiographic measurements and 2D Speckle tracking GLS.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Total patients</th>
<th>ICM N=32</th>
<th>NCM N=26</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>58±10</td>
<td>63±6</td>
<td>52±10</td>
<td>0.000$^*$</td>
</tr>
<tr>
<td>Gender</td>
<td>Male</td>
<td>36</td>
<td>22</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>22</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>BSA(kg/cm$^2$)</td>
<td>1.86±0.22</td>
<td>1.86±0.11</td>
<td>1.82±0.3</td>
<td>0.2</td>
</tr>
<tr>
<td>HR(beat/min)</td>
<td>88±22</td>
<td>81±7</td>
<td>77±13</td>
<td>0.09</td>
</tr>
</tbody>
</table>

Figure (3) 2D speckle tracking image demonstrating post-systolic index (PSI).
### Echocardiographic parameters of intraventricular dyssynchrony

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Cutoff value (ms)</th>
<th>Total patients</th>
<th>NCM</th>
<th>ICM</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SPWD (by M-mode)</td>
<td>&gt;130ms</td>
<td>183±94 (46.5%)</td>
<td>148±59 (57.69%)</td>
<td>130±50 (37.5%)</td>
<td>0.1</td>
</tr>
<tr>
<td>Ts opposite wall difference (by TDI)</td>
<td>&gt;65ms</td>
<td>85±41 (62%)</td>
<td>98±48 (76%)</td>
<td>74±30 (48.4%)</td>
<td>0.01†</td>
</tr>
<tr>
<td>Ts max. difference (by TDI)</td>
<td>&gt;110ms</td>
<td>121±51 (60.3%)</td>
<td>137±56 (73%)</td>
<td>107±87 (46.4%)</td>
<td>0.01†</td>
</tr>
<tr>
<td>Yu index (by TDI)</td>
<td>value &gt;33</td>
<td>41±15 (68.9 %)</td>
<td>46±17 (76.9%)</td>
<td>37±13 (59.3%)</td>
<td>0.01†</td>
</tr>
</tbody>
</table>

NCM = non ischemic cardiomyopathy, ICM= ischemic cardiomyopathy, BSA=body surface area, VTI= velocitytimeintegral, GLS= Global longitudinal strain.

* Significant difference (P <0.05).

Echocardiographic parameters of intraventricular dyssynchrony that were evaluated by M-mode, pulsed Doppler, TDI and strain were expressed as mean±SD (Table 2). Prevalence of significant intraventricular dyssynchrony indices in NCM and ICM were as shown in Table (2).
Prevalence of all the intraventricular dyssynchrony parameters were higher in NCM, except PSI was higher in ICM than NCM. The abnormalities of SPWD, Ts opposite wall delay, Ts max difference, and Yu index for total patients (NCM and ICM) were not shown to be correlated with lower ejection fraction (r=0.07, 0.05, 0.04 and 0.02, respectively, with P>0.05; Figures 1, 2, 3 and 4, respectively). There was non-significant negative correlations with SV (r=-0.2, -0.8, -0.29, and -0.12; Figures 5, 6, 7 and 8, respectively) and no correlation of these parameters with GLS except PSI which showed non-significant positive correlation (r=0.2, P>0.05; Figure 9).

**Figure (1)** Correlation study of ejection fraction (EF%) and septal posterior wall delay in non-ischemic cardiomyopathy and ischemic cardiomyopathy patients.

**Figure (2)** Correlation study of ejection fraction (EF%) and Ts opposite wall delay by TDI in non-ischemic cardiomyopathy and ischemic cardiomyopathy patients.
Figure (3) Correlation study of ejection fraction (EF%) and Ts maximum wall difference by TDI in non-ischemic cardiomyopathy and ischemic cardiomyopathy patients.

Figure (4) Correlation study of ejection fraction (EF%) and Yu index by TDI in non-ischemic and ischemic cardiomyopathy patients.

Figure (5) Correlation study of stroke volume (SV) and septal posterior wall delay (SPWD) by M mode in ischemic cardiomyopathy and non-ischemic cardiomyopathy patients.
**Figure (6)** Correlation study of stroke volume (SV) and Ts opposite wall difference by TDI in ischemic and non-ischemic cardiomyopathy patients.

![Figure 6](image1)

**Figure (7)** Correlation study of stroke volume (SV) and Ts maximum wall difference by TDI in ischemic and non-ischemic cardiomyopathy patients.

![Figure 7](image2)

**Figure (8)** Correlation study of stroke volume (SV) and Yu index by TDI in ischemic and non-ischemic cardiomyopathy patients.

![Figure 8](image3)

**Figure (9)** Correlation study of global longitudinal strain (GLS%) and post systolic index (PSI) in ischemic and non-ischemic cardiomyopathy patients.

![Figure 9](image4)

Significant intraventricular dyssynchrony was seen in 57.14% for those with QRS <120msec, and 87.87% for those with QRS duration >120ms, again with significant association with longer QRS (P=0.01; Figure 10).
Discussion

A previous study \(^{(23)}\) showed that in DCM, NYHA III to IV cardiac (dyssynchrony) was common and intraventricular (dyssynchrony) prevalence is 70%. Intraventricular dyssynchrony was more prevalent in NCM patients than ICM patients. ICM is a clear subset, because the scar leads to tissue inhomogeneity in dyskinetic and akinetic muscle, where non constant contraction, relaxation, and filling may progress and cause worsening of global systolic and diastolic function. This intraventricular (dyssynchrony) may be independent of electrical conduction delay \(^{(24)}\). (DCM) is due to abnormality of the cardiac muscle, disturbing both electrical and mechanical function of the ventricle \(^{(25,26)}\). Electrical stimuli of cardiac muscle fibers become unorganized with some delay in some parts in accordance to others; this is due to conduction system abnormality or due to scarred tissue with inhomogeneous spread of excitation wave \(^{(27)}\). This unorganized contraction disrupts cardiac contraction, and decreases pumping effectiveness \(^{(13,28)}\). These can highlight the negative correlation between LV systolic indices (demonstrated by SV) and intraventricular (dyssynchrony). SV is more informative than the EF that showed no correlation with intraventricular (dyssynchrony) indices. Moreover, \(^{(29)}\) demonstrated that Intraventricular dyssynchrony had important correlation with severity of cardiac function and left intraventricular dyssynchrony was an independent predictor of hemodynamic event in the study. Postsystolic shortening (PSS) is considered an important indicator of cardiac contractility impairment and it is a prognostic factor of long term cardiovascular events\(^{(30)}\). Also, the latter authors found that increasing numbers of walls with PSS were related to a lower GLS and so this can illustrate the positive correlation between the GLS and PSI. In this study, the higher value of GLS the worse systolic dysfunction the higher PSI. This study showed the high occurrence of intraventricular dyssynchrony with a longer QRS duration (≥120ms) and this was comparable to other studies \(^{(5,16,23,31,32,33)}\). Previous studies showed a considerable proportion of patients with QRS <120ms who also showed intra-LVD and there was a wide range of variability of intraventricular dyssynchrony prevalence. Also, \(^{(5)}\) found that LV dyssynchrony presented in 29.5% of patients with QRS <120ms. However, \(^{(33)}\) found the
prevalence was 72%. We found a considerable percentage of intraventricular dyssynchrony (about 57%) in patients with QRS <120ms. These findings argue the fact that the QRS duration is not a precise predictor of mechanical (dyssynchrony)\(^{(34)}\). Moreover,\(^{(23)}\) showed that intraventricular (dyssynchrony) could be found in DCM patients with narrow QRS. Despite this finding, several studies demonstrated that even with presence of mechanical (dyssynchrony) in patients with QRS <120ms, were with no or worse response with CRT\(^{(35,36)}\), and many studies suggested that life quality is not improved by CRT despite the presence of (dyssynchrony), if they were with narrow QRS width\(^{(37)}\). LV (dyssynchrony) in these patients with narrow QRS could be due to small areas of fibrosis or scars leads to segmental heterogeneity in LV contraction and may cause dyssynchronous contraction without electrical changes on QRS morphology\(^{(38)}\).

**Ethical Clearance**

The Research Ethical Committee at scientific research by ethical approval of both Environmental and Health and Higher Education and Scientific Research Ministries, Iraq.

**Conflict of Interest**

The authors declare that they have no conflict of interest

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