Relation Of Echocardiographic Ventricular Dyssynchrony With QRS Width On Surface Electrocardiogram

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Citation

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Abstract

Introduction; Heart failure (HF) is the major cause of mortality, morbidity, and hospitalization. Cardiac resynchronization therapy (CRT) significantly improves functional status, quality of life, reduces hospitalizations and decreased mortality in patients with congestive heart failure (CHF). Presence of intraventricular, left ventricular (LV) dyssynchrony, is an important factor determining response to CRT. Objective: To determine the Echocardiographic intra ventricular (Left Ventricle) dyssynchrony and its correlation with QRS width of surface ECG in patient with heart failure. Study Design: Cross-sectional study. Setting: The study was conducted at the National Institute of Cardiovascular Disease (NICVD). Duration Of Study: Six months, from March, 2007 to September, 2007. Patients And Methods: The patients presented to the Echo department through OPD or Ward at NICVD Karachi with severe HF. Informed consent was taken, ECG obtained and after performing the conventional transthoracic echocardiography, parasternal long axis 2D directed M-mode was obtain in left lateral position. Dyssynchrony between septum and posterior wall was measured in millisecond by identifying the peak posterior excursion (contraction) of intraventricular septum and anterior excursion (contraction) of Posterior wall. Result; The study included 100 patients, 85% were males. Among 45 patients of LV dyssynchrony, 80% were diagnosed to have Ischemic Cardiomyopathy (Group-1) and 20% were diagnosed as Non-Ischaemic Cardiomyopathy (Group-2). 68.08 % patients had LV dyssynchrony in Group-W QRS (120-160)ms and 24.52 % patients were in Group-N QRS (80-119) ms. Overall, 55% of our patients had no LV dyssynchrony. Out of these 55 patients, 75.47% were in group N while 31.91% were in group W. Conclusion; We did not find linear correlation between QRS width and dyssynchrony by septal-posterior wall delay (SPWMD) on M mode echocardiography. Patients with narrow QRS complex also have LV dyssynchrony and need echocardiographic criteria to confirm diagnosis.

INTRODUCTION

HF is defining as a complex clinical syndrome characterized bystructural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood[1] HF is the major cause of mortality, morbidity, and hospitalization in patients age ?60 years, and its costs represent 1% to 2% of the global health expenses ($35 billion in the United State.)[2-4]1 American Heart Association. New Medicine Reports 1997; 1999 Heart and Stroke Statistical Update. Dallas, TX: American Heart Association..Despite major advances in medical therapy, morbidity and mortality remain high. In a local study at NICVD all cause mortality was 15.9% of which 18.7% died of CHF[5].Left bundle branch block (LBBB), as evidenced by a widened QRS interval on ECG, is the most common cause of ventricular dyssynchrony in 30% to 50% of patients with systolic HF. Intraventricular conduction delay (IVCD), primarily LBBB pattern, is present in approximately 25% of HF patients. The intraventricular dyssynchrony worsens ventricular function and results in increased myocardial oxygen demand, ventricular remodeling, decreased left ventricle ejection fraction (EF) and increased mortalit[6,7]. Observational echocardiographic studies have clearly demonstrated that the presence of intraventricular LV dyssynchrony is an important factor determining response to CRT[8]. CRT involves pacing multiple ventricles sites simultaneously to allow the ventricle to contract in a synchronized coordinated fashion. CRT corrects inter- and intraventricular conduction delays and maintains Atrio- ventricular (AV) synchrony and reduces the negative hemodynamic effects of asynchrony. This is achieved by rapid ventricular depolarization and subsequent coordinated contraction[9] Overall, restoring the coordinated contractions of the ventricles improves systolic function, EF, and other functional parameters. It also reduces LV end-systolic/diastolic volumes and mitral regurgitation (MR). CRT significantly improves functional status, quality of life, exercise capacity and fewer hospitalizations and decreased mortality in patients with congestive heart

failure[10-12]. Although 70% to 80% of patients with moderate to severe HF respond favorably to CRT. In remaining 20% to 30% of patients treated with CRT do not appear to benefit[13-17]. Historically, patients eligible for CRT were required to have New York Heart Association (NYHA) class III-IV HF, sinus rhythm, and IVCD, defined as QRS interval > 120 msec. However, strong evidence has emerged documenting that QRS duration on surface ECG is a poor predictor of clinical response to CRT and may not accurately reflect the presence or absence of ventricular dyssynchrony and selecting patient for CRT[18,19]. Since then attention has focused on the fact that mechanical dyssynchrony is not necessarily related to electrical dyssynchrony. For instance, it has been shown that significant mechanical dyssynchrony can exist in patients with narrow QRS intervals[13,19,20-22] and may also be noticeably absent in patients with widened complexes[19,21]. American College of Cardiology / American Heart Association guideline recommended CRT only on the basis of QRS width without taking into consideration echocardiographic LV dyssynchrony. Patients with wide QRS complex may be responsible for higher non responder rate for CRT. Ventricular dyssynchrony can be seen in Patients with narrow QRS complex that could potentially have benefited from CRT. In our country, large number of Patients belongs to low socioeconomic class and the cost of CRT is high. In this study, we assessed the relationship between echocardiographic dyssynchrony and QRS width. By this we can reduce the cost for unnecessary expenditure on CRT.

OPERATIONAL DEFINITIONS

What is CHF?

HF is a complex clinical syndrome characterized by impaired myocardial performance and progressive activation of the neuroendocrine system leading to circulatory insufficiency and congestion. The term CHF is often over used to describe HF, but not all patients with HF have sign and symptoms of congestion.

Interventricular dyssynchrony

LBBB is one of the most common causes of ventricular dyssynchrony in systolic HF patients. LBBB slows the electrical conduction to the LV, causing a delay between ventricular contractions, known as interventricular dyssynchrony. The delay between the ventricles results in earlier right ventricular activation in which right ventricular ejection occurs during the LV end-diastolic period. This delay in LV activation primarily affects interventricular septal motion and ultimately has an effect on EF[24]. The primary consequence of interventricular dyssynchrony decreases forward cardiac output by reduced LV filling, decreased septal contribution, and increased functional MR and decreases forward cardiac out put.

Intraventricular dyssynchrony

The delay in LV activation generates area of early and late contraction within the LV[25]. The lack of coordinated LV contraction is termed intraventricular dyssynchrony. Early activation occurs when pressure is still low and no ejection is occurring, and late activation occurs at higher stress and causes the early activated areas to undergo paradoxical stretch. However, the end result is impaired systolic function, reduced cardiac output, and increased end-systolic volume and wall stress[25,26]. Unfortunately, in patients with existing LV dysfunction, the effects of dyssynchrony put additional burden on an already compromised ventricle.

What is CRT?

In this procedure the patient has leads implanted in the right atrium, right ventricle and lateral wall of LV. This LV lead can be placed by endocardial approach where the lead is introduced into the coronary sinus (CS) through right atrium and into a lateral epicardial vein. (See figure 4). It can also be implanted via an open surgical approach and placement of an epicardial lead. This allows LV stimulation at the septum and lateral wall simultaneously negating the effect of delay caused by underlying BBB. CRT can be achieved with a device designed only for pacing or can be incorporated into a combination device with an ICD.

CRT is recommended in patients with advanced HF (usually NYHA class III or IV), severe systolic dysfunction (e.g. LVEF ?35 percent) and IVCD (e.g. QRS >120 msec). CRT improve HF symptoms, exercisecapacity, and LV systolic performance associated with a reductionin rehospitalization for HF and improved long-termsurvival compared with optimized medicaltherapy[11,12]. CRT corrects inter- and intraventricular conduction delays.

Role Of Echocardiography In CRT

Doppler echocardiography has played a crucial role in elucidating the mechanism of reverse structural and functional LV remodeling after CRT. Doppler echocardiography has also been used to identify patients likely to respond to CRT before device implantation. Recently, large CRT trials[10-12] have used serial Doppler

echocardiography to characterize reverse structure and function

Different Techniques For Detection Of Left Ventricular Dyssynchrony

***Septal to Posterior Wall Motion Delay (SPWMD) using M-mode*;**

The first developed and simplest method for evaluating dyssynchrony is the analysis of the activation of the posterior wall compared to the septum using M-mode. Delayed activation of the posterior wall versus the septum is consistent with dyssynchrony. A maximum SPWMD of ?120ms was found to be predictive of reverse remodeling and improvement in HF status, though this has not been seen in all studies. This method is restricted to two opposing walls of the LV and measurement of this time delay may not be possible when the septum is infarcted and does not thicken or move posteriorly in the normal direction.

Pitzalis et al demonstrated that the presence of a delay in posterior wall contraction in comparison with the septum is a useful marker for selecting the patients who benefit most in terms of post-CRT reverse remodeling. In 20 patients with advanced HF caused by Ischaemic cardiomyopathy (ICMP) (n = 4) or non-ICMP (n = 16) and LBBB, LV end-diastolic volume index and LVESV index were calculated before and one month after CRT. This study showed SPWMD 130 ms to predict reduction in LVESV index >15% with a sensitivity of 100% and a specificity of 63% during 1 month[27]. Several Doppler echocardiography methodologies have been advocated for the diagnosis of dyssynchrony.

Pulse wave Doppler measurement:

Pulse wave Doppler measurements at the Left ventricular outflow tract and Right Ventricular out flow tract can provide information about both intraventricular and interventricular dyssnchrony. The aortic preejection time is measured with Pulse wave Doppler echocardiography from the onset of the QRS complex on ECG to the onset of Left ventricular out flow tract flow ejection. It is important to onset of the QRS complex as the reference point for measuring aortic preejection time because it represents the time from electrical activation to the onset of flow through the Left ventricular out flow tract. The aortic preejection time is prolong in the patients who have LV dyssynchrony.

Tissue Doppler imaging (TDI):

TDI is currently the most widely studied method for direct measurement of dyssynchrony[16,28-30]. Doppler echocardiography measures velocity of movement at a defined point within the heart. By measuring the velocities of myocardial movement (tissue Doppler), one can assess the timing of contraction in various segments of the LV. Comparing the timing of contraction in different ventricular segments permits calculation of ventricular dyssynchrony. A variety of measures have been proposed, including the absolute differences in time to peak contraction and the standard deviation of the time to contraction in multiple segments. Furthermore, among 100 patients with TDI evidence of dyssynchrony at baseline, reverse remodeling with CRT only occurred in those whose TDI dyssynchrony assessment improved by at least 20 percent[31].

Rational For Assessing SPWMD Using M-Mode Technique In Pakistan.

Due to limited resources**,** most hospitals in Pakistan don

OBJECTIVE

To determine the Echocardiographic intra ventricular (Left Ventricle) dyssynchrony and its correlation with QRS width of surface ECG in patient with Heart Failure.

DATA COLLECTION PROCEDURE

The patients who fulfilled the inclusion criteria and presented to the Echo department through OPD or Ward at NICVD Karachi with severe HF were selected for the study. The purpose, procedure and risk/ benefits of the study were explained to the patient and informed consent was taken. All patients had 12 lead surface ECG.After performing the conventional transthoracic echocardiography, parasternal long axis 2D directed M-mode will be obtain in left lateral position. Dyssynchrony between septum and posterior wall will be measured in millisecond by identifying the peak posterior excursion (contraction) of IVS and peak anterior excursion (contraction) of Posterior wall.

DATA ANALYSIS: The data feeding and analysis was on computer package SPSS (Statistical Packages of Social Sciences) version 11.0. Frequency and percentage was computed for qualitative variables like sex and age groups. Mean and standard deviation were calculated for quantitative variable age. The relationship between echocardiography dyssynchrony and QRS width was estimated by correlation. Correlation coefficient (r) was also computed with statistical significance. P ? 0.05 was considered level of significance.

RESULTS

This study was conducted on 100 HF patients at NICVD Karachi. The study duration was March, 2007 to September, 2007. **Age:** Among 100 patients of HF, the mean age was 55.71 ? 12.63 years, ranging from 28 to 92 years of age. 69 % of the patients were below 60 years of age. Maximum numbers of patients were found in age group of 41-60 years. Table: 1. **Sex**: There were 85 (85%) males and 15 (15%) females. The female: male ratio was 1: 5.7. Table: 1

Table 1

AGE AND GENDER DISTRIBUTION OF HEART FAILURE PATIENTS

(n = 100)

Age	*Gender*		
	Male	*Female*	*Total*
20-40 years	*10*	*3*	*13*
41-60 years	*47*	*8*	*56*
>60 years	*28*	*4*	*31*
Total	*85*	*15*	*100*
Mean ± SD	*55.7 ± 12.6 years*		
Age Range:	*28 – 92 years*		

The QRS duration (milliseconds) were classified into two groups: Group-N QRS (80-119) ms and Group-W QRS (120-160) ms. (Table 2). Out of 100 patients, 47(47%) were in *group W*QRS and 53(53%) were in g*roup N*QRS**.** Out of 47 patients in group W QRS,32 (68.08%) patients had LV dyssynchrony and 15 (31.91%) had no LV dyssynchrony. In g*roup N* QRS, 13 out of 53 (24.52%) patients had LV dyssynchrony while 40 out of 53 (75.47%) had no LV dyssynchrony**.**The results were found statistically significant when tested by Chi-square test of independence with Chi-sqaure-19.09, p=0.001. (Table 3).

Table 2

DIAGNOSIS WITH QRS DURATION (ms)

(n=100)

		QRS duration in ECG(ms) in groups		
Diagnosis		*Group N (80-119)ms*	*Group W (120-160)ms*	*Total*
	N	*47*	*38*	*85*
	% within diagnosis	*55.3*	*44.7*	*100*
	N	*6*	*9*	*15*
	% within diagnosis	*40.0*	*60.0*	*100*
	N	*53*	*47*	*100*
	% within diagnosis	*53.0*	*47.0*	*100*
ms: milli second		*Chi-Square= 1.197, p=0.27 (n.s.)*		
n.s.: Non- significant				

Table 3

QRS DURATION IN ECG (ms) IN COMPARISON WITH ECHOCARDIOGRAPHY FINDINGS

	ECHOCARDIOGRPAHY FINDINGS		
QRS duration in ECG(ms) in groups	*LV dyssynchrony absent*	*LV dyssynchrony present*	*Total*
Group N (80-119)	*40*	*13*	*53*
Group W (120-160)	*15*	*32*	*47*
Total	*55*	*45*	*100*

Chi-square=19.09, p=0.001

Table 4

AGE & GENDER DISTRIBUTION OF LV DYSSYNCHRONY

			LV Dyssynchrony		
			Dyssynchrony	*Non Dyssynchrony*	
Group1 (20-40)	*sex*	*male*	*5*	*5*	*10*
		female	*2*	*1*	*3*
Total			*7*	*6*	*13*
Group2 (40-60)	*sex*	*male*	*19*	*28*	*47*
		female	*6*	*2*	*8*
Total			*25*	*30*	*55*
Group3 (>60)	*sex*	*male*	*12*	*16*	*28*
		female	*1*	*3*	*4*
Total			*13*	*19*	*32*
Mean □ SD			*56.04 ± 13.5 years*		
Range:			*28 - 92 years*		

Comparison of age = chi – square = 0.66, P = 0.71
Comparison of Gender Fisher exact Test = 0.13, P = 0.11

DISCUSSION

As the experience about CRT has increased, it has shown that a wide QRS complex alone is not a good marker of LV dyssynchrony. Several large randomized trials conducted on CRT had taken a wide QRS (>120m/s) [11, 32, 33] as a major entry criterion in patients with HF with LVEF < 35%. Linde C et al [33] demonstrate first time favorable one year result in exercise tolerance and quality of life by BiV in patient with severe HF and IVCD. In addition improvement in LV function and a decrease in HF related hospitalization were found.

In previous studies [34, 35] patients were considered eligible in the presence of QRS prolongation on surface ECG as the only marker of dyssynchrony, and with no demonstration of ventricular dyssynchrony using techniques.In the past it has been shown that a wide QRS complex increases the likelihood of associated mechanical LV dyssynchrony [11, 12, 36-38]. But in fact the correlation between these is not strong. Some patients with a wide QRS and a severely depressed LVEF may show no area of substantial mechanical delay [19, 21, 39]. Thus the predictive value of the basal QRS complex

duration is poor for responders and non-responders. This also explains why 20-30 % of the patients in the major trials did not have successfulresponse to CRT [13, 14-17].

The relatively high percentage of non responder to CRT (varying from 20% to 30%), however, indicates that additional criteria is required to predict the response to CRT [61, 40]. The presence of LV dyssynchrony has been proposed as an alternative criterion [16]. Leclercq et al [41]and Kass [13] showed that mechanical dyssynchrony in LV contraction determined from the septal to lateral delay in myocardial contraction is not reflected by electrical dyssynchrony (wide QRS complex) on ECG. This study shows that 31.91% patients, who had wide QRS were candidates by conventional echocardiography criterion for CRT, did not have dyssynchrony by SPWMD echocardiographic criterion. These patients may perhaps not benefit from CRT. We know that 20-30 % of patients with wide QRS are non responders. This number is nearly similar (31.91%) to our patients who have no dyssynchrony despite wide QRS complex.

Two studies evaluated the role of QRS complex as a marker of mechanical LV dyssynchrony. In the study of the Bleeker et al [19] severe intra ventricular dyssynchrony (defined as septal to lateral delay > 60 ms on TDI) was observed in 60% to 70% patients with QRS duration > 120 ms. Ghio et alconfirmed the absence of intra ventricular dyssynchrony (defined as presence of one or more difference > 50 ms among the regional pre-ejection period) 29% to 43% of patients with QRS > 120 ms. Otherstudies have also shown that among patients with wide QRS complex (>120ms) a high percentage (30-40%) did not show any evidence of mechanical LV dyssynchrony indicating that a large QRS complex is not a good marker of LV dyssynchrony [19, 32]. The percentage of patients with wide QRS complex not showing LV dyssynchrony is similar (31.91% Vs 30

CONCLUSION

1. We did not find linear correlation between QRS width and dyssynchrony by SPWMD. An isolated wide QRS complex may therefore not be a reliable indicator of LV dyssynchrony.

2. Our study, reported by others, highlights an important fact, that patients with narrow QRS complex may also have LV dyssynchrony. This finding can be potentially beneficial for the some of the patients, deprived of CRT in the past.

3. Large scale randomized studies are recommended to establish the correlation of surface ECG with the Echo/Doppler derived LV dyssnchrony and the clinical impact of CRT.

4 .Robust technologies, in hands of properly trained, conscientious physicians, are essentials, for cost effective use of an expensive procedure, CRT, in public sector of developing country like Pakistan.

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