RESEARCH ARTICLE

Role of Speckle Tracking Echocardiography in Prediction of Responders to Cardiac Resynchronization Therapy

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Abstract

Objective: To assess heart failure patients by speckle tracking echocardiography before cardiac resynchronisation therapy implantation to predict the outcome.

Method: The descriptive follow-up study was conducted at Kafrelsheikh and Mansoura University Hospitals, Egypt, from January 2020 to May 2022, and comprised heart failure patients of either gender who had ejection fraction ≤35%, QRS width ≥130msec and symptomatic even on optimal medical therapy. They were subjected to full clinical evaluation, electrocardiogram, basic and speckle tracking echocardiography with calculation of maximal septal to lateral delay at baseline and 3 months after cardiac resynchronisation therapy implantation. The patients were classified according to response into group I having non-responders, group II having patients who improved clinically only, and group III having responders both in clinical and echocardiographic terms. Data was analysed using SPSS 23.

Results: Of the 38 patients with mean age 55.24±11.23 years, 16(42%) were females and 22(57.9%) were males. There were 7(18.4%) patients in group I, 7(18.4%) in group II and 24(63.2%) in group III. There was a positive significant correlation between response to cardiac resynchronisation therapy CRT and increase in maximal septal to lateral delay at baseline (p<0.01). There was an inverse significant correlation between response to cardiac resynchronisation therapy and decrease in maximal septal to lateral delay 3 months after the implantation (p=0.036).

Conclusion: Maximal septal to lateral delay was found to be a good tool to predict the response to cardiac resynchronisation therapy before implantation.

Keywords: Speckle tracking, Cardiac resynchronisation therapy, Heart failure. **DOI: 10.47391/JPMA.EGY-S4-21**

Introduction

Heart failure (HF) is one of the common causes of coronary care unit (CCU) and intensive care unit (ICU) admission. According to ejection fraction (EF), it can be classified into HF with reduced EF (HFrEF ≤40%), HF with mildly reduced EF (HFmrEF 41-49%) and HF with preserved EF (HFpEF ≥50%).[1] Patients usually present with symptoms and signs of systemic and/or pulmonary congestion.[2] In developed countries, the prevalence of HF ranges 1-2% and >10% among those aged >70 years.[3] Myocardial involvement is usually the primary pathology. However, pathology of other parts of the heart can also lead to HF. So, it is important to detect the primary cause of HF to use the appropriate treatment, like valve replacement or repair for valvular lesions. The cornerstone of managing HFrEF is Beta blockers (BB), Angiotensin converting enzyme inhibitors (ACEI), and mineralocorticoid receptor antagonists (MRA) unless contraindicated or poorly tolerated, and they have shown to improve survival and prevent recurrent hospitalisation.[5] In addition to those drugs, angiotensin receptor-neprilysin inhibitor (ARNI) sacubitril-valsartan was introduced in the last few years as an important cornerstone drug in the management of HFrEF. It combines valsartan and sacubitril (a neprilysin inhibitor) and in order to decrease the risk of HF hospitalisation and death, it is currently advised to replace ACEIs in patients with HFrEF.[6] Sodium-glucose co-transporter 2 inhibitors (SGLT2-I), dapagliflozin and empagliflozin have recently been recommended for patients with HFrEF also to reduce the risk of death and recurrent hospitalisation.[7] In patients with signs and/or symptoms of congestion, diuretics should be added to the drug regimen. Restoring electro-mechanical synchronisation between the walls of the left ventricle (LV) to enhance LV function, and, as a result, increase functional capacity in HF patients who have electro-mechanical dyssynchrony is possible with cardiac resynchronisation therapy (CRT).[8] It is an option for symptomatic individuals with HFrEF who have received appropriate medical treatment for at least 3 months, and yet have EF ≤35% and QRS width ≥130ms.[9] Unfortunately, not all patients who undergo CRT implantation will respond to the treatment modality; 60-70% patients are responders, 30-40% are either non-responders or negative responders post-CRT implantation.[10] CRT response is defined as at least one-step improvement in New York Heart Association (NYHA) class, and decrease in end systolic volume (ESV) ≥15% of its pre-existing value 3-6 months after the implantation.[11] It is crucial to find a non-invasive imaging parameter that may

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predict response pre-implantation. Strain and strain rate can be measured on an offline software after good acquisition of images and tracking the movement of speckles during systole and diastole.[12]

The maximum time delay between the earliest and latest segments of the six segments obtained from longitudinal strain of the apical four chamber view can be utilised to determine the CRT response, which is known as maximal septal to lateral delay.[13]

The current study was planned to assess HF patients by speckle tracking echocardiography before CRT implantation to predict the outcome.

Patients and Methods

The descriptive, follow-up study was conducted at Kafrelsheikh and Mansoura University Hospitals, Egypt, from January 2020 to May 2022. After approval of the institutional ethics review committee of the two hospitals, non-probability convenient sampling technique was used and the sample was raised from HF patients who had sinus rhythm with QRS width ≥130 msec and left bundle branch block (LBBB) morphology and EF ≤35% who were symptomatic even after 3 months of medical therapy and were due to undergo CRT implantation. Informed consent was taken from all the patients, and those not willing to participate were excluded.

All patients were subjected to comprehensive evaluation at baseline and 3 months post-intervention. Clinical assessment included electrocardiogram (ECG) as well as basic and advanced echocardiographic parameters. (Epic 7c Philips with S5-1 transducer). Basic and tissue doppler parameters measured included septal e` (Sep e`), lateral e` (Lat e`), E wave (E), average E/e`, E/A, tricuspid annular plane systolic excursion (TAPSE) and right ventricular peak systolic velocity (RV S`). Left ventricular global longitudinal strain (GLS) was measured using offline software from apical 2-chamber, 3-chamber and 4-chamber views. Peak systolic GLS is the average of the 17 LV segments obtained. Maximal septal to lateral delay between 6 segments was also calculated as the maximum time delay between earliest and the latest segments of septal and lateral walls in apical 4-chamber basal, mid and apical views and used as a predictor of CRT response.

According to change (Δ) in NYHA functional class (14) and ESV before and after CRT, the response was determined and the patients were divided into group I having responders (ΔNYHA class 0 and ΔESV <15%), group II having patients who improved clinically only (ΔNYHA class ≥1 and ΔESV <15%) and group III having responders both in clinical and echocardiographic terms (ΔNYHA class ≥1 and DESV ≥15%).

Data was analysed using SPSS 23. Quantitative data was presented as mean, standard deviations and ranges when the variables were parametric, and as median and interquartile range (IQR) when they were non-parametric. Frequencies and percentages were used to present qualitative data. Chi-square test was used to compare qualitative data among the groups. One-way analysis of variance (ANOVA) was used to compare the means of more than two groups using quantitative data and parametric distribution. Receiver operating characteristic (ROC) curve was drawn between responders and non-responders. P<0.01 was considered highly significant (HS), p<0.05 significant (S) and p>0.05 non-significant (NS).

Results

Of the 38 patients with mean age 55.24±11.23 years, 16(42%) were females and 22(57.9%) were males. There were 7(18.4%) patients in group I, 7(18.4%) in group II and 24(63.2%) in group III.

There was a positive significant correlation between response to CRT and increase in maximal septal to lateral delay at baseline (p<0.01). There was an inverse significant correlation between response to CRT and decrease in maximal septal to lateral delay 3 months post-implantation (p=0.036).

NYHA class, end diastolic dimension (EDD), end systolic dimension (ESD), left atrial (LA) dimension, EF, GLS, maximal septal to lateral delay, E wave velocity, E/A, Sep e` velocity, average E/e`, TAPSE, tricuspid (TR) velocity showed a highly significant difference between baseline and follow-up values (P<0.01). RV S` velocity had a significant difference (p=0.039). A cut-off value of 176 msec for maximal septal to lateral delay before CRT implantation with a very good area under curve (AUC) of 0.889 was determined between non-responders and responders, indicating that a patient was likely to be a responder when Sep to Lat delay ≥176 msec with sensitivity 0.875 and specificity 0.75.

A positive significant correlation was detected between response to CRT and DNYHA class, DESV, EF follow-up (FU), GLS FU, increase in maximal Sep to Lat delay, TR velocity FU, ESD FU, LA, LA FU, average E/ e` and TAPSE FU (p<0.05). There was an inverse significant correlation between response to CRT and decrease in maximal Sep to Lat delay FU (p=0.036) (Table 1, Figure). No significant correlation was detected between response to CRT and age, gender, EDD, EDD FU, ESD, EF, GLS, E velocity, E velocity FU, E/A, E/A FU, average E/e`, Sep e`, Sep e` FU, Lat e`, Lat e` FU, TAPSE, RV S`, RV S` FU and TR velocity (Tables 1-2).

Table-1: Comparison between the study groups.

		(Group I) No. = 7	(Group II) No. = 7	(Group III) No. = 24	Test value•	*p-value*	Sig.
Δ NYHA class	0	7 (100.0%)	0 (0.0%)	0 (0.0%)	40.000*	0.000	HS
	I	0 (0.0%)	7 (100.0%)	24 (100.0%)			
EDD (cm)	Mean ± SD	7.60 ± 0.55	7.43 ± 1.08	7.01 ± 0.89	1.669	0.202	NS
	Range	6.7 – 8.4	5.2 – 8.6	5.5 – 8.8			
EDD FU (cm)	Mean ± SD	7.49 ± 0.49	7.29 ± 1.00	6.80 ± 0.88	2.457	0.100	NS
	Range	6.7 – 8.2	5.3 – 8.5	5.3 – 8.6			
ESD (cm)	Mean ± SD	6.75 ± 0.54	6.55 ± 0.94	6.07 ± 0.86	2.481	0.098	NS
	Range	5.8 – 7.4	4.6 – 7.7	4.6 – 7.7			
ESD FU (cm)	Mean ± SD	6.59 ± 0.52	6.26 ± 0.83	5.66 ± 0.81	5.104	0.011	S
	Range	5.7 – 7.1	4.6 – 7.3	4.2 – 7.2			
Δ ESV %	Mean ± SD	3.35 ± 2.12	10.67 ± 3.14	18.84 ± 3.70	69.409	0.000	HS
	Range	0.9 – 6.8	5.7 – 13.9	15.1 – 27.4			
EF (%)	Mean ± SD	22.59 ± 5.60	22.29 ± 2.20	25.46 ± 5.02	1.962	0.155	NS
	Range	15 – 30.4	19.4 – 25.9	19.3 – 34.5			
EF FU (%)	Mean ± SD	23.75 ± 5.26	28.31 ± 2.83	33.56 ± 4.82	14.811	0.000	HS
	Range	16.1 – 31.1	24.1 – 32.5	27.5 – 45.1			
GLS (%)	Mean ± SD	-5.86 ± 3.14	-6.54 ± 2.24	-7.15 ± 1.89	1.049	0.360	NS
	Range	-11.6 – -3.2	-10.6 – -4.2	-10.7 – -4.3			
GLS FU (%)	Mean ± SD	-6.95 ± 3.17	-9.58 ± 2.36	-11.09 ± 1.71	10.919	0.000	HS
	Range	-13 – -4.3	-13.1 – -5.3	-14.2 – -8.6			
Sep-Lat delay (msec)	Mean ± SD	167.13 ± 22.96	185.75 ± 17.09	230.42 ± 40.47	12.512	0.000	HS
	Range	117 – 198	165 – 210	143 – 295			
Sep-Lat delay FU (msec)	Mean ± SD	140.63 ± 14.85	133.38 ± 9.46	125.21 ± 15.72	3.640	0.036	S
	Range	107 – 155	120 – 146	80 – 149			
LA (Cm)	Mean ± SD	5.33 ± 1.27	4.83 ± 0.57	4.56 ± 0.47	3.504	0.040	S
	Range	4.4 – 8.3	3.9 – 5.6	3.8 – 5.6			
LA FU (cm)	Mean ± SD	5.31 ± 1.28	4.76 ± 0.53	4.50 ± 0.48	3.963	0.028	S
	Range	4.4 – 8.3	3.9 – 5.5	3.8 – 5.6			

P-value >0.05: Non-significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS)

*: Chi-square test; •: One-way analysis of variance (ANOVA) test.

Abbreviations: Δ NYHA class: Change in class of New York Heart Association classification, EDD: End diastolic dimensions, EDD FU: End diastolic dimensions follow-up, ESD: End systolic dimensions, ESD FU: End systolic dimensions follow-up, Δ ESV%: Percent of decrease in end systolic volume, EF: Ejection fraction, EF FU: Ejection fraction follow-up, GLS: Global longitudinal strain, GLS FU: Global longitudinal strain follow-up, Sep-Lat delay: Maximal time delay between septal and lateral segments, Sep-Lat delay FU: Maximal time delay between septal and lateral segments follow-up, LA: Left atrial dimeter, LA FU: Left atrial dimeter follow-up.

Table-2: Comparison between the study groups

		(Group I) No. = 7	(Group II) No. = 7	(Group III) No. = 24	Test value•	*p-value*	Sig.
E wave (cm/s)	Mean ± SD	112.30 ± 47.92	101.88 ± 47.85	93.01 ± 30.45	0.810•	0.453	NS
	Range	52.3 – 180	47.8 – 195	42.3 – 162.9			
E wave FU (cm/s)	Mean ± SD	100.79 ± 46.66	97.54 ± 47.59	71.06 ± 23.92	3.089•	0.058	NS
	Range	50.7 – 174.6	54.8 – 188	21.9 – 110			
E/A	Mean ± SD	2.13 ± 1.11	1.73 ± 1.31	1.75 ± 1.06	0.517‡	0.772	NS
	Range	0.6 – 3.4	0.4 – 3.7	0.5 – 3.9			
E/A FU	Mean ± SD	1.81 ± 0.85	1.60 ± 1.18	1.25 ± 0.72	3.052‡	0.217	NS
	Range	0.6 – 3	0.6 – 3.7	0.4 – 2.6			
Sep e` wave (cm/s)	Mean ± SD	4.91 ± 1.22	5.51 ± 2.39	4.84 ± 1.00	0.695	0.505	NS
	Range	3.1 – 7.2	3.3 – 10.8	3.1 – 6.8			
Sep e` wave FU (cm/s)	Mean ± SD	4.99 ± 1.48	5.83 ± 1.46	5.64 ± 1.35	0.856	0.433	NS
	Range	3.2 – 7.2	3.6 – 8.3	4 – 9.7			
Lat e` wave (cm/s)	Mean ± SD	6.08 ± 0.95	7.68 ± 2.65	6.46 ± 1.36	2.236	0.121	NS
	Range	4.8 – 7.3	4.6 – 11.5	4.2 – 9.3			

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Table-2: *continued from previous page*

		(Group I) No. = 7	(Group II) No. = 7	(Group III) No. = 24	Test value•	*p-value*	Sig.
Lat e` wave FU (cm/s)	Mean ± SD	5.99 ± 1.05	7.83 ± 2.48	6.88 ± 1.80	1.999	0.150	NS
	Range	4.2 – 7.4	4.5 – 11.5	4 – 12.3			
Average E/e`	Mean ± SD	20.18 ± 8.00	16.25 ± 7.88	16.35 ± 5.73	1.069•	0.354	NS
	Range	8.4 – 32	9.2 – 32.5	4.8 – 29			
Average E/e` FU	Mean ± SD	18.48 ± 7.06	14.54 ± 6.80	11.95 ± 4.47	4.272•	0.021	S
	Range	8.1 – 26.2	8.7 – 30	3.2 – 21.3			
RV S` (cm/s)	Mean ± SD	9.88 ± 2.44	10.75 ± 2.20	10.66 ± 1.64	0.568•	0.572	NS
	Range	8 – 15.7	8.8 – 14.6	6.6 – 14.2			
RV S` FU (cm/s)	Mean ± SD	9.96 ± 2.17	10.59 ± 1.63	11.33 ± 2.08	1.507•	0.235	NS
	Range	8 – 15	9 – 13.9	6.8 – 15.9			
TAPSE (cm)	Mean ± SD	1.64 ± 0.37	1.84 ± 0.33	1.92 ± 0.30	2.285•	0.116	NS
	Range	1.3 – 2.4	1.4 – 2.3	1.2 – 2.4			
TAPSE FU (cm)	Mean ± SD	1.69 ± 0.33	1.89 ± 0.27	2.02 ± 0.30	3.711•	0.034	S
	Range	1.4 – 2.4	1.5 – 2.3	1.2 – 2.6			
TR velocity (m/s)	Mean ± SD	3.24 ± 0.26	3.05 ± 0.69	2.76 ± 0.53	2.809•	0.073	NS
	Range	2.9 – 3.8	2.5 – 4.6	2 – 3.96			
TR velocity FU (m/s)	Mean ± SD	3.14 ± 0.23	2.89 ± 0.71	2.53 ± 0.44	5.661•	0.007	HS
	Range	2.9 – 3.6	2.4 – 4.5	1.9 – 3.3			

P-value >0.05: Non-significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS)

•: One-way analysis of variance (ANOVA) test; ‡: Kruskal Wallis test

E wave: Peak of early diastolic wave, E wave FU: Peak of early diastolic wave follow-up, A wave: Peak of late diastolic wave, A wave FU: Peak of late diastolic wave follow-up, Sep e`: Septal e` wave, Sep e` FU: Septal e` wave follow-up, Lat e`: Lateral e` wave, Lat e` FU: Lateral e` wave follow-up, RV S`: Right ventricular peak systolic velocity, RV S` FU: Right ventricular peak systolic velocity follow-up, TAPSE: Tricuspid annular plane systolic excursion, TAPSE FU: Tricuspid annular plane systolic excursion follow-up, TR velocity: Tricuspid regurge peak systolic velocity, TR velocity FU: Tricuspid regurge peak systolic velocity follow-up.

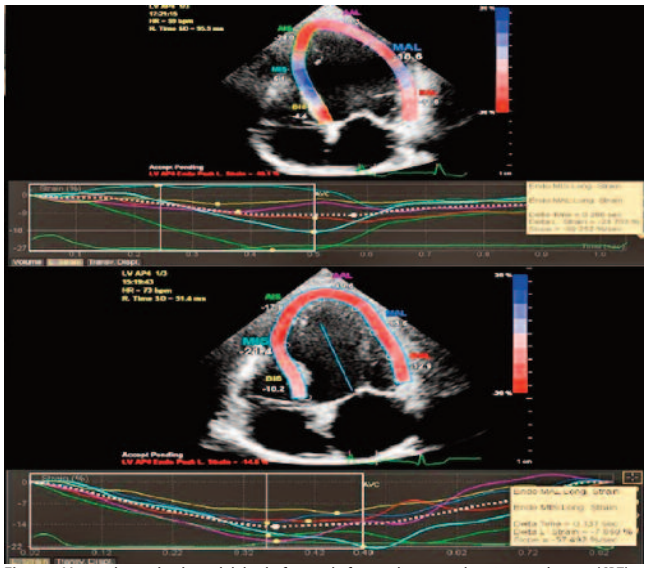

Figure: Maximal septal to lateral delay before and after cardiac resynchronisation therapy (CRT) in a patient of the responder group.

Discussion

CRT is an option for those on optimal medical therapy and still symptomatic, but, unfortunately, not all patients respond positively to it, indicating a need to find a parameter that may predict the response before CRT implantation. The current study used speckle tracking echocardiography as a predictor of response to CRT.

The study had 38 patients and there was no significant difference in terms of age amd gender in relation to response to CRT (p=0.118 and p=0.085).

Ghani A et al.[13], Sugano A et al.[15] and Galli E et al.(16) reported similar findings. Galli E et al.[16] presented findings similar to those of the current study regarding EDD, ESD, EF and GLS. Sugano A et al.[15] presented findings regarding NYHA class and EF that were similar to those of the current study. The current findings were also in agreement with Ghani A et al.[13] with respect to Sep to Lat delay before and after CRT implantation in responders and non-responders. Galli E et al.[16] and Ghani A et al.[13] presented findings that were different from the current findings related to NYHA class and response to CRT which may be due to strict inclusion criteria of the current study and a relatively small number of patients. Th current results were in line with literature[13,16,17] related to baseline EF and response to CRT. Ghani A et al.[13] reported findings similar to the current ones in which responders had more baseline maximal Sept to Lat delay, but the result were not similar with respect to maximal Sep to Lat delay FU values.

The current results were also in contrast to the findings of Galli E et al.[16] with respect to GLS and response to CRT. This also may be attributed to the strict inclusion criterion of the

current study and a relatively small number of patients.

Conclusion

Maximal Sep to Lat delay derived from longitudinal strain in apical 4-chamber view was found to be an effective tool to predict response to CRT before implantation.

Limitation: The current study has its limitations as the sample size was not calculated and we depended on non-probability convenient sampling technique and the number of studied patients were the patients who were admitted to our hospital during the period of the study and the sample was small which could have a negative effect on the power of the study. The reason was limitations of movement owing to the coronavirus disease-2019 (COVID-19) pandemic.

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