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ORIGINAL ARTICLE

Validity of Baseline and Sequential Global Longitudinal Strain in Prediction of Anthracyclines Induced Cardiotoxicity

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ABSTRACT

Background: Hematologic cancers and solid tumors are frequently treated with anthracyclines. In approximately 9% of patients, they may cause cardiac dysfunction associated to cancer therapy (CTRD). Two-dimensional speckle tracking echocardiography (STE) with global longitudinal strain (GLS) and left atrial strain (LAS) have been developed as a significant and established tools for predicting subsequent CTRD during anthracycline therapy. CTRD is traditionally defined as a decreased left ventricular ejection fraction (LVEF) of less than 50% or ≥ 5% absolute reduction in symptomatic patients or ≥10% in asymptomatic patients.

Methods: A cohort prospective study was carried out in the Cardiology department from February 2023 to May 2024. Prior to receiving chemotherapy, 143 patients had evaluations. Serial echocardiography was carried out at 0, 3, 6, and up to a year's follow-up, with 2D-STE conducted at 0 and 3 months.

Results &Conclusion:

This one-year cohort research comprised 143 patients receiving anthracycline chemotherapy (mean age of 56 years, 77.6% women). We evaluated the predictive relevance of LV GLS, LAs, brain natriuretic peptide (BNP), and troponin (TnT). Cardiotoxcity was consistently associated with proportionate reductions in LV GLS, LASct, and LASr, as well as 3-month increases in BNP levels. Multivariate Cox Regression Analysis showed that at 3 months after treatment, the development of cardiotoxicity was highly accurately and independently linked with Δ LV GLS ≥ 18.4%.

Keywords: Anthracycline;Left atrial strain; Global longitudinal strain ;Left ventricle ejection fraction

INTRODUCTION

Anthracyclines are commonly used to treat solid tumours and hematologic malignancies. However, in about 9% of patients, anthracyclines can cause cancer therapy-related cardiac dysfunction (CTRD), which is usually identified in individuals who are monitored during the first year of their medication. Reducing cardiac function (e.g., LVEF 55%, pre-existing ischemic or valvular sickness), age over 60, cardiovascular risk factors, high cumulative dose (e.g., doxorubicin 250 mg/m2), or any dosage in conjunction with radiation therapy in the heart field are recognized risk factors [1]. Nevertheless, anthracycline-induced

cardiomyopathy occurs in some people who do not have these high-risk characteristics.

These patients may be identified earlier if subclinical myocardial impairment is detected, which would enable closer clinical surveillance both during and after anthracycline therapy and before possibly cardioprotective therapies are started. CTRD is commonly defined as a decreased left ventricular ejection fraction (LVEF) <50 or ≥ 5% absolute drop in symptomatic patients or ≥10% in asymptomatic patients. An important and well-established methods have evolved for predicting the occurrence of CTRD after anthracycline therapy such as two-dimensional left atrial strain (LAs) and global longitudinal strain (GLS) speckle tracking echocardiogram (STE) [2–6]. Baseline GLS before to starting anthracycline medication is predictive of left ventricular systolic dysfunction or significant adverse cardiac events, such as cardiac mortality or symptomatic heart failure, according to recent retrospective investigations of individuals with haematological malignancies [7-9].

METHODS

Study design

This cohort prospective study was conducted in our cardiovascular department, Zagazig university from February 2023 to May 2024.

The ethical committee at our university gave it its approval (IRB approval number:10997-13-8-2023). The involved patient provided written consent. Following the inclusion criteria, 143 individuals were found to be eligible: male and female patients over the age of 18 and patients with newly-diagnosed malignancies of any kind for which cardiotoxic chemotherapeutic drugs were anticipated to be provided in their treatment regimens. Patients with diabetes, rheumatoid arthritis, congenital ischemic heart disease, hypertensive patients, patients with metabolic, systemic disorders that affect the heart, and patients on any other cardiotoxic therapeutic medications were among the exclusion criteria. A thorough medical history and clinical examination were performed on the patients. Renal function, BNP levels, and troponin levels were calculated.

Patients were evaluated before initiating anthracycline therapy, every 3 months during the first year of anthracycline treatment by transthoracic echocardiography . 2D Speckle tracking echocardiography was performed to included patients before initation of anthracycline and after 3 months from therapy .

Anthracyclines induced cardiotoxicity is commonly defined as a decreased left ventricular ejection fraction (LVEF) <50 or ≥ 5% absolute drop in symptomatic patients or ≥10% in asymptomatic patients [2].

A comprehensive echocardiographic examination was performed using a siemens ultrasound machine equipped with an M5S-D or M5Sc-D transducer that complies with consensus guidelines and recommendations[10]. The biplane disc summation approach was used to calculate the left ventricular (LV) end-diastolic and end-systolic volumes in the 4- and 2-chamber apical views, allowing the two-dimensional LVEF to be determined.

The longitudinal LV and LA stresses were analyzed using commercial software (siemensUltrasound, and Automated Function Imaging [AFI] LV and LA).To guarantee accurate analysis, the frame rate of every image was kept at ≥ 50Hz/s. The programme automatically recognised the region of interest and tracked the speckle motion in succeeding frames after determining the endocardial

border in each view. There were three common LV apical views used [11].

. LA strain study allows for the measurement of LA phasic function as well as the early identification of LA dysfunction. We calculate strain on LA in three stages: conduit (LAScd), contraction (LASct), and reservoir (LASr). For all LA strain experiments, the zero strain reference was chosen during LV end-diastole in compliance with guidelines. Using the method ([strain value at baseline−strain value at 3 months]/[strain value at baseline]×100), we were able to determine the percentage changes in the strain values for the LV GLS and LA. The absolute value of the strain parameters was disclosed by the percentage variation (relative change) in the strain values; negative and positive changes, respectively, indicated deteriorating and improving deformations. [12].

STATISCAL ANALYSIS

Statistical Package for the Social Sciences (SPSS) version 22 database software was used to code, enter, present, and analyse the gathered data [13]. Frequencies and percentages were used to depict qualitative data. For quantitative variables, the median was calculated for data that was not normally distributed, and the mean and standard deviation were calculated for data that was regularly distributed. Tests called chi-square (X2) were utilised to find relationships between various qualitative variables. If the cell anticipated value is less than 5, Fisher's exact test was performed. The difference between the quantitative variables in the two groups' normally distributed data was calculated using the Independent T test. When dealing with non-normally distributed data, the Mann-Whitney test was utilised to determine the difference between quantitative variables in two groups.

The survival function created by Cox's proportional hazards regression model indicates the likelihood that a specific event will occur at a given period. The receiver operating characteristic (ROC) curve was analyzed in order to determine the cutoff values that correspond to a particular occurrence. The significance level for each of the statistical tests indicated above has been established. The significance criterion is the 5% level (p value), where a p value of ≤ 0.05 indicates significant results and a p value of > 0.05 indicates non-significant results (NS).

RESULTS

This one year cohort study included 143 patients receiving anthracycline-containing chemotherapy (77.6 % women , mean age 56 years).

Table (1) explained the clinical and echocardiographic parameters of the cohort. Prior to participation, all patients had maintained left ventricular contractility (LVEF >55%) and were in sinus rhythm for a minimum of three months. Nineteen patients (13.2%) had cardiotoxicity (reduction of LVEF ≥10 or <50%) throughout the 1-year follow-up.

Baseline characteristics

Patients without cardiotoxicity and those with it did not differ in demographic information or anthracycline dosage. Nevertheless, patients with cardiotoxicity had higher baseline TnT levels, and baseline BNP levels were not different. Of the echocardiographic variables analyzed, there was no difference between the two groups in terms of LVEF, E/A ratio, septal and lateral e´ velocities, LV GLS, and LA Sr **(Table 1).**

Echocardiographic parameters and biomarkers at three months

Three months after starting anthracycline regimen, the biomarkers and echocardiographic parameters were presented

in Table 2. It was shown that patients with cardiotoxicity had greater BNP levels. LV GLS, LASct, and LASr were the echocardiographic parameters that were more dramatically decreased in patients who had cardiotoxicity. At the 3-month follow-up, individuals with cardiotoxicity showed more marked relative declines in LV GLS, LASct, and LASr than did patients without cardiotoxicity.

Predictors of cardiotoxicity

We evaluated the strains of TnT, BNP, LV GLS, and LA for their predictive relevance. Cardiotoxicity was univariably associated with proportionate declines in LV GLS, LASct, and LASr, as well as 3-month BNP levels as showed in **Table (3).** Three months after chemotherapy, there was an independent correlation between the development of cardiotoxicity and Δ LV GLS, as demonstrated by the multivariate cox regression analyses presented in **Table (4).**

ROC analysis as **Figure (1)** and **Table (5)** show that the greatest discriminating values for early cardiotoxicity were ΔLV GLS ≥18.4% at 3 months, with high sensitivity of 89% and high specificity of 77%.

Kaplan–Meier curve analysis of cardiotoxicity as described in **Figure (2)** showed the survival probability from cardiotoxicity. The incidence of cardiotoxicity after chemotherapy was significantly different among the two GLS change groups (divided by cutoff value :18.4 ,log-rank test =33.84, p = <0.001).

Table 1: Baseline clinical and echocardiographic characteristics of all patients and of those with and without cardiotoxicity

	All (n=143)	No Cardiotoxicity (n=124)	Cardiotoxicity (n=19)	P value
Age , years	56.55 ±8.56	56.10 ±8.14	59.53 ±10.68	0.10
Female %	111 (77.6)	97 (78.2)	14 (73.7)	0.65
BSA (m2)	1.55±0.06	1.55 ±0.07	1.53±0.05	0.31
HTN	62 (43.4%)	53 (42.7)	9 (47.4)	0.70
Systolic Blood pressure	130.70±14.67	130.08±14.73	134.74±13.99	0.19
Diastoic blood pressure	78.81 ±10.63	78.43 ±10.16	81.32 ±13.31	0.27
DM	56 (39.2%)	45 (36.3)	11 (57.9)	0.07
Obesity	29 (20.3%)	26 (21)	3 (15.8)	0.60
Smoking	24 (16.8%)	18 (14.5)	6 (31.6)	0.06
eGFR (mi/min/1.73 m2)	93.26±16.6	94.21 ±16.7	87.05 ±14.7	0.08
Previous CVD	26 (18.2%)	20 (16.1)	6 (31.6)	
Baseline medication				
ACEI	25 (17.5%)	20(16.1)	5 (26.3)	0.16
B Blocker	24 (16.8%)	19 (15.3)	5 (26.3)	
Statin	8 (5.6%)	6 (4.8)	2(10.5)	
No medication	86 (60.1)	79 (63.7)	7 (36.8)	
Oncological disease				
Breast cancer	77 (53.8%)	66 (53.2)	11 (57.9)	0.79
Leukemia	32 (22.4%)	27 (21.8)	5 (26.3)	

	All (n=143)	No Cardiotoxicity (n=124)	Cardiotoxicity (n=19)	P value
Ovarian tumour	7 (4.9%)	6 (4.8)	1 (5.3)	
Lymphoma	27 (18.9%)	25 (20.2)	2 (10.5)	
Oncological treatment				
AC-Taxen	53 (37.1)	47(37.9)	6 (31.6)	
Adriamycin-Vincristine-prednisone	32 (22.4)	27 (21.8)	5 (26.3)	
AC-Taxen-Trastuzumab	24 (16.8)	19 (15.3)	5 (26.3)	0.46
Adriamycin-endoxan	8 (5.6)	6 (4.8)	2 (10.5)	
CHOP	20 (14)	19 (15.3)	1 (5.3)	
ABVD	6 (4.2)	6 (4.8)	0	
cumulative docurubicib dose, mg/m2	443.36±65.75	445.97±62.54	426.32±83.81	0.22
HER2 targeted therapy	39 (27.3)	32 (25.8)	7 (36.8)	0.22
Baseline Biomarkers				
BNP ,pg/ml	22.94 ±6.64	22.66 ±6.66	24.76±6.36	0.20
Hs TnT, ng/ml	11.07± 4.37	10.96 ± 4.31	11.78± 4.77	0.44
Baseline Echocardiography				
LVEDV ,ml	84.30± 7.7	84.22 ±7.31	84.84±10.51	0.74
LVESV ,ml	30.61±3.2	30.52±3.30	31.21±2.61	0.38
LVEF %	63.87±2.7	64.01±2.68	63.00±3.14	0.13
E/A	0.96 ±0.22	0.97±0.21	0.89 ±0.30	0.18
Septal ē cm/s	7.53±1.4	7.56±1.56	7.35±0.95	0.56
Lateral ē cm/s	9.66±1.3	9.65±1.41	9.77±0.54	0.71
Average E/ē	7.27 ±0.8	7.23±0.7797	7.53±1.24	0.16
LAVI (ml/m2)	25.98 ±3.1	26.00±3.00	25.84 ±4.34	0.84
LV GLS %	-19.09 ±2.4	-19.10 ±2.63	-19.00 ±1.39	0.86
LA Sr %	-27.53±3.1	-27.54±3.06	-27.48±3.92	0.94
LASct %	-14.13±1.6	-14.07±1.61	-14.49±1.47	0.28
LAScd %	-15.32±2.5	-15.33 ±2.61	-15.24±2.45	0.88

Data are expressed as n (%), mean±SD, LV GLS ,left ventricular global longitudinal strain . LA strains left atrium strain . ACEI/ARB, angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker; BMI, body mass index; BNP, B-type natriuretic peptide; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; HER2, human epidermal growth factor receptor 2; LAScd, left atrial strain during conduit phase; LASct, left atrial strain during contraction phase; LASr, left atrial strain during reservoir phase; LAVI, left atrial volume index; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume; LV GLS, left ventricular global longitudinal strain; TnT, troponin T.

Table 2: Biomarkers and echocardiographic parameters at 3 months

	All (n =143)	No cardiotoxcity n=124	Cardiotoxicity n=19	P value
Biomarker (at 3 months)				
BNP ,pg/ml	29.53 ±10.6	28.29 ± 8.78	37.65± 17.09	<0.001
Hs TnT, ng/ml	15.18 ±6.15	14.84± 5.81	17.43±7.85	0.08
Follow up echo (3 months)				
LVEDV ,ml	86.99±7.02	86.85±7.32	87.89± 4.71	0.54
LVESV ,ml	31.71±3.89	31.56±3.64	32.63± 5.26	0.26
LVEF %	62.11±2.9	62.28± 2.92	61.00± 2.74	0.07
E/A	0.96±0.20	0.97 ±0.21	0.90±0.11	0.17
Septal ē cm/s	7.43±1.05	7.48 ±0.98	7.13 ±1.41	0.18
Lateral ē cm/s	8.91±1.53	8.99±1.42	8.42 ±2.05	0.13
Average E/ē	7.83±1.21	7.77 ±1.07	8.25±1.87	0.10
LAVI (ml/m2)	27.29±3.3	27.13±3.31	28.32±3.41	0.15
LV GLS %	-16.92 ±3.15	-17.53±2.88	-12.98 ±1.70	<0.001
ΔLVGLS	10.85±14.5	7.73±12.35	31.24±10.68	<0.001
LA Sr %	-26.32±3.48	-26.60±3.36	-24.51 ±3.80	0.01
ΔLASr	3.94 ±10.8	2.94 ±10.76	10.50 ±9.12	<0.001
LASct %	-13.16± 1.37	-13.24 ±1.34	-11.63 ±1.48	0.02
ΔLA Sct	12.93±13.05	12.001±12.93	19.02±12.45	0.02
LAScd %	-14.17 ±2.23	-14.28±2.20	-13.39 ±2.34	0.10
Δ LA Scd	5.74±16.91	4.98 ±16.84	10.70 ±16.95	0.18

Data are expressed as n (%), mean±SD,). Δ is the relative reduction between baseline measurement and measurement at 3 months after anthracycline administration. Abbreviations as in Table 1.

Table (3) Univariate Cox proportional hazard model analysis of cardiotoxicity predictors:

Variable	OR (95% CI)	
BNP (follow up)	1.08 (1.13 - 1.04)	<0.001
Troponin (follow up)	1.05 (1.12 -0.98)	0.10
LV GLS (follow up)	2.15 (2.98 -1.54)	<0.001
ΔLV GLS	1.11 (1.15 -1.07)	<0.001
LASr (follow up)	1.20 (1.42 -1.02)	0.025
Δ LASr	1.05 (1.09 -1.01)	0.007
LASct (follow up)	1.40 (2.04 - 0.97)	0.07
Δ LASct	1.04 (1.08 -1.00)	0.03
LAScd (follow up)	1.21 (1.51 - 0.96)	0.09
ΔLAScd	1.02 (1.04 - 0.99)	0.15

Δ is the relative reduction between baseline measurement and measurement at 3 months after anthracycline administration. CI, confidence interval; OR, odds ratio. Other abbreviations as in Table 1

Table 4: Multivariate Cox proportional hazard model analysis of cardiotoxicity predictors :

Variable	OR (95% CI)	
Δ LV GLS	1.07 (1.13 -1.01)	0.02
GLS (follow up)	1.28 (1.91 - 0.85)	0.22
Δ LASr	1.02 (1.06 - 0.97)	0.38
Δ LASct	1.02 (1.06 - 0.98)	0.25
BNP (follow up)	1.03 (1.07- 0.99)	0.08

Δ is the relative reduction between baseline measurement and measurement at 3 months after anthracycline administration. CI, confidence interval; OR, odds ratio. Other abbreviations as in Table 1

Table 5:Performance of Δ LV GLS in prediction of early cardiotoxicity:

Cutoff point	AUC	Sensitivity	Specificity	P
18.4 %	0.91	89	77	<0.001

AUC ,area under curve

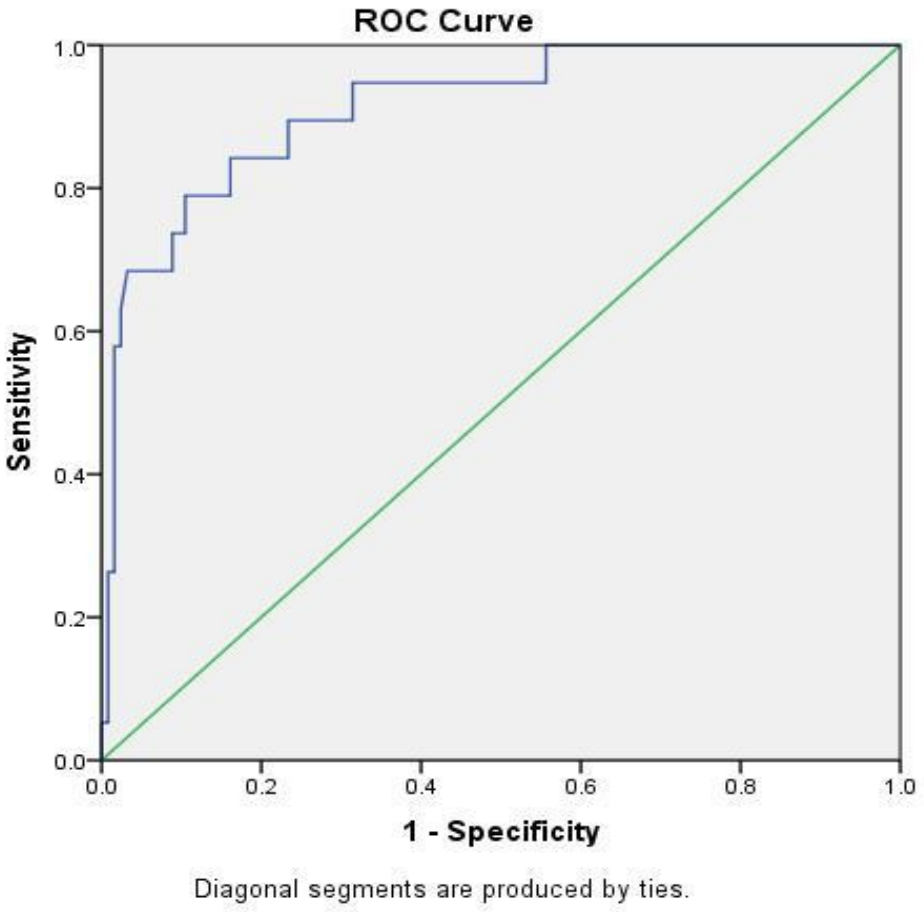

Figure (1):ROC Curve showing performance of LV GLS change in prediction of early cardiotoxicity

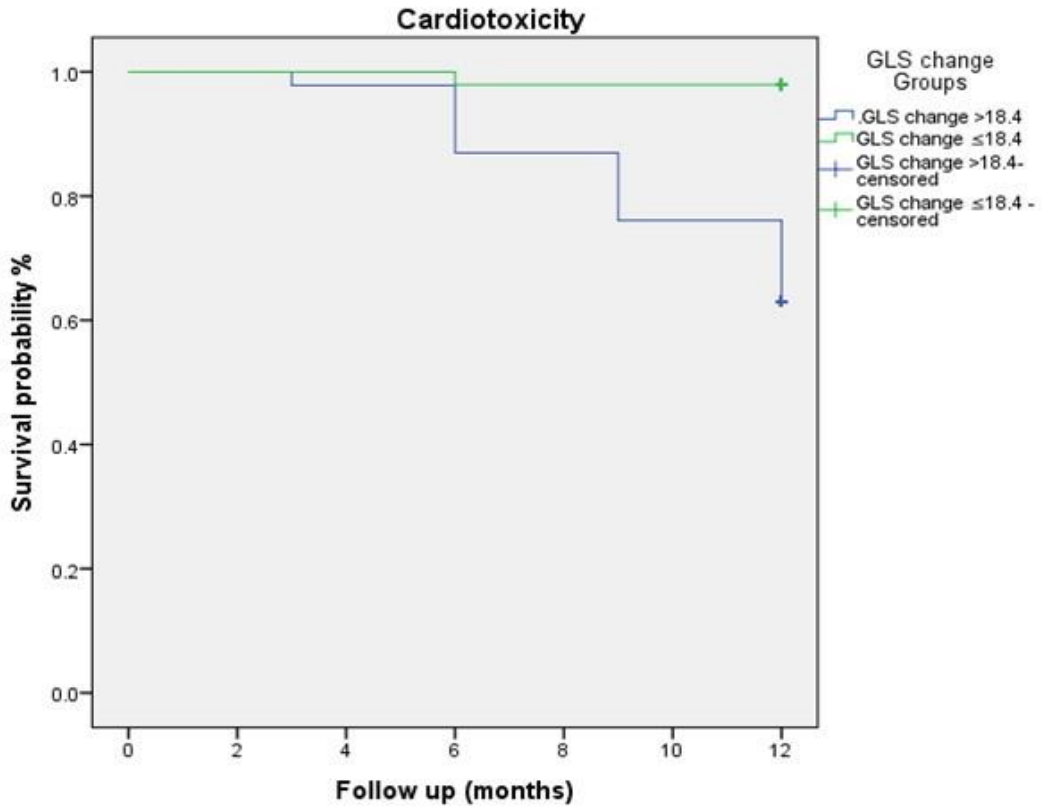

Figure (2): Kaplan–Meier curve analysis of cardiotoxicity. The figureshows the survival probability from cardiotoxicity. The incidence of cardiotoxicityafter chemotherapy was significantly different among the two GLS change groups (divided by cutoff value :18.4 ,log-rank test =33.84, p = <0.001).

DISCUSSION

Anthracyclines induced cardiotoxicity (AIC) is major adverse event that complicates drugs therapy in cancer patients. The extent of cardiotoxicity depend on many factors as type of drug, dose, cumulative dose , route of administration, age, history of preexisting cardiac disease, and hypertension.Techniques used for predicting cardiotoxicity include imaging ,biomarkers. In our study we used speckle tracking techniques to assess serial left ventricular global longitudinal strain and Left atrial strain for early prediction of AIC.

Our study's median patient age was 56 years, and the proportion of female patients was 77.8% of the total number of patients examined. These findings were consistent with those of ***K. Inoue et al***., who examined the cardiotoxic effects of anthracyclines on 383 patients, 93% of whom were female [14]. According to **Baratta et al.'s** study, It looked at patients receiving chemotherapy for a neoplastic disease with a normal cardiac mass and LVEF ≥ 55%. The most typically given medications were trastuzumab in 22% of instances and doxorubicin in 58% of cases. According to our research, AC/T was the most often utilized chemotherapy regimen (43%), with adriamycin coming in second(22%).
[15].

Three months after starting anthracyclines, patients in our study who experienced cardiotoxicity showed larger decreases in LV GLS, LASr, and LASct. Additionally, a relative decline in LV GLS was found to be

independently linked to the development of cardiotoxicity in the future. Our research supported the findings of Inoue et al who found that ΔLASr and ΔLV GLS were independently linked to cardiotoxicity [14]. ***Baratta et al. p***rovided additional support, reporting that the GLS at 0 months was (−20.3% ± 2.7%) and at 3 months was (−18.9 ± 2.5%) before and after treatment [15].

Sawaya et al. examined 81 women with newly diagnosed breast cancer who received anthracyclines, taxanes, and trastuzumab as treatment. The study found that the GLS was affected both before and after chemotherapy at 0 months (−21% ± 2%) and 3 months (−19.2% ± 2%). After three months, the GLS in the individuals under study has decreased by 8.67% from the baseline. In contrast, 23 of the study patients (23.2%) showed a substantial reduction in the GLS at 3 months when compared to the baseline [16]. This is a percentage drop of 10.27%. Furthermore, it was in agreement with ***Negishi et al***.'s research of 81 female patients receiving trastuzumab (mean age, 50 ± 11), which suggested that the best predictor of chemotherapy-related cardiac damage is the GLS at six months**.** They came to the conclusion that a reduction in GLS of less than 8% from the baseline seems to be clinically insignificant, whereas a reduction of more than 15% seems to be clinically significant [17].

Study limitations

The study was limited by its small sample size and short follow-up duration, which affected its ability to assess the early identification of subclinical LV systolic dysfunction using speckle tracking echocardiography.

Conclusion

Myocardial deformation indicators, such LV GLS and LAs, are thought to be a great tool for identifying subclinical LV dysfunction in cancer patients receiving chemotherapy early on. Because it is more reproducible and does not depend on angle, LV GLS is preferred. Physicians can identify patients who may benefit from cardioprotective therapy and chemotherapeutic regimen modification to prevent the development of overt heart failure by detecting an early reduction in LV-GLS.

Our study revealed that at three months follow up after initation of chemotherapy , Δ LV GLS at ≥18.4 % demonstrated a reasonably high prediction ability in prediction of early subclinical cardiotoxicity. According to our findings, LV GLS is a independant helpful indicator for forecasting the development of cardiotoxicity.

Conflicts of interest: None

Financial disclosures: None

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