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Early detection of chemotherapy-induced cardiotoxicity in breast cancer patients using NT-proBNP and echocardiography LV-GLS

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Abstract
Aims: To evaluate the utility of N-terminal pro-B-type natriuretic peptide (NT-proBNP) and global longitudinal strain (GLS) measured via echocardiography for the early detection of subclinical cardiotoxicity in breast cancer patients undergoing anthracycline-based chemotherapy.
Methods: This prospective observational study included a total of 100 breast cancer patients (aged ≥18 years, LVEF ≥50%) scheduled to receive anthracycline-based adjuvant chemotherapy were enrolled. Exclusion criteria included prior chemotherapy or radiotherapy, significant cardiac disease, or poor echocardiographic windows. Baseline and post-chemotherapy (12 weeks) measurements included 2D-transthoracic echocardiography for GLS (average, 4C, 3C, 2C), LVEF, E/e', and NT-proBNP levels. Cardiotoxicity was defined by relative reductions in GLS ≥8% and changes in NT-proBNP and EF. Statistical analyses included paired t-tests and Pearson correlation.
Results: Post-chemotherapy, there was a significant decline in average GLS (from -20.9±1.8 to -19.8±2.2; $p<0.001$) and a rise in NT-proBNP levels (from 67.6±24.3 to 154.2±109.2 pg/mL; $p<0.001$), indicating early myocardial stress. Ejection fraction decreased (from 62.7±3.1 to 59.2±4.6; $p<0.001$), and E/e' increased (from 11.5±1.5 to 12.8±1.5; $p<0.001$). A relative GLS reduction ≥8% was observed in 22 patients and was associated with higher changes in NT-proBNP (median: -109 vs. -48.3; $p<0.001$) and EF (median: 4 vs. 2; $p<0.001$). NT-proBNP correlated significantly with GLS (r=0.512), E/e' (r=0.394), and EF (r=-0.644); $p<0.001$.
Conclusion: NT-proBNP and GLS are sensitive, non-invasive markers for detecting early subclinical cardiotoxicity in breast cancer patients receiving anthracycline chemotherapy. Their integration into routine cardiac monitoring can enable timely identification and intervention, potentially preventing progression to overt heart failure. Further large-scale, long-term studies are warranted to validate these findings and establish standardized protocols.

Keywords: Cardiotoxicity, Echocardiography, Global longitudinal strain (GLS), N-terminal pro-BNP

Introduction
Breast cancer is the most commonly diagnosed cancer in women globally and the fifth leading cause of cancer death [1]. Advances in early detection and therapies have improved survival and reduced cancer recurrence, but treatment-related adverse effects remain a significant concern for survivors [2]. Specifically, cardiotoxicity is a known complication of certain chemotherapeutic agents, particularly anthracyclines, which can lead to congestive heart failure and left ventricular dysfunction, especially in patients with prior cardiac conditions or those who have undergone mediastinal irradiation [3].
Cardiotoxicity, a significant side effect of anthracyclines and other chemotherapeutics, presents as either early onset anthracycline-induced cardiotoxicity, which occurs within the first year post-chemotherapy and is difficult to treat, often leading to progressive heart failure [4, 5], or as reversible cardiotoxicity, typically seen with cyclophosphamide treatment, characterized by transient myocardial dysfunction due to oedema and pericardial involvement [6, 7].
Current recommendations for diagnosing chemotherapy-induced cardiotoxicity involve echocardiographic measurements such as left ventricular diameters, volumes, fractional shortening (FS), and ejection fraction (LVEF) [8, 9].

However, these methods often detect cardiac dysfunction at a late stage when it may be irreversible. Therefore, there is a need for more sensitive and early detection methods. Global longitudinal strain (GLS) has emerged as a strong predictor of cardiac dysfunction and a reliable marker of cardiotoxicity [10].

Cardiac biomarkers like troponin T (TnT), troponin I (TnI), B-type natriuretic peptide (BNP), N-terminal pro-BNP (NT ProBNP), and myeloperoxidase (MPO) can detect early cardiotoxic effects of chemotherapy, ideally during treatment [11, 12]. Combining GLS with cardiac biomarkers, particularly NT ProBNP, may offer a promising approach for identifying and monitoring patients at risk of chemotherapy-induced cardiotoxicity.

There is a significant gap in data regarding the early detection of chemotherapy-induced cardiotoxicity using LV-GLS and NT-proBNP, particularly in predicting long-term cardiac outcomes. Additionally, no studies currently focus on the Indian population, creating a void in region-specific evidence. Furthermore, the link between changes in GLS and long-term clinical outcomes, such as heart failure, remains unclear, highlighting the need for more extensive longitudinal studies to better understand and validate the predictive role of GLS in this context.

Methods

This prospective observational study was conducted at the Department of Medical Oncology, KIMSHEALTH, Trivandrum, from March 2023 to September 2024. A total of 100 patients aged 18 years and above, newly diagnosed with breast cancer and scheduled to receive anthracycline-based adjuvant chemotherapy between February 2023 and February 2024, were enrolled after obtaining informed consent. Eligible participants were asymptomatic, had a left ventricular ejection fraction (LVEF) ≥ 50%, and an ECOG performance status of 0 to 2. Patients with prior chemotherapy or chest radiotherapy, coronary artery disease, arrhythmias, significant valvular heart disease, uncontrolled hypertension (SBP > 160 mmHg or DBP >100 mmHg), overt heart failure (EF < 50%), poor acoustic windows, or those unwilling to consent were excluded.

All patients underwent clinical evaluation, anthropometric measurements, ECG, and transthoracic echocardiography (2D-TTE) at baseline and 12 weeks post-chemotherapy. Echocardiograms were performed using a Vivid E9 machine by a single experienced operator. Venous blood samples for NT-proBNP levels were obtained before the first chemotherapy cycle and 12 weeks after the last dose. Left ventricular global longitudinal strain (LV-GLS) and NT-proBNP were evaluated for their utility in detecting early subclinical cardiotoxicity.

The American Society of Echocardiography (ASE) defines cancer therapeutics-related cardiac dysfunction (CTRCD) as a drop in LVEF ≥ 5% in symptomatic patients or a drop in LVEF ≥ 10% to an EF < 53% in asymptomatic patients [13]. On the basis of numerous studies, it is recommended that a relative decrease in GLS ≥ 15% from baseline should be considered abnormal and an indicator of early LV subclinical dysfunction, whereas a GLS reduction < 8% is unlikely to be clinically significant. A relative percentage reduction in GLS between 8% and 15% represents a gray zone [14-17]. The reference value of NT proBNP levels was set to 5-110 pg/mL.

Statistical analysis was conducted using SPSS v22, with quantitative variables expressed as mean ± SD and categorical variables as frequencies and percentages. Paired and unpaired t-tests, along with chi-square or Fisher's exact tests, were used where appropriate. A p-value <0.05 was considered statistically significant.

Results

The study included 100 breast cancer patients receiving anthracycline-based adjuvant chemotherapy, with a mean age of 49.4±6 years, ranging from 36 to 60 years. Among the 100 patients, 30 patients were aged between 35-45years, 42 patients were aged between 45-55years and 28 patients were aged more than 55years. The age distribution is relevant because cardiotoxicity risk may vary with age, and this cohort represents a broad range of adult patients at risk for chemotherapy-induced cardiotoxicity.

The study measured changes in global longitudinal strain (GLS) and NT-proBNP levels as early markers of cardiotoxicity. The mean change in GLS from baseline was -1.1±0.6, with similar reductions in specific cardiac views (GLS4C: -1.1±0.8, GLS3C: -1.1±0.7, GLS2C: -0.9±0.8). NT-proBNP levels significantly increased from baseline (-86.6±89.6), indicating heightened cardiac stress following chemotherapy. Additionally, the mean change in the E/e' ratio was -1.3±0.9, while global ejection fraction decreased by 3.5±2.1, further suggesting reduced cardiac function post-chemotherapy.

Table 1: Descriptive statistics of changes of variables

Variable	Mean	SD
GLS	-1.1	0.6
GLS4C	-1.1	0.8
GLS3C	-1.1	0.7
GLS2C	-0.9	0.8
NTproBNP	-86.6	89.6
E/e'	-1.3	0.9
Global EF	3.5	2.1

Age had a statistically significant negative correlation with changes in E/e' (r=-0.226, P=0.024) and positive correlation with changes in global ejection fraction (r=0.249, P=0.013). This suggests that older patients may experience less severe diastolic dysfunction (E/e') but more pronounced systolic changes (ejection fraction) after chemotherapy, highlighting age as a potential factor influencing the extent of chemotherapy-induced cardiac dysfunction.

Table 2: Correlation of age with relative change of variables from baseline to post findings

	Age		
Relative change of Variables	Correlation coefficient	p value	N
GLS	0.073	0.472	100
GLS4C	-0.065	0.521	100
GLS3C	0.019	0.851	100
GLS2C	0.001	0.993	100
NTproBNP	-0.159	0.113	100
E/e'	-0.226	0.024	100
Global EF	0.249	0.013	100

The average GLS decreased significantly from a mean ± SD of -20.9±1.8 to -19.8±2.2 (p<0.001). Median (Q1, Q3) values also showed a shift from -21.1 (-22.5, -18.7) to -20.4 (-21.6, -17.6), suggesting a decline in systolic function.

- **GLS 4-Chamber View (GLS 4C):** Mean ± SD changed from -21.0±2.1 to -19.8±2.6, with median (Q1, Q3) shifting from -21.2 (-22.8, -18.8) to -20.4 (-22.0, -17.3).
- **GLS 3-Chamber View (GLS 3C):** Mean ± SD changed from -20.6±1.8 to -19.5±2.1, and median (Q1, Q3) from -20.8 (-22, -18.8) to -19.8 (-21.4, -17.2).
- **GLS 2-Chamber View (GLS 2C):** Mean ± SD changed from -19.4±8.4 to -18.4±8.0, with a median change from -21.2 (-22.6, -18.8) to -21.0 (-21.8, -17.8).

There was a significant rise in NT-proBNP, a marker of myocardial stress, from 67.6±24.3 to 154.2±109.2 pg/mL (p<0.001). Median (Q1, Q3) levels increased from 64 (49, 82.5) to 112.4 (90, 189.5). E/e' ratio increased from 11.5±1.5 to 12.8±1.5, and the median rose from 12 (10, 12) to 13 (12, 14), indicating elevated left ventricular filling pressures post-chemotherapy. A reduction in global LVEF was noted, decreasing from 62.7±3.1 to 59.2±4.6, with a median change from 63 (60, 65) to 60 (55, 63), reinforcing the presence of early systolic dysfunction.

Table 3: Comparison of changes of from baseline to post findings

Variables		Mean	Std. Deviation	Median (Q1, Q3)	P-Value
Average GLS	Pre	-20.9	1.8	-21.1(-22.5, -18.7)	<0.001
	Post	-19.8	2.2	-20.4(-21.6, -17.6)	
GLS4C	Pre	-21.0	2.1	-21.2(-22.8,-18.8)	<0.001
	Post	-19.8	2.6	-20.4(-22.0,-17.3)	
GLS3C	Pre	-20.6	1.8	-20.8(-22,-18.8)	<0.001
	Post	-19.5	2.1	-19.8(-21.4, -17.2)	
GLS2C	Pre	-19.4	8.4	-21.2(-22.6, -18.8)	<0.001
	Post	-18.4	8.0	-21(-21.8, -17.8)	
NTproBNP	Pre	67.6	24.3	64(49, 82.5)	<0.001
	Post	154.2	109.2	112.4(90, 189.5)	
E/e'	Pre	11.5	1.5	12(10, 12)	<0.001
	Post	12.8	1.5	13(12, 14)	
Global EF	Pre	62.7	3.1	63(60, 65)	<0.001
	Post	59.2	4.6	60(55, 63)	

A moderate positive correlation was found between relative changes in NT-proBNP and changes in GLS (r=0.512, p<0.001), GLS4C (r=0.691, p<0.001), and GLS3C (r=0.506, p<0.001). Additionally, NT-proBNP showed a significant positive correlation with E/e' (r=0.394, p<0.001) and a moderate negative correlation with global ejection fraction (r=-0.644, p<0.001). These correlations underscore the link between elevated NT-proBNP levels and worsening cardiac strain, as measured by GLS and ejection fraction.

Table 4: Correlation of Relative change of NT-proBNP with changes of variables from baseline to post findings

Relative change of Variables		Relative change of NT-pro BNP	
	Correlation coefficient	P-Value	n
GLS	0.512	<0.001	100
GLS4C	0.691	<0.001	100
GLS3C	0.506	<0.001	100
GLS2C	0.170	0.092	100
E/e'	0.394	<0.001	100
Global EF	-0.644	<0.001	100

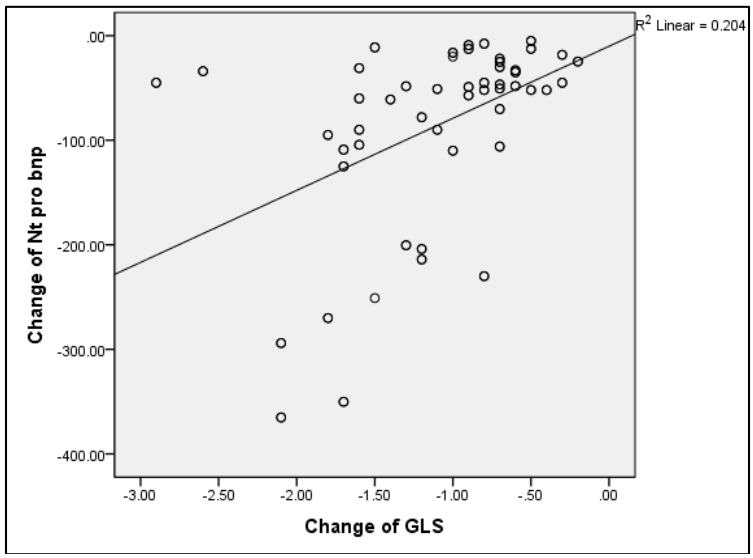

Fig 1: Correlation between relative change of NT-proBNP with GLS

The median relative change in NT-proBNP for patients with less than an 8% change in average GLS was -48.3 (-63.3, -24.0), while for those with an 8% or greater change in average GLS, it was -109 (-294, -90). This indicates that patients with more than an 8% change in average GLS had a higher median change in NT pro BNP.

Table 6: Comparison of relative change of NT proBNP with relative percentage change class of average GLS

Percentage change of average GLS	n	Median(Q1-Q3)	P-Value
<8%	78	-48.3(-63.3, -24.0)	<0.001
≥8%	22	-109(-294, -90)	

The median relative change in global ejection fraction for patients with less than an 8% change in average GLS was 2(2-5), while for those with an 8% or greater change in average GLS, it was 4(3-7). This indicates that patients with an 8% or greater change in average GLS had a higher median change in global ejection fraction.

Table 7: Comparison of relative change of global ejection fraction with relative percentage change class of average GLS

Percentage change of average GLS	n	Median(Q1-Q3)	P-Value
<8%	78	2(2-5)	<0.001
≥8%	22	4(3-7)	

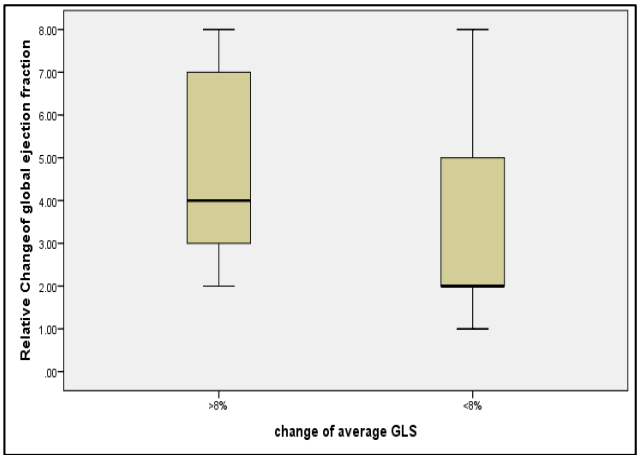

Fig 2: Relative change of global ejection fraction with relative percentage change class of average GLS

Discussion

Our findings demonstrated significant reductions in various global longitudinal strain (GLS) parameters following chemotherapy. The mean change in GLS was -1.1±0.6, and all GLS components (GLS4C, GLS3C, and GLS2C) showed consistent decreases, indicating an overall decline in myocardial function. This is consistent with previous findings by Kittiwarawut *et al*., who reported reductions in left ventricular ejection fraction (LVEF) and a concurrent rise in NT-proBNP among breast cancer patients treated with anthracycline-based regimens, underscoring the subclinical cardiotoxicity associated with such treatments[18].

The NT-proBNP levels significantly increased post-chemotherapy (mean: 154.2, $p<0.001$), aligning with findings from other studies that demonstrate NT-proBNP as a sensitive marker for early detection of anthracycline-induced cardiotoxicity [10, 19]. Additionally, the E/e ratio exhibited a significant increase (mean: 12.8, $p<0.001$), further indicating diastolic dysfunction, while the global ejection fraction showed a statistically significant reduction (mean change: 3.5±2.1, $p<0.001$).

The correlation analysis revealed statistically significant, positive correlations between the relative change in NT-proBNP levels and GLS (r=0.512, $p<0.001$), GLS4C (r=0.691, $p<0.001$), and GLS3C (r=0.506, $p<0.001$). This suggests that increased NT-proBNP levels are associated with a decline in myocardial strain, supporting its role as a potential biomarker for detecting early myocardial injury. Conversely, there was a moderate negative correlation between NT-proBNP levels and global ejection fraction (r=-0.644, $p<0.001$), which indicates that NT-proBNP elevation is linked to a decrease in systolic function, consistent with the observations by Cil *et al*., who showed that elevated NT-proBNP levels are associated with reduced LVEF in breast cancer patients treated with anthracyclines[20].

Our study's findings corroborate previous research, which has highlighted the role of GLS and NT-proBNP in detecting subclinical cardiac dysfunction in breast cancer patients undergoing chemotherapy. Kittiwarawut *et al*. reported significant reductions in LVEF and an increase in NT-proBNP levels, consistent with our observations of significant strain parameter reductions post-chemotherapy[18]. Additionally, Cil *et al*. demonstrated that higher NT-proBNP levels were associated with reduced LVEF in asymptomatic breast cancer patients, suggesting that NT-proBNP could serve as an early indicator of anthracycline-induced cardiotoxicity [20].

Interestingly, our study found that while NT-proBNP and GLS changes correlated with cardiac dysfunction, the changes in absolute GLS values were not as sensitive in detecting early myocardial injury as the relative reductions in GLS. This finding aligns with the work of Calle *et al*., who demonstrated that abnormal GLS values could predict future drops in LVEF, even when LVEF values are within the normal range at baseline [21]. Our findings also support the suggestion that GLS should be used as a marker of stage B heart failure in patients treated with anthracycline-trastuzumab regimens.

The results of this study suggest that NT-proBNP and GLS parameters should be integrated into routine cardiac monitoring of breast cancer patients undergoing anthracycline-based chemotherapy. A relative reduction in GLS greater than 8% was associated with a significantly higher median change in NT-proBNP levels, which could serve as a valuable tool for early detection of chemotherapy-induced cardiotoxicity. This is particularly important as early identification of subclinical cardiac dysfunction allows for timely intervention with cardioprotective strategies, potentially preventing the progression to symptomatic heart failure.

This study focused only on the short-term effects of chemotherapy, and further research is needed to explore long-term outcomes. The sample size was relatively small, which may limit the generalizability of our findings. Future studies should aim to include larger cohorts with extended follow-up periods to validate the utility of NT-proBNP and GLS in predicting long-term cardiac outcomes in this patient population.

Our study demonstrates that NT-proBNP and GLS are sensitive markers for detecting early subclinical myocardial injury in breast cancer patients undergoing anthracycline-based chemotherapy. The integration of these markers into routine clinical practice could facilitate the early detection

and management of chemotherapy-induced cardiotoxicity, ultimately improving patient outcomes. Further studies are warranted to confirm these findings and to establish standardized protocols for the use of NT-proBNP and GLS in this context.

Integrating NT-proBNP with LV-GLS measurements may be particularly beneficial in identifying patients with subtle LV systolic dysfunction. Early detection and appropriate management of these patients can potentially improve outcomes and prevent the progression of overt heart failure. Further research is warranted to validate these approaches and establish their role in clinical practice.

Conflict of Interest

Not available

Financial Support

Not available

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