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GLOBAL LONGITUDINAL STRAIN AND 3D ECHOCARDIOGRAPHY FOR EARLY DETECTION OF ANTHRACYCLINE-INDUCED SUBCLINICAL CARDIOTOXICITY: NARRATIVE REVIEW

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ABSTRACT

Introduction and purpose: Anthracyclines are highly effective chemotherapeutic agents but carry a significant risk of dose-dependent cardiac toxicity, often progressing silently before left-ventricular ejection fraction (LVEF) declines. The objective of this review was to evaluate the role of three-dimensional echocardiography (3D-ECHO) and global longitudinal strain (GLS) in the early detection of subclinical anthracycline-induced cardiotoxicity and to compare their diagnostic performance with conventional 2D-LVEF assessment.

Methods: A narrative review was conducted using clinical and observational studies indexed in PubMed over the last ten years. Only peer-reviewed human research evaluating anthracycline-induced cardiotoxicity was included. Studies comparing 2D-LVEF with 3D-LVEF and/or global longitudinal strain (GLS), as well as those assessing early markers of subclinical left-ventricular dysfunction, were selected. Extracted data focused on diagnostic effectiveness, time-to-detection of myocardial injury, and prognostic relevance of strain-based parameters.

Conclusion: GLS and 3D-echocardiography outperform conventional 2D-LVEF in identifying early, subclinical anthracycline-related cardiotoxicity. GLS provides the highest sensitivity for early myocardial injury, while 3D-STE enhances spatial assessment and detects dysfunction before EF decline. Routine integration of these modalities into cardio-oncology surveillance may enable earlier intervention, prevent irreversible damage, and improve long-term cardiac outcomes.

KEYWORDS

Cardiotoxicity, Anthracyclines, 3D Echocardiography, Global Longitudinal Strain, GLS, Cardio-Oncology

CITATION

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Introduction

Anthracyclines, including doxorubicin, daunorubicin, epirubicin and idarubicin, continue to serve as core pharmacologic agents in the management of numerous cancers. Their effectiveness spans both hematologic malignancies and solid tumors, with established use in Hodgkin and non-Hodgkin lymphoma, acute leukemias, breast cancer, ovarian cancer and various sarcoma subtypes [1]. However, despite their therapeutic impact, these agents are strongly associated with dose-dependent and potentially irreversible cardiotoxic effects.

Anthracycline-induced cardiotoxicity is classically divided into three temporal forms. Acute toxicity may emerge during treatment or within two weeks of administration and typically presents as transient depression of myocardial contractility, often reversible with supportive management. Early-onset chronic cardiotoxicity develops within the first year following treatment completion and is frequently irreversible, commonly progressing into dilated or hypokinetic cardiomyopathy. In contrast, late-onset chronic cardiotoxicity manifests more than one year after exposure, mimicking early chronic patterns with ongoing ventricular dilatation, systolic dysfunction and poor therapeutic reversibility. Both chronic phenotypes are associated with an unfavorable long-term prognosis [2].

Epidemiological data indicate that early chronic cardiotoxicity affects approximately 2–5% of treated patients within the first year, while late-onset dysfunction is observed in an additional 2–10%. Notably, individuals may remain clinically asymptomatic for extended periods due to compensatory mechanisms preserving global function until a threshold of structural injury is surpassed. This delayed clinical expression underscores the necessity for early identification of subclinical myocardial damage to prevent progression towards overt heart failure and reduce chemotherapy-related morbidity [3].

On a mechanistic level, anthracycline toxicity is strongly linked to oxidative injury. These agents generate reactive oxygen species (ROS), which interact with intracellular iron to produce hydroxyl radicals capable of inducing marked oxidative stress. The resulting injury promotes myocyte apoptosis, necrosis, and fibrotic replacement, contributing to ventricular remodeling. Structural vulnerability may be more pronounced in the right ventricle due to its thinner myocardial wall and reduced physiological reserve [4].

Patient-related vulnerability also influences cardiotoxicity risk. Evidence suggests that individuals with pre-existing cardiovascular risk factors—including hypertension, diabetes mellitus, and obesity—experience higher rates of anthracycline-mediated cardiac injury compared to patients without these comorbidities. This highlights baseline cardiovascular status as a key determinant of susceptibility to treatment-related dysfunction and reinforces the importance of risk-stratified monitoring strategies [5].

Definition and Clinical Classification of Cancer Therapy–Related Cardiac Dysfunction

Cancer therapy–related cardiac dysfunction (CTRCD) is a broad term describing myocardial injury resulting from oncologic treatment, ranging from early asymptomatic impairment to advanced heart failure. Importantly, cardiotoxicity does not always present with symptoms; subclinical myocardial dysfunction may be present long before reductions in left ventricular ejection fraction become measurable. This silent phase is clinically significant, as subtle deformation abnormalities represent the earliest window for intervention to prevent irreversible myocardial damage.

To facilitate clinical stratification, CTRCD is commonly categorized into three severity levels.

1. **Mild dysfunction** is defined as preserved LVEF (≥50%) accompanied by a ≥15% relative decline in global longitudinal strain (GLS) or newly elevated cardiac biomarkers, indicating early myocardial alteration despite maintained systolic output.
2. **Moderate CTRCD** encompasses cases in which LVEF declines to 40–49%, reflecting a reduction in systolic reserve even in the absence of symptoms.
3. **Severe CTRCD** is diagnosed when LVEF falls below 40%, signifying advanced systolic failure with high risk of clinical progression.

This hierarchical framework provides consistency in diagnosis, supports individualized monitoring strategies and allows more rapid detection of subclinical myocardial injury — a key factor in preventing transition to irreversible cardiac dysfunction and overt heart failure [6].

Incidence of Chemotherapy-Induced Cardiotoxicity Across Drug Classes

Anthracyclines are widely recognized as some of the most cardiotoxic agents used in oncology, with risk increasing proportionally with cumulative exposure. Doxorubicin demonstrates a clear dose-dependent profile, with cardiotoxicity occurring in approximately 3–5% of patients at 400 mg/m², rising to 7–26% at 550 mg/m², and reaching as high as 18–48% once cumulative dosing approaches 700 mg/m². Comparable patterns have been observed with other drugs in this class: idarubicin is associated with cardiotoxicity in 5–18% of cases, while epirubicin generally exhibits lower overall incidence, ranging from 0.9% to 11.4% depending on total exposure. Even liposomal anthracycline formulations — designed specifically to minimize myocardial toxicity — still present measurable risk, around 2% in treated individuals. Similar observations apply to mitoxantrone, where cardiotoxicity is reported in approximately 2–3% of patients [7].

Beyond anthracyclines, several other chemotherapeutic classes contribute to treatment-related cardiac injury. Among alkylating agents, cyclophosphamide is associated with cardiotoxicity in 7–28% of cases, and ifosfamide exhibits notable dose-dependency, with incidence rising dramatically up to ~17% at cumulative doses above 10 g/m². Taxane-based therapies also vary in cardiac impact — docetaxel demonstrates cardiotoxicity in approximately 2–13% of patients, whereas paclitaxel is associated with a significantly lower rate, typically below 1%. Targeted monoclonal antibody therapies display wide variability, with trastuzumab carrying a cardiotoxic risk ranging from 1.7% to 20%, while bevacizumab shows a lower yet relevant incidence of approximately 1.6–4%, depending on patient characteristics and treatment conditions [8].

Limitations of Biomarkers and the Emerging Role of 3D Speckle-Tracking Echocardiography

Although serological biomarkers remain useful indicators of cardiac stress and injury, their diagnostic precision is limited when used in isolation. Research demonstrates that biomarkers, despite high sensitivity, lack sufficient specificity to reliably detect early diastolic dysfunction. Subtle myocardial impairment may therefore remain unrecognized unless biomarkers are complemented with imaging-based assessment [9].

Three-dimensional speckle-tracking echocardiography (3D-STE) has emerged as an advanced diagnostic modality capable of overcoming these limitations. Unlike traditional two-dimensional strain imaging, which analyzes myocardial deformation within a single plane, 3D-STE tracks speckle motion throughout the entire left ventricular volume. This enables evaluation of multidirectional myocardial fiber contraction and provides deformation indices such as area strain, offering a physiologically more accurate representation of ventricular mechanics. Because ventricular contraction is inherently three-dimensional, 3D-based analysis supports a more detailed and realistic characterization of myocardial performance [10].

In comparison with 2D techniques, 3D-STE offers greater reproducibility and more accurate quantification of left-ventricular strain, geometry and functional parameters. Studies consistently report higher measurement stability and improved reliability of volumetric assessment when using 3D imaging [11]. Additional advantages include the ability to assess complex mechanics such as twist, torsion and principal strain, all of which contribute valuable insight into subtle functional impairment preceding changes in global ejection performance [10].

A key technical strength of three-dimensional echocardiography is the lack of geometric assumptions required for chamber modeling. The modality allows unrestricted visualization planes, minimizes foreshortening and generates anatomically realistic imaging reconstructions — features not achievable with conventional 2D-based evaluation [12]. Furthermore, evidence indicates that 3D echocardiography produces more reproducible LVEF measurements than standard 2D imaging, even among less experienced operators, and requires shorter learning curves. This highlights its suitability for longitudinal cardiac monitoring in clinical oncology [13].

Importantly, emerging data suggest that 3D-STE — particularly when analyzing global area strain — may detect myocardial injury at an earlier stage than traditional ejection fraction measurements. Decline in 3D-derived deformation indices has been observed at relatively low cumulative anthracycline doses, often preceding measurable reduction in LVEF. These findings support the role of 3D-STE as a sensitive tool for identifying subclinical cardiotoxicity and emphasize its value in early surveillance during anthracycline-based therapy [14].

Global Longitudinal Strain (GLS) as a Diagnostic Marker of Subclinical Cardiotoxicity

Global longitudinal strain (GLS) is an echocardiographic metric derived using speckle-tracking technology and reflects the degree of longitudinal myocardial deformation within the left ventricle [15]. It is widely used as a quantitative index of systolic performance and is considered more sensitive than conventional LVEF for detecting early functional impairment. Normative reference data indicate that values more negative than −18% are typically regarded as normal, while GLS between approximately −16% and −18% is classified as borderline — a variability influenced in part by the load-dependent nature of strain assessment [16].

A relative GLS reduction of 10–15% during therapy is strongly predictive of emerging cardiotoxicity, including both symptomatic and asymptomatic ventricular dysfunction. Reported diagnostic thresholds vary, with confidence intervals typically ranging 8–15%. When relative values cannot be assessed, absolute GLS values less negative than −19% or −20.5% have been associated with higher cardiotoxicity risk [17]. Across available studies, GLS demonstrates a moderate but clinically relevant predictive capacity, with mean sensitivity and specificity approximating 70%, indicating balanced performance in identifying patients likely to develop dysfunction while limiting false-classification rates [18].

Importantly, GLS deterioration often precedes EF decline by a substantial margin. Observational data suggest that a relative GLS reduction of ~15% becomes detectable at around 205 days, whereas LVEF impairment typically emerges later, highlighting the superiority of GLS for early detection of subclinical myocardial injury [19].

Recent evidence strongly supports integration of GLS into routine cardio-oncologic monitoring. Literature consistently emphasizes that GLS should not be reserved solely for high-risk individuals but may serve as a standard component of comprehensive echocardiographic evaluation in oncologic care [20], [21]. Furthermore, inter-vendor reproducibility of GLS measurements is well demonstrated. Comparative studies across different ultrasound platforms show close agreement with minimal bias, narrow variability, and strong correlation coefficients, confirming GLS as a robust and reliable clinical parameter suitable for routine follow-up [22].

Methodology

This article is based on a narrative review of clinical studies retrieved from PubMed, limited to publications from the past ten years. Only human research investigating anthracycline-related cardiotoxicity was considered. Studies using global longitudinal strain (GLS) and/or three-dimensional echocardiography (3D-ECHO), particularly those comparing these methods with standard 2D-LVEF or evaluating subclinical myocardial injury, were included. Articles lacking echocardiographic assessment, relying solely on biomarkers, or involving non-clinical models were excluded. Relevant findings were extracted manually and summarized descriptively, with emphasis on diagnostic performance, sensitivity to early dysfunction, and prognostic value.

Results

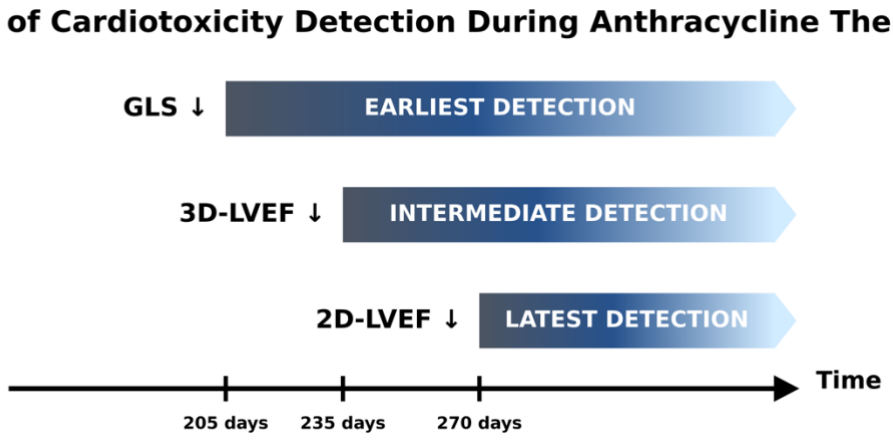
Research comparing traditional 2D left ventricular ejection fraction (2D-LVEF), three-dimensional ejection fraction (3D-LVEF) and global longitudinal strain (GLS) demonstrates a clear diagnostic hierarchy. Across multiple studies, GLS consistently emerges as the most sensitive parameter for detecting early left-ventricular systolic impairment, identifying subclinical dysfunction before measurable reductions in ejection fraction occur. Although 3D-LVEF provides more accurate volumetric analysis than 2D-LVEF due to fewer geometric assumptions, it still lags behind GLS in identifying early myocardial alteration [23].

Further comparative evidence confirms that GLS detects cardiotoxic injury sooner than both 2D and 3D LVEF, with deformation changes appearing even when standard systolic indices remain within normal limits. In contrast, 2D- and 3D-derived ejection fractions typically decline only after myocardial strain deterioration is already underway, reinforcing GLS as the earliest and most sensitive marker of ventricular dysfunction [24].

Time-dependent differences in detection further support this observation. In prospective assessment, GLS deterioration became evident at a mean of 205 days, whereas a decline in 3D-LVEF occurred later — around **235 days**. Changes in 2D-LVEF appeared much later, only after more advanced dysfunction had developed. This establishes a clear temporal progression:

GLS detects dysfunction first → 3D-LVEF second → 2D-LVEF last [19].

Strain-based imaging has also demonstrated greater predictive value for long-term functional decline. Even in patients with normal baseline ejection fraction, abnormalities in GLS — measured in both 2D and 3D modalities — correlate more strongly with future deterioration than volumetric indices alone. These findings suggest that deformation-based parameters identify high-risk individuals earlier and more reliably than ejection-fraction-based metrics [25].



Technical and Methodological Limitations of GLS and 3D Echocardiography

Although imaging-based deformation analysis offers superior sensitivity over conventional LVEF assessment, its practical implementation is influenced by several technical factors. Speckle-tracking derived strain requires specialized post-processing software, and reported values may vary between vendors due to differences in image reconstruction and tracking algorithms. While global longitudinal strain (GLS) shows good reproducibility, segmental strain remains more variable, particularly when image quality is suboptimal. For that reason, consistency in equipment selection is essential for serial monitoring [26].

Three-dimensional speckle-tracking echocardiography (3D-STE), despite offering comprehensive volumetric deformation analysis, is limited by lower temporal resolution, dependency on high-quality acoustic

windows, and the frequent requirement for offline post-processing. Variability in measurement platforms further reduces standardization, and operator dependency may affect reproducibility in real-world practice [27].

Studies comparing different acquisition systems confirm that 3D-based strain measurements vary significantly between software vendors, requiring independent reference ranges rather than direct comparison with 2D-derived values. This methodological mismatch reflects fundamental differences in acquisition technique and processing architecture [28].

Clinical feasibility also remains a challenge. In practice, 3D-LVEF assessment is possible in only ~40% of patients due to limited border-tracking quality, while 2D-based feasibility is significantly higher — 73% for GLS and 81% for 2D-LVEF. This gap demonstrates that although 3D echocardiography offers theoretical diagnostic superiority, its clinical usability continues to depend heavily on imaging conditions and equipment performance [29].

Discussion and conclusion

The evidence summarized in this review demonstrates that conventional LVEF measurement is not sufficient for early identification of anthracycline-related myocardial injury. Because LVEF decline occurs only after substantial structural damage has already developed, relying on this parameter alone delays diagnosis to a stage where dysfunction may be irreversible. In contrast, global longitudinal strain (GLS) consistently detects subclinical impairment earlier, with reductions of ~15% appearing at approximately 205 days of treatment — well before measurable changes in either 3D- or 2D-derived ejection fraction. This early responsiveness, combined with high reproducibility and suitability for routine assessment, supports GLS as the most effective first-line tool for cardio-oncology surveillance.

Three-dimensional echocardiography offers additional value by providing more accurate volumetric quantification and advanced deformation analysis, and early deterioration in 3D strain may also precede EF decline. However, the technique is limited by image-quality dependence, vendor variability, and lower feasibility in clinical conditions — with complete diagnostic acquisition achievable in only ~40% of patients. For this reason, 3D-STE is best positioned as a complementary method rather than a universal screening standard.

Altogether, the optimal diagnostic model appears to be one in which GLS serves as the primary marker of early dysfunction, supported by 3D-echocardiography when feasible, and LVEF is used mainly to confirm established systolic decline. Such an approach may allow treatment adjustment or cardioprotective intervention before permanent damage occurs, shifting practice from late detection to preventative management.

In conclusion, GLS is currently the most sensitive and clinically practical imaging parameter for detecting early anthracycline-induced cardiotoxicity. Three-dimensional echocardiography enhances assessment when available but remains limited by feasibility constraints. Future work should focus on standardizing strain thresholds, improving 3D-tracking technology and evaluating whether GLS-guided monitoring improves long-term cardiac outcomes. Early detection remains the most critical element in preventing progression to irreversible heart failure in oncology patients.

All authors have read and agreed with the published version of the manuscript.

Conflict of Interest Statement: The authors declare that they have no conflict of interest.

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