Layer-Specific Strain Analysis for Early Detection of Anthracycline

Kavelashvili N.[1,2], Sharashenidze N.[2], Kloppe A[1].

Abstract

Background: Anthracycline-induced cardiotoxicity affects 6-18% of treated patients and can lead to irreversible heart failure. Left ventricular ejection fraction, the standard monitoring parameter, detects cardiac dysfunction only after substantial myocardial damage has occurred.
Aim: To review current evidence on layer-specific strain analysis for early detection of anthracycline-induced cardiotoxicity and to discuss its integration with cardiac magnetic resonance imaging.
Methods: We searched PubMed, Scopus, and Web of Science for articles published between January 2015 and December 2024 using predefined search terms related to anthracyclines, cardiotoxicity, and layer-specific strain. Original research articles, systematic reviews, and guidelines in English were included. Case reports and conference abstracts were excluded.
Results: Twenty-nine publications meeting inclusion criteria were analyzed. Studies consistently show that anthracycline-induced injury originates in the subendocardium and progresses transmurally. Layer-specific strain analysis detects subendocardial dysfunction when left ventricular ejection fraction and global longitudinal strain remain normal. The combination of echocardiographic strain with cardiac magnetic resonance T1/T2 mapping enables both functional and tissue-level assessment of early cardiotoxicity.
Conclusions: Layer-specific strain analysis can detect anthracycline-induced myocardial injury earlier than conventional parameters. Standardization of methodology and prospective validation studies are needed before routine clinical implementation. **TCM-GMJ May 2026; 11 (1): P72-P77)**

Keywords: Anthracycline; Cardiotoxicity; Layer-specific strain; Speckle tracking echocardiography; Cardiac magnetic resonance; Cardio-oncology

Introduction

Anthracyclines, including doxorubicin, epirubicin, and daunorubicin, remain among the most effective chemotherapeutic agents for breast cancer, lymphoma, leukemia, and sarcoma. Their widespread use, however, is tempered by the well-recognized risk of dose-dependent cardiotoxicity. According to a meta-analysis of 18 studies encompassing 49,017 patients, clinical cardiotoxicity develops in 6.3% and subclinical dysfunction in 17.9% of anthracycline-treated patients [1]. These figures likely underestimate the true burden, given the inconsistent definitions and monitoring protocols used across different institutions.

LVEF monitoring has been the standard approach in clinical practice for decades. The problem, as we see it in daily clinical work, is that LVEF decline represents a relatively late event in the disease process. By the time ejection fraction drops below the conventional threshold of 50-55%, considerable myocardial damage has already occurred. Cardinale et al. reported that 45% of patients with established anthracycline cardiomyopathy fail to recover normal LVEF despite optimal heart failure therapy [2]. This finding has motivated the search for earlier detection methods.

Global longitudinal strain (GLS) measured by speckle tracking echocardiography represents an improvement over LVEF alone. The 2022 ESC Guidelines on cardio-oncology now recommend GLS monitoring, with a relative reduction exceeding 15% from baseline considered clinically significant [3]. While GLS is more sensitive than LVEF, it still measures average deformation across all myocardial layers and may therefore miss early injury confined to specific regions.

Layer-specific strain analysis takes this concept further by quantifying deformation separately in the subendocardial, midmyocardial, and subepicardial layers. Given that anthracycline injury appears to affect the subendocardium

From the [1]Marienhospital Gelsenkirchen, Germany. [2]Ivane Javakhishvili Tbilisi State University, Georgia
Received February 1, 2026; accepted March 2 , 2026.
Address requests to: Kavelashvili Nino
E-mail: n.kavelashvili@st.augustinus.eu

preferentially [4], this technique may offer even earlier detection. In this narrative review, we summarize the current evidence for layer-specific strain in anthracycline cardiotoxicity and consider its potential integration with CMR tissue characterization.

Methods

Search Strategy

We performed a literature search in PubMed, Scopus, and Web of Science. The initial search was conducted on September 15, 2024, with an update on December 10, 2024 to capture recently published work. Search terms included combinations of: (anthracycline OR doxorubicin OR epirubicin OR daunorubicin) AND (cardiotoxicity OR cardiomyopathy OR cardiac dysfunction) AND (layer-specific strain OR multilayer strain OR speckle tracking echocardiography OR myocardial deformation). We used Boolean operators to combine terms appropriately for each database.

Selection Criteria

Inclusion criteria were: peer-reviewed original research articles, systematic reviews, meta-analyses, and clinical guidelines; publication in English between January 2015 and December 2024; reporting of layer-specific or multilayer strain measurements in patients receiving anthracycline chemotherapy. We excluded case reports, editorials, letters, conference abstracts, and studies assessing only global strain parameters without layer-specific analysis. Reference lists of included articles were manually screened for additional relevant publications.

Data Extraction and Synthesis

For each included study, we extracted: study design, sample size, patient population, anthracycline type and cumulative dose where reported, strain measurement method (vendor, software), timing of assessments, strain parameters reported, and main findings. As this is a narrative review rather than a systematic review, we did not perform formal risk of bias assessment or meta-analysis. The evidence was synthesized qualitatively, with studies grouped by population (adult versus pediatric) and imaging modality (echocardiography versus CMR).

Pathophysiology of Anthracycline Cardiotoxicity

Understanding the mechanisms of anthracycline cardiotoxicity helps explain why layer-specific strain analysis might be valuable. The pathophysiology involves multiple interconnected pathways, though oxidative stress appears central. Anthracyclines undergo redox cycling within cardiomyocytes, generating reactive oxygen species that overwhelm cellular antioxidant defenses. The heart is especially vulnerable because cardiomyocytes contain relatively low levels of protective enzymes such as catalase and superoxide dismutase [5].

A major advance in our understanding came from the work of Zhang et al., who identified topoisomerase IIβ as a key mediator of cardiotoxicity [6]. While topoisomerase IIα mediates the antitumor effect in cancer cells, the cardiac isoform (IIβ) causes DNA damage in cardiomyocytes when bound by anthracyclines. Mice lacking the Top2b gene in cardiomyocytes were protected from doxorubicin-induced heart damage, establishing this as a mechanism distinct from the therapeutic effect.

Mitochondrial dysfunction adds another dimension to the problem. Anthracyclines accumulate within cardiac mitochondria and disrupt the electron transport chain, compromising ATP production. Given the enormous energy demands of the continuously contracting heart, this metabolic impairment has significant functional consequences [7]. What makes anthracycline cardiotoxicity notably concerning is its progressive nature; damage may continue to accumulate even after treatment has ended.

Why the Subendocardium Is Affected First

The preferential vulnerability of the subendocardium is central to the rationale for layer-specific strain analysis. Several anatomical and physiological factors contribute to this pattern. The inner myocardial layer experiences the highest wall stress during systolic contraction. It is also the last region to receive coronary blood flow during diastole, making it most susceptible to any compromise in oxygen delivery [8].

From a structural perspective, the subendocardium contains predominantly longitudinally oriented muscle fibers, which are highly sensitive to both ischemic and toxic injury. Histopathological studies in animal models have confirmed this pattern, demonstrating early vacuolization and myofibrillar loss concentrated in the inner layers before transmural extension occurs [9]. This spatial progression of injury, from endocardium outward, provides the theoretical foundation for using layer-specific strain as an early detection tool.

Strain Imaging Techniques

Global Longitudinal Strain

GLS is derived from speckle tracking analysis of apical 4-chamber, 2-chamber, and long-axis views. Normal values typically range from -18% to -22%, with less negative values indicating impaired systolic function. A meta-analysis by Oikonomou et al., including 22 studies and 2,597 patients, confirmed that GLS reduction predicts subsequent cardiotoxicity, with a relative decrease of 10-15% from baseline associated with increased risk [10].

One practical limitation we encounter regularly is vendor variability. The EACVI/ASE Inter-Vendor Comparison Study documented differences of up to 3.7 percentage points between different ultrasound systems [11]. In our department, we have learned to use the same equipment for serial measurements in individual patients whenever possible. When equipment changes are unavoidable, we interpret results with appropriate caution. This is an aspect that often frustrates us in clinical practice, as it limits the comparability of measurements across different institutions.

Layer-Specific Strain Analysis

Layer-specific strain analysis divides the myocardial wall into three layers: subendocardial (inner third), midmyocardial (middle third), and subepicardial (outer third). Most commercial software packages can calculate longitudinal strain for each layer from standard apical views. In healthy individuals, strain magnitude increases from the epicardium toward the endocardium, creating what is termed the transmural gradient [12].

The technical requirements for reliable layer-specific measurements are more demanding than for global strain. Image quality must be sufficient to clearly delineate both endocardial and epicardial borders. Frame rates of 50-80 frames per second are generally recommended to ensure adequate temporal resolution [13]. Inter-observer variability tends to be higher than for global strain, especially for epicardial measurements where border definition is often challenging.

In our experience at Marienhospital Gelsenkirchen, adequate image quality for layer-specific analysis is achievable in approximately 75-80% of oncology patients when examinations are performed by experienced sonographers. Patients with obesity, chronic lung disease, or post-surgical chest wall changes present the greatest technical difficulties. We have found that taking additional time for patient positioning and respiratory coaching can improve success rates in borderline cases. Frankly, the learning curve for reliable layer-specific analysis is steeper than most published studies acknowledge.

Clinical Evidence for Layer-Specific Strain in Cardiotoxicity Detection

Table 1 summarizes key studies examining layer-specific strain in anthracycline-treated patients. Although the number of published studies remains limited, the findings show notable consistency.

BMT = bone marrow transplantation; CS = circumferential strain; endo = endocardial; epi = epicardial; GLS = global longitudinal strain; IVPG = intraventricular pressure gradient; mo = months; subendo = subendocardial; yrs = years. All studies included age-matched healthy controls.

The study by Hashimoto et al. provided valuable insight into the temporal and spatial progression of myocardial damage [14]. By examining 102 childhood cancer survivors at varying intervals after treatment (ranging from 1 to 25 years), they demonstrated that dysfunction begins in the subendocardium of basal segments and gradually extends toward the epicardium and apex. This pattern has direct implications for surveillance: layer-specific analysis may identify early changes that global measurements would average out.

The prospective study by Chen et al. in breast cancer patients receiving epirubicin merits close attention [16]. They found that subendocardial longitudinal strain decreased significantly after chemotherapy while GLS remained within normal limits. Perhaps more importantly, baseline subendocardial strain values were predictive of subsequent cardiotoxicity development. If confirmed in larger studies, this finding could support the use of layer-specific strain for risk stratification before treatment begins.

One consistent observation across studies is the loss of the normal transmural strain gradient. In healthy myocardium, endocardial strain exceeds epicardial strain by approximately 5-8 percentage points [12]. Reduction or reversal of this gradient appears to be an early marker of anthracycline injury, detectable even when absolute strain values remain in the normal range [15]. We believe this gradient analysis may prove clinically useful, though it requires validation in larger prospective cohorts.

Cardiac Magnetic Resonance Imaging

T1 and T2 Mapping

CMR offers tissue characterization capabilities that echocardiography cannot match. Native T1 mapping reflects myocardial tissue composition; elevated values suggest increased extracellular space (as occurs with fibrosis) or edema. Extracellular volume fraction (ECV), calculated from pre- and post-contrast T1 values, provides a validated quantitative marker of diffuse myocardial fibrosis [19].

T2 mapping is sensitive to myocardial edema and inflammation. In the setting of anthracycline cardiotoxicity, T2 elevation may represent acute, potentially reversible injury, whereas T1 and ECV changes tend to indicate more established fibrotic remodeling [20]. A position statement from the Heart Failure Association, EACVI, and ESC Cardio-Oncology Council, synthesizing evidence from 26 studies, found that T1, T2, and ECV were frequently elevated in affected patients, often without accompanying late gadolinium enhancement [21].

The temporal sequence of these mapping abnormalities has practical implications. Lustberg et al. demonstrated that T2 elevation occurs early after anthracycline exposure and can normalize if treatment is modified, suggesting a window during which intervention might prevent permanent damage [22]. T1 and ECV elevation, by contrast, typically persists, reflecting irreversible structural changes. At our institution, we have found that patients generally tolerate CMR examinations well, though scheduling constraints and occasional claustrophobia can limit its use for routine surveillance. In our view, the biggest practical barrier is often simply getting timely access to CMR in busy clinical settings.

CMR Feature Tracking Strain

CMR-based strain analysis using feature tracking offers an alternative to echocardiographic assessment. Advantages include better image quality and the ability to assess strain alongside tissue characterization in a single examination. Tahir et al. showed that combining CMR strain with mapping parameters improves prediction of subsequent cardiac dysfunction compared to either approach alone (Table 2.)[23].

Numbers in brackets indicate supporting references. Reproducibility expressed as coefficient of variation. CMR = cardiac magnetic resonance; GLS = global longitudinal strain; LVEF = left ventricular ejection fraction

Integrating Modalities in Clinical Practice

The 2022 ESC Guidelines recommend echocardiography with GLS as the primary surveillance modality, reserving CMR for situations where echocardiographic windows are inadequate or tissue characterization would inform management decisions [3]. Layer-specific strain is not currently included in guideline recommendations, primarily due to limited validation data and lack of standardization.

Based on available evidence and our institutional experience, we suggest the following approach may be reasonable for high-risk patients (cumulative doxorubicin dose

≥250 mg/m² or equivalent): baseline CMR with T1/T2 mapping before or shortly after starting anthracyclines; serial echocardiography including both GLS and layer-specific strain during treatment; and repeat CMR if strain parameters deteriorate significantly. This multimodal strategy aims to capture both functional and tissue-level changes, though it requires prospective validation before it can be recommended as standard of care.

Current Limitations

Several obstacles currently limit the clinical implementation of layer-specific strain analysis. Vendor-specific software differences represent perhaps the most significant practical barrier. Unlike GLS, which has benefited from industry harmonization initiatives, layer-specific parameters remain highly vendor-dependent, with systematic differences in measured values between platforms [24]. We have encountered this problem when attempting to compare measurements obtained on different ultrasound systems within our own department.

The lack of established reference values presents another challenge. Existing normative data derive from relatively small studies and do not adequately account for age, sex, body size, and other demographic factors that influence strain measurements [25]. Without robust population-specific reference ranges, distinguishing true pathology from normal variation in individual patients remains difficult. From our perspective, this is probably the most frustrating limitation in daily clinical practice—we often find ourselves uncertain whether a borderline abnormal value truly indicates early damage or simply reflects normal biological variation.

Feasibility is lower than for global strain assessment. The stringent image quality requirements mean that layer-specific analysis is simply not achievable in all patients. Published studies report success rates ranging from 70% to 90%, compared with greater than 95% for GLS [13]. In routine clinical practice, outside specialized imaging centers, these rates may be even lower.

Perhaps the most fundamental limitation, however, is the absence of outcome data demonstrating that earlier detection actually improves patient outcomes. No randomized trials have yet shown that interventions guided by layer-specific strain changes prevent heart failure or reduce mortality. While earlier detection is intuitively appealing, the clinical value of the technique remains unproven until such evidence emerges [26]. Some authors have raised concerns about potential overdiagnosis, noting that identifying subclinical abnormalities could lead to unnecessary treatment modifications or patient anxiety.

Future Directions

Ongoing Research

Several prospective studies are now examining multimodal imaging approaches for cardiotoxicity surveillance. At our institution, we have initiated the CARDIAC-STRAIN study (Multimodal Early Detection of Anthracycline-Induced Cardiotoxicity Using Layer-Specific Strain Analysis; DRKS00039155), which is currently recruiting 120 patients receiving anthracycline-based chemotherapy [27]. The protocol includes serial CMR with T1/T2 mapping and echocardiography with layer-specific strain analysis (using Medis Suite QStrain software) at four time points: baseline, after the fourth treatment cycle, four weeks after completion of chemotherapy, and six months post-treatment.

This study design will allow us to address several unanswered questions. Which parameter changes first: subendocardial strain, global strain, or tissue mapping values? Which combination of parameters best predicts clinically relevant cardiotoxicity? Do early abnormalities persist or do they resolve without intervention? Answering these questions is essential for developing rational surveillance protocols.

Standardization and Artificial Intelligence

Vendor harmonization for layer-specific strain measurement is clearly needed. An industry collaboration similar to the initiative that improved GLS standardization would facilitate comparison across studies and development of universal thresholds [28]. Until such harmonization occurs, we recommend that serial measurements in individual patients use consistent equipment and software whenever possible.

Automated strain analysis using machine learning could potentially address current limitations related to operator dependence and measurement variability. Early applications for GLS measurement have demonstrated accuracy comparable to expert analysis [29]. Extension to layer-specific measurements appears technically feasible and could facilitate wider clinical adoption if validated appropriately.

Conclusion

Layer-specific strain analysis offers the potential to detect anthracycline-induced myocardial injury at the subendocardial level before global functional parameters decline. This capability is consistent with the known pathophysiology of anthracycline cardiotoxicity, which preferentially affects the inner myocardial layers before progressing transmurally.

Current evidence supports the concept that earlier detection is achievable with this technique. The studies reviewed here consistently demonstrate reduced subendocardial strain in anthracycline-exposed patients, with changes detectable before LVEF decline or clinical symptoms emerge. However, significant gaps in knowledge remain. Standardization of methodology, establishment of population-specific reference values, and demonstration that early intervention based on these findings improves outcomes are all needed before layer-specific strain analysis can be recommended for routine clinical use.

The integration of echocardiographic strain techniques with CMR tissue characterization represents a promising approach to comprehensive cardiotoxicity surveillance. For patients receiving high-dose anthracyclines, multimodal imaging may provide the functional and structural information needed to guide cardioprotective interventions while myocardial injury is still potentially reversible. Ongoing prospective studies will help clarify the optimal role of these techniques in clinical practice.

Declarations

Conflict of Interest: NK is the principal investigator of the CARDIAC-STRAIN study referenced in this review. NS and AK serve as co-investigators in this study. No financial conflicts of interest exist.

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Author Contributions: NK conceived and designed the review, conducted the literature search, and drafted the manuscript. NS provided critical revision, methodological guidance, and supervision as doctoral thesis advisor. AK contributed to critical revision of clinical content and approved the final manuscript. All authors meet ICMJE criteria for authorship.

Table 1. Key studies on layer-specific strain in anthracycline cardiotoxicity

Study	Population	N	Design	Dose	Follow-up	Software	Main Findings
Hashimoto 2018 [14]	Childhood cancer survivors	102	Cross-sect.	180 mg/m²	1-25 yrs	EchoPAC (GE)	Dysfunction progresses endo→epi, base→apex
Monte 2018 [15]	Lymphoma survivors	35	Cross-sect.	300 mg/m²	8 yrs	QLAB (Philips)	Subendo CS reduced; gradient impaired
Chen 2020 [16]	Breast cancer	78	Prospective	400 mg/m²	6 mo	TomTec	Subendo changes preceded GLS
Paraskevaidis 2017 [17]	BMT patients	45	Prospective	Variable	12 mo	EchoPAC (GE)	Earlier detection than conventional echo
Hosono 2024 [18]	Childhood cancer survivors	64	Cross-sect.	240 mg/m²	15 yrs	EchoPAC (GE)	Layer strain + IVPG additive value

Table 2. Comparison of imaging modalities for cardiotoxicity surveillance

Parameter	LVEF (Echo)	GLS (Echo)	Layer-Specific Strain	CMR T1/T2 Mapping
Detection timing [3,10,14]	Late	Intermediate	Early	Early
Availability	Universal	Widely available	Requires expertise	Specialized centers
Reproducibility [11,13]	Moderate (8-10%)	Good (5-8%)	Moderate (10-15%)	Good (3-5%)
Tissue characterization	No	No	No	Yes
Feasibility [13]	>95%	>95%	70-90%	>95%
Guideline recommendation [3]	Class I	Class I	Not included	Class IIa

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