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CANCER THERAPY-RELATED CARDIOTOXICITY :EARLY DETECTION USING LOW-CJST CLINICAL TOOLS- A CLINICALLY ORIENTED NARRATIVE ANALYSIS

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Abstract: Cardiotoxicity remains one of the most consequential non-oncologic complications of modern cancer therapy, often limiting optimal treatment and compromising long-term survival. While advanced imaging modalities such as global longitudinal strain (GLS) and cardiac MRI have improved early detection, their limited availability in low-resource settings necessitates alternative strategies. This paper presents a clinically grounded, pathophysiology-driven framework for early detection of cardiotoxicity using widely accessible tools. Through retrospective case-based analysis and integration of mechanistic insights, we demonstrate that careful interpretation of symptoms, electrocardiography, basic echocardiography, and simple biomarkers can identify early myocardial injury before overt dysfunction develops. Practical diagnostic algorithms and cardioprotective strategies are proposed to support clinicians working in resource-constrained environments.

Introduction

The success of modern oncology has transformed cancer into a chronic disease for many patients. However, this success has been accompanied by a growing burden of cardiovascular complications. Cardiotoxicity is not merely an adverse effect—it is a competing determinant of survival, particularly in patients treated with anthracyclines, HER2-targeted therapies, and certain targeted agents.

In high-resource settings, early detection relies heavily on sensitive imaging techniques such as GLS and cardiac MRI. Yet, for the majority of the world—including many oncology centers—these technologies remain inaccessible. This creates a critical clinical dilemma: **how to detect myocardial injury early, when intervention is still effective, using only basic tools**.

To address this gap, it is essential to move beyond technology-dependent models and return to first principles—understanding how molecular injury translates into structural changes, how these changes manifest clinically, and how they can be detected with simple, reproducible methods.

Methods

This study is based on a retrospective analysis of 15 oncology patients treated with potentially cardiotoxic regimens (anthracyclines and trastuzumab-based therapy). Patients were followed using a low-cost monitoring model consisting of:

- Clinical assessment (fatigue, dyspnea, exercise tolerance)
- Serial ECG
- Standard echocardiography (LVEF, chamber size, diastolic function)

- Basic biomarkers (troponin when available)

Two representative clinical cases were selected to illustrate typical diagnostic challenges and decision-making pathways.

The analytical framework focused on correlating:
mechanism of injury → myocardial structural change → clinical manifestation → detectable low-cost marker

Results

From Molecular Injury to Clinical Signal

Cardiotoxicity begins at the cellular level. Anthracyclines induce oxidative stress, mitochondrial dysfunction, and DNA damage, leading to apoptosis of cardiomyocytes. Trastuzumab interferes with HER2-mediated survival pathways, impairing myocardial repair.

These early changes are **microscopic and subclinical**, but they trigger:

- Reduced contractile reserve
- Early diastolic dysfunction
- Subtle changes in myocardial deformation (GLS-level changes)

In the absence of GLS, these alterations can still manifest as:

- Mild fatigue disproportionate to anemia
- Slight reduction in exercise tolerance
- Early ECG repolarization abnormalities
- Diastolic dysfunction on echocardiography (E/A reversal, E/e′ increase)

What to Look for in Echocardiography (Without GLS)

In low-resource settings, echocardiography becomes the cornerstone—but only if interpreted beyond LVEF.

Clinicians should actively assess:

- **Diastolic function (EARLY marker)**
  - E/A ratio < 1
  - Increased E/e′ (suggesting elevated filling pressures)
- **Subtle LVEF trend (not absolute value)**
  - A drop from 65% → 55% is clinically significant
- **Left ventricular geometry**
  - Early dilation or increased wall stress

- **Mitral annular tissue Doppler (e′ velocity)**
  - Reduced values indicate impaired relaxation

In many patients, **diastolic dysfunction precedes systolic decline**.

Clinical Case 1 — Silent Early Cardiotoxicity

A 52-year-old woman with breast cancer receiving doxorubicin presented with mild fatigue. Hemoglobin was normal. LVEF remained 60%.

However:

- E/A ratio decreased
- E/e′ mildly elevated
- New nonspecific ST-T changes on ECG

Interpretation: early myocardial injury despite preserved LVEF
Action: initiation of ACE inhibitor and close monitoring
Outcome: prevention of overt heart failure

Clinical Case 2 — Delayed Detection

A 60-year-old patient on trastuzumab therapy presented late with dyspnea.

Findings:

- LVEF reduced to 40%
- LV dilation
- Prior subtle symptoms were overlooked

Interpretation: missed early phase
Outcome: treatment interruption and partial recovery only

Cardiotoxic Drugs and Mechanisms

Drug/Class	Mechanism of Cardiotoxicity	Clinical Pattern
Anthracyclines	ROS, mitochondrial damage, apoptosis	Dose-dependent cardiomyopathy
Trastuzumab	HER2 blockade → impaired repair	Reversible LV dysfunction
Cyclophosphamide	Endothelial damage, myocarditis	Acute heart failure
Tyrosine kinase inhibitors	Vascular dysfunction, hypertension	Ischemia, LV dysfunction
Immune checkpoint inhibitors	Autoimmune myocarditis	Acute, severe myocarditis

Discussion

The central message of this study is that **cardiotoxicity is not invisible without advanced tools—it is simply under-recognized**.

The reliance on LVEF alone creates a dangerous diagnostic delay. By the time LVEF declines, substantial myocardial damage has already occurred. In contrast, integrating clinical observation with simple echocardiographic and ECG findings allows earlier recognition.

In low-resource settings, the clinician's role becomes even more critical. Diagnosis shifts from technology-driven to **thinking-driven medicine**.

A key conceptual shift is needed:

Instead of asking "Is LVEF normal?", clinicians should ask
"Is this myocardium behaving normally?"

Clinical Algorithm for Low-Resource Settings

Step 1 — Baseline before therapy

- Clinical exam
- ECG
- Echocardiography (LVEF + diastolic function)

Step 2 — During therapy

- Monitor symptoms (fatigue, dyspnea)
- Repeat ECG
- Echo every 3 cycles (focus on trends, not thresholds)

Step 3 — Red flags

- New fatigue without anemia
- Diastolic dysfunction
- LVEF drop ≥10% (even if >50%)

Step 4 — Action

- Start cardioprotection (ACE inhibitors, beta-blockers)
- Consider modifying chemotherapy

Cardioprotective Strategies

Effective cardioprotection must be proactive, not reactive.

- **ACE inhibitors / ARBs**

Improve remodeling and reduce progression

- **Beta-blockers (especially carvedilol)**

Reduce oxidative stress and myocardial workload

- **Dexrazoxane (selected cases)**

Reduces anthracycline-induced injury

- **Strict blood pressure control**

Particularly important with targeted therapies

Limitations

This analysis is based on a limited retrospective cohort and lacks advanced imaging confirmation such as GLS or cardiac MRI. However, this limitation reflects real-world conditions in many clinical settings and underscores the practical relevance of the proposed model.

Conclusions

Early detection of cardiotoxicity does not depend solely on advanced technology. By understanding the continuum from molecular injury to clinical manifestation, clinicians can identify early warning signs using accessible tools.

In resource-limited environments, **careful clinical reasoning, combined with targeted use of ECG and echocardiography, can bridge the gap between late diagnosis and timely intervention**.

Ultimately, preserving the heart during cancer therapy is not only possible—it is essential for truly improving patient survival and quality of life.

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