

Expert Consensus for Multimodality Imaging Evaluation of Adult Patients during and after Cancer Therapy: A Report from the American Society of Echocardiography and the European Association of Cardiovascular Imaging

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Abbreviations

ASE = American Society of Echocardiography
BNP = Brain-type natriuretic peptide
CAD = Coronary artery disease
CMR = Cardiac magnetic resonance
CTRCD = Cancer therapeutics-related cardiac dysfunction
DTI = Doppler tissue imaging
EACVI = European Association of Cardiovascular Imaging
EAE = European Association of Echocardiography
GLS = Global longitudinal strain
HF = Heart failure
LGE = Late gadolinium enhancement
LV = Left ventricular
LVEF = Left ventricular ejection fraction
MUGA = Multigated blood pool imaging
NT-proBNP = N-terminal pro-B-type natriuretic peptide
RV = Right ventricular
STE = Speckle-tracking echocardiography
3D = Three-dimensional
3DE = Three-dimensional echocardiography
TnI = Troponin I
2D = Two-dimensional
2DE = Two-dimensional echocardiography
VEGF = Vascular endothelial growth factor

TABLE OF CONTENTS

I. Cancer Therapeutics-Related Cardiac Dysfunction	912
A. Definition, Classification, and Mechanisms of Toxicity	912
1. Definition of Cancer Therapeutics-Related Cardiac Dysfunction (CTRCD)	912
2. Classification by Mechanism of Toxicity	913
a. Type I CTRCD	913
b. Type II CTRCD	913
II. Echocardiographic Evaluation of Cardiac Structure and Function in Cancer Patients	914
A. LV Systolic Function	914
B. LV Diastolic Function	915
C. RV Function	915
D. Valvular Heart Disease	916
E. Pericardial Disease	917
F. 3DE	918
G. Contrast Echocardiography	919
H. Stress Echocardiography	919
I. Other	920
III. Detection of Subclinical LV Dysfunction	920
A. Detection of Subclinical LV Dysfunction Using Imaging	920
1. LVEF as a Tool to Detect Subclinical LV Dysfunction	920
2. Diastolic Dysfunction: Early Signs and Prognostic Value	920
3. Detection of Subclinical LV Dysfunction Using DTI Velocities	922
4. Early Detection of LV Dysfunction Using Strain and Strain Rate	922
B. Detection of Subclinical LV Dysfunction Using Biomarkers	924
1. Troponins	924
2. Other Biomarkers	926
C. An Integrated Approach of Imaging and Biomarkers	926
D. Implications of Early Detection on Therapeutic Approaches	926
IV. Other Imaging Modalities	927
A. Radionuclide Approaches for Monitoring Chemotherapy-Induced Cardiotoxicity	927
1. MUGA	927
2. MUGA Compared with Other Modalities	927
B. CMR for Monitoring CTRCD	928
1. CMR in the Assessment of Cardiac Structure and Function	928
2. CMR and Echocardiography	928
3. Beyond the LVEF: Advanced CMR Assessments	929
C. Specific Challenges	929
V. Integrated Approach	930
A. Baseline Assessment and Monitoring	930
1. Type I Agents	930
2. Type II Agents	930
B. Detection of Subclinical LV Dysfunction	931
Executive Summary	932
Notice and Disclaimer	933
References	933

I. CANCER THERAPEUTICS-RELATED CARDIAC DYSFUNCTION

A. Definition, Classification, and Mechanisms of Toxicity

Cardiac dysfunction resulting from exposure to cancer therapeutics was first recognized in the 1960s, with the widespread introduction of anthracyclines into the oncologic therapeutic armamentarium.¹ Heart failure (HF) associated with anthracyclines was then recognized as an important side effect. As a result, physicians learned to limit their doses to avoid cardiac dysfunction.² Several strategies have been used over the past decades to detect it. Two of them evolved over time to be very useful: endomyocardial biopsies and monitoring of left ventricular (LV) ejection fraction (LVEF) by cardiac imaging. Examination of endomyocardial biopsies proved to be the most sensitive and specific parameter for the identification of anthracycline-induced LV dysfunction and became the gold standard in the 1970s. However, the interest in endomyocardial biopsy has diminished over time because of the reduction in the cumulative dosages used to treat malignancies, the invasive nature of the procedure, and the remarkable progress made in noninvasive cardiac imaging. The noninvasive evaluation of LVEF has gained importance, and notwithstanding the limitations of the techniques used for its calculation, has emerged as the most widely used strategy for monitoring the changes in cardiac function, both during and after the administration of potentially cardiotoxic cancer treatment.³⁻⁵

The timing of LV dysfunction can vary among agents. In the case of anthracyclines, the damage occurs immediately after the exposure⁶; for others, the time frame between drug administration and detectable cardiac dysfunction appears to be more variable. Nevertheless, the heart has significant cardiac reserve, and the expression of damage in the form of alterations in systolic or diastolic parameters may not be overt until a substantial amount of cardiac reserve has been exhausted. Thus, cardiac damage may not become apparent until years or even decades after receiving the cardiotoxic treatment. This is particularly applicable to adult survivors of childhood cancers.

Not all cancer treatments affect the heart in the same way. Therefore these agents cannot be viewed as a single class of drugs.

1. Definition of Cancer Therapeutics-Related Cardiac Dysfunction (CTRCD). Different definitions of CTRCD have been used historically.⁷ It is the consensus of this committee to define CTRCD as a decrease in the LVEF of >10 percentage points, to a value <53% (normal reference value for two-dimensional (2D) echocardiography (2DE) (see Section II). This decrease should be confirmed by repeated cardiac imaging. The repeat study should be

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