ESC Cardio-Oncology 2026: Heart Failure Phenotyping in Cancer

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Heart failure (HF) is a significant comorbidity in cancer patients, often arising from cardiotoxic cancer therapies or shared risk factors.1 Differentiating HF phenotypes in this population is critical for guiding treatment and improving outcomes, yet current diagnostic approaches can be complex due to overlapping symptoms and treatment effects. The ESC Cardio-Oncology 2026 session on heart failure phenotyping in patients with cancer explored the utility of advanced imaging and clinical algorithms to inform therapeutic strategies.

KEY TAKEAWAYS

- The Pivot: The integration of advanced cardiac imaging with clinical data offers a more precise approach to phenotyping heart failure in cancer patients.
- The Data: Early detection of subclinical myocardial dysfunction via strain imaging can precede symptomatic heart failure by months to years in patients receiving cardiotoxic chemotherapy.
- The Action: Clinicians should consider implementing routine, comprehensive cardiac imaging protocols, including global longitudinal strain (GLS), for cancer patients at risk of cardiotoxicity to enable timely intervention.

The intersection of oncology and cardiology presents unique challenges, particularly in the management of heart failure (HF) in cancer patients. Cardiotoxicity, a common adverse effect of many cancer treatments, can manifest as various HF phenotypes, including heart failure with reduced ejection fraction (HFrEF), heart failure with preserved ejection fraction (HFpEF), and stress-induced cardiomyopathy.1 Accurate phenotyping is essential for tailoring treatment, as therapies effective for one HF phenotype may be ineffective or even harmful for another.2 The prevalence of HF in cancer survivors is increasing, driven by improved cancer survival rates and the cardiotoxic effects of modern cancer therapies. Understanding the specific mechanisms by which different cancer treatments induce myocardial damage is crucial for developing targeted prevention and treatment strategies. For instance, anthracyclines are known to cause dose-dependent myocardial injury through oxidative stress and topoisomerase II²inhibition, leading primarily to HFrEF. In contrast, HER2-targeted therapies like trastuzumab can cause reversible myocardial dysfunction without significant myocyte necrosis, often presenting as HFrEF. Immune checkpoint inhibitors, while highly effective against various cancers, can induce myocarditis, a distinct form of cardiotoxicity that can rapidly progress to severe HF. These diverse mechanisms underscore the necessity of precise HF phenotyping in this vulnerable population.

Traditional methods of HF diagnosis, such as left ventricular ejection fraction (LVEF) assessment, may not fully capture the spectrum of myocardial dysfunction in cancer patients. For instance, LVEF can remain within normal limits despite significant subclinical myocardial injury, particularly in HFpEF.3 The ESC Cardio-Oncology 2026 session highlighted

the need for more sophisticated diagnostic tools to identify specific HF phenotypes early, thereby enabling proactive management and potentially mitigating disease progression.4 This early identification is particularly critical given that many cancer patients are already burdened by comorbidities and the physiological stress of their underlying malignancy and its treatment. Delaying the diagnosis of cardiotoxicity can lead to irreversible myocardial damage and significantly impact both quality of life and long-term survival.

Imaging Findings and Clinical Decisions

The session emphasized the role of advanced cardiac imaging techniques in characterizing myocardial changes in cancer patients. Echocardiography, particularly speckle-tracking echocardiography for global longitudinal strain (GLS), was presented as a sensitive marker for detecting subclinical myocardial dysfunction before changes in LVEF become apparent.5 A reduction in GLS by >10-15% from baseline, or an absolute GLS value of , has been identified as an early predictor of cardiotoxicity in patients receiving anthracyclines or trastuzumab.6 This early detection allows for the initiation of cardioprotective therapies, such as beta-blockers or ACE inhibitors, potentially preventing overt HF.7 The application of GLS extends beyond anthracycline and trastuzumab cardiotoxicity, showing promise in monitoring patients on other cardiotoxic agents, including certain tyrosine kinase inhibitors and immune checkpoint inhibitors, where early detection of myocardial dysfunction can guide treatment modifications.

Cardiac magnetic resonance imaging (CMR) offers comprehensive tissue characterization, providing insights into myocardial fibrosis, inflammation, and edema, which are critical for differentiating HF phenotypes.8 For example, late gadolinium enhancement (LGE) on CMR can identify areas of myocardial fibrosis, which is common in HFpEF and can be exacerbated by certain cancer treatments.9 CMR also provides accurate and reproducible measurements of ventricular volumes and function, which are particularly valuable in complex cases or when echocardiographic windows are suboptimal.10 The ability of CMR to precisely quantify myocardial tissue characteristics, such as T1 and T2 mapping for diffuse fibrosis and edema, offers a deeper understanding of the underlying pathology contributing to HF phenotypes in cancer patients, aiding in more personalized treatment strategies.

Beyond imaging, the session discussed the integration of clinical biomarkers and risk stratification models. Elevated levels of N-terminal pro-B-type natriuretic peptide (NT-proBNP) and high-sensitivity cardiac troponin (hs-cTn) can indicate myocardial stress and injury, respectively.11 Serial monitoring of these biomarkers, in conjunction with imaging, can help identify patients at highest risk for developing HF and guide the intensity of surveillance and intervention.12 The discussion underscored that a multidisciplinary approach, involving cardiologists, oncologists, and imaging specialists, is paramount for optimal patient care.13 This collaborative model ensures that cancer treatment efficacy is balanced with cardiovascular safety, leading to improved overall outcomes for patients.

Limitations in current practice include the variability in imaging protocols across institutions and the lack of standardized guidelines for integrating advanced imaging into routine cardio-oncology care.14 The cost and accessibility of advanced imaging modalities like CMR also present barriers, particularly in resource-limited settings. Furthermore, the optimal frequency and duration of surveillance for cardiotoxicity remain areas of ongoing research, with current recommendations often varying based on the specific cancer therapy and patient risk factors. Future directions involve developing more precise risk prediction models that combine clinical, imaging, and biomarker data, as well as exploring novel imaging techniques and artificial intelligence applications to automate and standardize the assessment of myocardial function and tissue characteristics. The integration of genetic and genomic markers may also offer future

avenues for identifying individuals at higher risk of cardiotoxicity, allowing for even more personalized prevention strategies.

CLINICAL IMPLICATIONS

The emphasis on advanced heart failure phenotyping at ESC Cardio-Oncology 2026 underscores a critical shift in managing cardiotoxicity in cancer patients. Relying solely on left ventricular ejection fraction (LVEF) is no longer sufficient; clinicians must adopt more sensitive tools like global longitudinal strain (GLS) to detect subclinical myocardial dysfunction. This means that for patients undergoing potentially cardiotoxic therapies, such as anthracyclines or HER2-targeted agents, a baseline echocardiogram with GLS and subsequent serial monitoring should become standard practice, not an optional extra. Failure to implement these more precise diagnostic methods risks delaying cardioprotective interventions, leading to avoidable heart failure hospitalisations and poorer long-term outcomes for patients already facing a cancer diagnosis.

The pharmaceutical industry has a clear interest here. Early identification of cardiotoxicity creates an expanded window for cardioprotective agents, including established beta-blockers and ACE inhibitors, but also for newer therapies. Companies developing novel cardioprotective drugs or those with existing cardiovascular portfolios should be actively engaging with cardio-oncology guidelines to ensure their products are considered in these evolving management algorithms. Furthermore, the push for advanced imaging highlights a potential market for improved imaging software and AI-driven analysis tools that can standardise and streamline the interpretation of complex echocardiographic and CMR data, reducing variability and improving diagnostic accuracy across different clinical settings.

For patients, these advancements offer the prospect of a more personalised and proactive approach to managing their cardiovascular health while undergoing cancer treatment. The ability to detect cardiac injury before symptoms develop means interventions can be initiated earlier, potentially preserving cardiac function and improving quality of life. However, this also places a greater burden on healthcare systems to ensure access to these advanced imaging modalities and the specialist expertise required for their interpretation. Without equitable access, the benefits of these sophisticated phenotyping strategies will remain confined to well-resourced centres, exacerbating disparities in cancer and cardiovascular care.

EDITORIAL & AI STANDARDS

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