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Cardiovascular Magnetic Resonance Imaging in Cardio-Oncology: An Expert Consensus Statement from the Society for Cardiovascular Magnetic Resonance. Endorsed by the International Cardio-Oncology Society (ICOS)CMR in Cardio-Oncology

Lauren A. Baldassarre, Samuel W. Reinhardt, Jennifer H. Jordan, W. Patricia Bandettini, Ana Barac, Chiara Bucciarelli-Ducci, Juliano de Lara Fernandes, Susan Dent, Vanessa Ferreira, Marianna Fontana, Luba Frank, W. Gregory Hundley, Mohamad H. Khattab, Tim Leiner, Juan Lopez-Mattei, Vineeta Ojha, Carlos E. Rochitte, Jeanette Schulz-Menger, M. Barbara Srichai-Parsia, Paaladinesh Thavendiranathan, Olga Toro-Salazar, Jonathan Weinsaft, Charlotte Manisty

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Cardiovascular Magnetic Resonance Imaging in Cardio-Oncology: An Expert Consensus Statement from the Society for Cardiovascular Magnetic Resonance. Endorsed by the International Cardio-Oncology Society (ICOS)

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Abstract:

This document represents a position statement from the Society for Cardiovascular Magnetic Resonance (SCMR) on recommendations for clinical utilization of cardiovascular magnetic resonance (CMR) in patients with cancer. The document was prepared by the SCMR Consensus Group on CMR Imaging for Cardio-Oncology and endorsed by the SCMR Publications Committee and SCMR Executive Committee. This document aims to: (1) guide the informed selection and timing of CMR; (2) inform clinical decision-making based on CMR findings; (3) educate stakeholders on the advantages and limitations of CMR in specific clinical scenarios; and (4) provide suggested protocols to tailor the CMR exam to the particular clinical question. The statements of clinical utility presented in the current document focus on the following clinical scenarios: evaluating left ventricular dysfunction with ejection fraction and volumes; identifying pre-clinical left ventricular (LV) dysfunction with myocardial strain; and myocardial injury or edema with parametric mapping and late gadolinium enhancement (LGE); myocarditis; vascular toxicity, with a focus on coronary artery disease (CAD); iron overload; cardiac amyloidosis; pericardial disease; valvular disease; pulmonary hypertension; and cardiac masses. Sections devoted to the special situations of radiation therapy (RT), hematopoietic stem cell transplantation (HSCT), and survivorship and pediatrics are also included. As an expert consensus statement, some recommendations are based on expert opinion and accepted best practices. This position statement emphasizes the optimal utilization of CMR in oncology patients with known or suspected cardiovascular disease; however, the authors acknowledge that CMR is not always locally available or accessible. Finally, the principal aim of this position statement is to provide specific recommendations on the use of CMR to help improve the health of cancer patients with cardiovascular disease.

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Key words: cardiovascular magnetic resonance, cardio-oncology, cardiotoxicity, cancer

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List of Abbreviations

2D, two-dimensional
3D, three-dimensional
4Ch, four chamber
4D, four-dimensional
ACC, American College of Cardiology
ACR, American College of Radiology
AHA, American Heart Association
AL, light chain (amyloidosis)
ASCO, American Society of Clinical Oncology
ASE, American Society of Echocardiography
ATTR, transthyretin amyloidosis
BCR-ABL, Breakpoint Cluster Region-Abelson Murine Leukemia Viral Oncogene Homolog 1
bSSFP, balanced steady state free precession
CA, cardiac amyloidosis
CAD, coronary artery disease
CAYA, child and young adult
CAR, chimeric antigen receptor
CMR, cardiovascular magnetic resonance
CTRCD, chemotherapy related cardiac dysfunction
CV, cardiovascular
DENSE, displacement encoding with stimulated echoes
EACVI, European Society of Cardiovascular Imaging
ECV, extracellular volume
ESC, European Society of Cardiology
EF, ejection fraction
EGE, early gadolinium enhancement
eGFR, estimated glomerular filtration rate
EMB, endomyocardial biopsy
ESC, European Society of Cardiology
FFR, fractional flow reserve
GBCA, gadolinium-based contrast agent
GCS, global circumferential strain
GLS, global longitudinal strain
Gy, Gray
HER2, human epidermal growth factor receptor 2
HF, heart failure
HSCT, hematopoietic stem cell transplantation
ICI, immune checkpoint inhibitor
IOC, iron overload cardiomyopathy
IRAE, immune related adverse events
JACC, Journal of the American College of Cardiology
L, distance between ascending and descending thoracic aorta flow measurements (in pulse wave velocity calculation)
LA, left atrium/left atrial

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LGE, late gadolinium enhancement
LV, left ventricle/left ventricular
MACE, major adverse cardiovascular event
MUGA, multigated acquisition
MRA, magnetic resonance angiography
OR, odds ratio
PA, pulmonary artery
PH, pulmonary hypertension
PSIR, phase sensitive inversion recovery
PWV, pulse wave velocity
Qp:Qs, pulmonary to systemic flow ratio
RT, radiation therapy
RV, right ventricle/right ventricular
SAx, short axis
SCMR, Society for Cardiovascular Magnetic Resonance
SENC, strain-encoded imaging
SPAIR, spectral attenuated inversion recovery
SPAMM, spatial modulation of magnetization
SSFP, steady state free precession
STIR, short-tau inversion recovery
SUCCOUR-MRI, Strain surveillance during chemotherapy for improving cardiovascular outcomes
ΔT, wave foot-to-foot time (in pulse wave velocity calculation)
T1w, T1-weighted
T2w, T2-weighted
TI, inversion time
TSE, turbo spin echo
TTR, transthyretin
TSE, turbo spin echo
VEGF, vascular endothelial growth factor

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Recommendations for the clinical utility of CMR in patients with cancer and cancer survivors

As this is an expert consensus statement, rather than a clinical guideline, some of these recommendations are based on limited data, as outlined in detail in the individual sections, along with expert opinion and accepted best practices.

1. CMR should be performed for assessment of left ventricular (LV) systolic function (including ejection fraction [EF]) assessment when echocardiography is non-diagnostic, not available or when a confirmatory test is needed for diagnosis of cancer therapy related cardiac dysfunction (CTRCD). Where access is not a limiting factor, CMR may also be considered as a first-line modality, given its greater precision and reproducibility.
2. Global longitudinal strain measurement may be considered as an adjunct to conventional indices of systolic function (e.g., LVEF, wall motion) when CMR is used for serial monitoring and for evaluation of CTRCD.
3. Abbreviated non-contrast protocols focused on functional assessment should be considered when CMR is used for serial monitoring for CTRCD.
4. Multiparametric CMR including late gadolinium enhancement imaging should be performed in the event of suspected CTRCD or new onset LV systolic dysfunction to help quantify and understand the etiology.
5. Multiparametric CMR including late gadolinium enhancement imaging should be considered for detection of myocarditis from cancer therapeutics in all suspected cases, including when echocardiography is normal. Immune checkpoint inhibitor (ICI)-associated myocarditis is a clinical diagnosis and a normal CMR alone does not exclude the diagnosis and should not delay clinically indicated management.

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6. Stress CMR including late gadolinium enhancement imaging should be considered for risk stratification prior to administration of cancer therapies associated with a high risk of ischemic or vascular toxicity in patients with ischemic symptoms.
7. Stress CMR with late gadolinium enhancement imaging should be considered during or following cardiotoxic cancer therapies for investigation of suspected ischemia.
8. CMR including parametric mapping and late gadolinium enhancement imaging should be considered in hematology patients with known plasma cell dyscrasias for investigation of suspected cardiac amyloidosis.
9. CMR including parametric mapping and late gadolinium enhancement imaging should be performed in patients with known extra-cardiac amyloidosis (of all types) with suspected cardiac involvement.
10. CMR including parametric mapping and late gadolinium enhancement imaging may be considered for patients with known cardiac amyloidosis for monitoring organ response to treatment.
11. CMR including T2* imaging should be performed for the quantification of myocardial iron in hematology and oncology patients with suspected iron overload.
12. CMR should be considered for the evaluation of pericardial disease in oncology patients, including for evaluation of constriction.
13. CMR should be considered for the evaluation of valve disease associated with cancer therapeutics, especially when echocardiography is not diagnostic.
14. CMR should be considered for the assessment of right ventricular structure and function in oncology patients with pulmonary hypertension.

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15. CMR should be considered in patients post radiation therapy or hematopoietic stem cell transplantation for evaluation of suspected cardiotoxic effects.
16. CMR should be performed in pediatric oncology patients for (LVEF) assessment when diagnostic echocardiography cannot be obtained, or when left ventricular function is abnormal or borderline abnormal by echocardiography.
17. CMR may be considered for serial monitoring in pediatric oncology patients at moderate or high risk for cardiotoxicity.
18. CMR including multiparametric assessment should be performed for identification and tissue characterization of cardiac masses and adjacent structures, and for assessing response to cancer treatment or surgery.

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Table 1. Clinical Scenarios and Recommended Role of CMR in Cancer Survivors

Clinical Scenario	**Recommended Role of CMR**
Baseline assessment before cardiotoxic cancer therapy	Gold standard for LV volumes, EF, and mass, used particularly when echocardiography is suboptimal quality or LVEF is borderline.
Serial monitoring during/after therapy	High precision for detecting small changes in EF and volumes. Abbreviated non-contrast protocols may be considered for repeated monitoring.
Suspected subclinical LV dysfunction	Myocardial strain (feature tracking, tagging, or SENC/DENSE) to detect early changes prior to EF decline.
Suspected myocardial injury or inflammation	Parametric mapping (T1, T2, ECV) and LGE to detect edema, fibrosis, or myocarditis.
Coronary artery disease / ischemia evaluation	Stress CMR with perfusion and LGE for risk stratification in patients with chest pain/suspected ischemia.
Iron overload	T2* imaging for myocardial iron quantification (± liver iron); complementary T1 mapping if available.
Cardiac amyloidosis	LGE plus T1 mapping / ECV for diagnosis, prognosis, and treatment monitoring in patients with known or suspected amyloidosis.
Pericardial disease	Cine imaging, T2 (edema), and LGE for inflammation, fibrosis, and constrictive physiology assessment.
Valvular disease	Phase-contrast velocity mapping and cine imaging for quantification when echocardiography is non-diagnostic.
Pulmonary hypertension	RV size/function, flow quantification, pulmonary MRA, and LGE for insertion point fibrosis.
Radiation therapy survivors	Comprehensive assessment for coronary disease, fibrosis, pericardial disease, or valvulopathy.
Hematopoietic stem cell transplant patients	Multiparametric CMR to evaluate cardiomyopathy, myocarditis, or iron overload following hematopoietic stem cell transplant.
Pediatric oncology patients	Preferred when echocardiography is limited; surveillance in moderate/high-risk children for early detection of CTRCD.
Cardiac masses	Multiparametric CMR with cine, LGE, mapping, and perfusion for diagnosis, tissue characterization, and prognostication.

Abbreviations: CMR, cardiovascular magnetic resonance; CTRCD, chemotherapy related cardiac dysfunction; DENSE, displacement encoding with stimulated echoes; ECV, extracellular volume; EF; ejection fraction; ICI, immune checkpoint inhibitor: LGE, late gadolinium enhancement; LV, left ventricle/left ventricular; MRA, magnetic resonance angiography; RV, right ventricle; SENC, strain encoded.

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Introduction

This document intends to present a wide range of potential clinical questions or scenarios where cardiovascular magnetic resonance (CMR) can prove helpful in guiding care for cancer survivors, as summarized in the recommendation statements. Each scenario provides a detailed discussion of CMR's strengths in addressing the specific clinical question, and most also include a recommended protocol for obtaining the necessary images. Our aim is to provide a useful reference that will be broadly applicable, but, as with all consensus documents, these protocols are meant to be starting points that should be tailored to individual patients and their particular circumstances. A summary of clinical scenarios and corresponding CMR techniques that may prove useful is included in Table 1. The recommendations are broadly aligned with international societal guidelines and appropriateness criteria, but provide greater detail both for clinicians caring for cancer survivors, and for cardiologists and radiologists delivering CMR services to these individuals

Methodology

The Society for Cardiovascular Magnetic Resonance (SCMR) Cardio-Oncology Special Interest Group has identified the need for an Expert Consensus Statement to provide guidance for clinicians involved in the cardiovascular care of cancer survivors, including CMR physicians.

The expert panel and writing group for this document were formed following guidance of the SCMR Executive Committee and membership was approved by the SCMR Board of Trustees. The expert panel and writing group included cardiologists (both cardio-oncology and cardiac imaging) and radiologists from diverse geographical locations, with both adult and pediatric expertise, as well as a clinical oncologist. The writing group reviewed the literature and

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prepared a manuscript draft which was reviewed and edited by the expert panel. Each recommendation was discussed and agreed by majority. The document was then reviewed by an external group of experts in the field of CMR under the leadership of the SCMR Publications Committee.

Results

Clinical Scenarios

Assessment for left ventricular dysfunction: ejection fraction, volumetric analysis, and myocardial mass quantification

Guidelines and consensus statements from cardiovascular (CV) and oncology societies state that baseline cardiac imaging evaluation should be performed in any patient scheduled to receive potentially cardiotoxic cancer therapy associated with heart failure (HF) or left ventricular (LV) dysfunction.[1-7] This aids risk stratification in patients with reduced or borderline function at greatest risk of cardiotoxicity[8, 9], while also generating an individualized reference LV ejection fraction (EF) measurement against which future surveillance imaging results can be compared.

Current guidelines recommend echocardiography as the first-line imaging modality for LVEF assessment, incorporating 3D imaging where feasible. [3-7, 10] CMR should be performed as a second line modality[3-7, 10], given that it is considered the gold standard for noninvasive assessment of volumes and function [11, 12] and holds particular utility when echocardiography cannot accurately assess these measures (e.g., poor acoustic windows) and/or when clinical decisions require a more precise quantification when complementary imaging

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method measurements are borderline for normality.[4] CMR is well-suited to serial monitoring where detection of significant changes in LVEF govern clinical decision making (altering cancer therapy and diagnosis of pre-clinical cardiotoxicity) due to its high measurement precision. [13, 14] Functional assessment, especially with serial imaging, is preferred with CMR as opposed to MUGA given the radiation exposure associated with the latter. Recent data from the Strain surveillance during chemotherapy for improving cardiovascular outcomes (SUCCOUR-MRI) trial provide support for a strain-guided approach using echocardiography for administration of cardioprotective medications to reduce the primary outcome of CMR-defined changes in LVEF.[15] Of note, the endpoint of LVEF as measured by echocardiography was not met in the trial, likely due to increased precision of LVEF measurement by CMR.

For patients undergoing assessment for cancer therapy related cardiac dysfunction (CTRCD) by CMR, a stack of breath-held segmented balanced steady state free precession (bSSFP) short-axis cines slices to cover the LV should be acquired, with post-processing as discussed in the 2020 SCMR Position Statement to determine volumes, mass, and function.[16] In patients who have difficulty with breath-holding, a real-time triggered free-breathing cine stack may be considered, recognizing that the temporal and spatial resolution result in higher measurement variability than segmented breath-held cines. Compressed sensing techniques can also help decrease breath-hold requirements for patients.

There are 3 specific considerations to improve measurement performance in this context:

- The LV cine stack should be aligned to the mitral valve to minimize measurement variability between serial scans. LVEF should be interpreted in the context of LV end-diastolic and end-systolic volumes; patients with cancer may be volume depleted (sepsis, vomiting) and potentially curative cancer therapy may be

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inadvertently withheld due to an abnormal LVEF in the context of volume depletion.[17, 18]

- LV mass may be an important marker in anthracycline-treated patients as reductions in LV mass have been associated with HF symptoms and may portend worse outcomes.[19, 20]
- Serial imaging should be performed using the same sequences utilized on baseline imaging, with analysis performed using consistent segmentation techniques (e.g. trabeculated versus smooth contours).

Parameters for detection of subclinical LV dysfunction: Myocardial strain quantification.

Myocardial strain is a robust measure to identify subclinical cardiomyopathy, and abnormalities may manifest prior to clinical deterioration of LVEF.[21] Strain may be derived from standard SSFP cine images using feature tracking or measured using dedicated sequences. The latter includes tagging, phase velocity mapping, displacement encoding with stimulated echoes (DENSE), and more recently, strain-encoded imaging (SENC).[22]

Strain may aid both CTRCD risk stratification prior to initiation of potentially cardiotoxic cancer therapy and early detection of subclinical cardiac dysfunction to forecast future CTRCD risk in cancer survivors during and following completion of treatment..[23, 24] Although studies examining pre-treatment risk assessment are limited, a recent study using a fast-SENC protocol demonstrated that the proportion of myocardial segments with normal (≤-17.0%) longitudinal or circumferential strain identified subsequent risk of CTRCD in anthracycline-treated patients.[23] Having <80% of the myocardial segments with normal strain (defined based on 16 longitudinal and 21 circumferential strain segments) had modest discriminatory value for future CTRCD. However, current literature lacks large prospective studies on the prognostic significance of

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CMR-defined impaired strain in cancer survivors, and further research is needed to define the role of CMR strain imaging in clinical practice.

During cancer therapy, changes in global circumferential (GCS) and/or global longitudinal strain (GLS), measured from feature tracking methods or tagging data, track changes in LVEF in the context of anthracycline or trastuzumab therapy.[24-30] In addition, an early 15% relative change in CMR GLS or GCS (measured using tagging or feature tracking) in women receiving anthracyclines followed by trastuzumab therapy was recently shown to identify risk of future CTRCD with an odds ratio (OR) of 1.47-1.83.[24] However, the incremental value over CMR-measured LVEF changes was limited. In a separate study, a combination of ≥10% worsening of GCS and an increase in left ventricular end-systolic volume at 3 months identified anthracycline-treated patients who had a >5% reduction in LVEF from baseline at two years of follow-up.[31]

It is important to consider that temporal resolution impacts strain measurement (with higher temporal resolution associated with higher strain values), hence images should be acquired using temporal resolutions ≥30 frames/cardiac cycle.[32] Recent work has also highlighted the need to consider strain changes in the context of a patient's volume status,[17, 18] and the importance of validating a strain change with repeated testing before making clinical decisions. CMR-derived strain continues to face challenges with reproducibility, techniques and differences between vendor software, and as such, while it can be considered in certain situations, it is not broadly used in clinical practice at this time.[33]

Parameters for detection of subclinical left ventricular dysfunction: tissue characterization including T1, T2 and extracellular volume mapping and late gadolinium enhancement

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CMR offers a unique ability to provide tissue characterization including late gadolinium enhancement (LGE) and parametric mapping techniques of T1, T2, and extracellular volume (ECV) mapping – all within a single imaging exam. Focal fibrosis detectable with LGE is rarely seen in patients with CTRCD related to anthracycline or human epidermal growth factor receptor 2 (HER2)-targeted therapies, however other causes should be considered in patients presenting with new onset LV dysfunction. All patients being monitored for CTRCD using CMR should therefore have a comprehensive baseline study including LGE, with focused non-contrast protocols considered for subsequent surveillance scans. For patients with new LV dysfunction, LGE should be administered in line with standard protocols.[34]

Anthracycline-based chemotherapies have been associated with increased interstitial myocardial fibrosis measured with T1 or ECV both in patients undergoing chemotherapy, and in cancer survivors.[20, 35, 36] Several studies (both in patients and large animal models) have suggested that interstitial fibrosis follows a period of myocardial edema, as measured with elevated T2 values.[37-39] Early changes in T1-based and T2-based measures during chemotherapy have however shown mixed results as early predictors of future CTRCD.[38-42] Several factors likely contribute to the difference in study findings including trial design (small study numbers, mixed imaging intervals, variable treatment protocols, heterogeneity within patient populations and risk factor profiles), measurement noise, changes in blood T1 values and the relatively small effect size.[38-42]

When available, native T1 mapping and T2 mapping should be included in cardio-oncology CMR protocols in short-axis planes following SCMR consensus recommendations for mapping[43] and post-processing.[16] Further, if advanced mapping sequences are unavailable, T2-weighted (T2w) short-tau inversion recovery (STIR) or spectral attenuated inversion recovery

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(SPAIR) imaging may be used to identify acute inflammation and myocardial edema; additionally, STIR and SPAIR are useful for assessment of acute inflammation and edema in patients with cardiac implantable electronic devices.

Suggested Protocol

- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and right ventricular (RV) volumes, regional contractility, and EF.
- Optional: strain imaging: (feature tracking from bSSFP cines or SENC/DENSE/tagging)
- Native T1 mapping for evaluation of edema, diffuse fibrosis, or infiltration.
- Native T2 mapping (preferred) or T2w STIR imaging for detection of myocardial edema.
- T1-weighted (T1w) LGE, ideally with both magnitude and phase sensitive inversion recovery (PSIR) reconstructions, for detection of fibrosis/scar at baseline or if cardiac dysfunction is present.
- Post-contrast T1 mapping for calculation of extracellular volume (ECV).

Myocarditis

Myocarditis has been reported with a variety of cancer therapeutics, and the utility of CMR tissue characterization is highest in the evaluation of suspected immune checkpoint inhibitor (ICI) myocarditis, [44-47] for which it has a class 1 indication.[48-51] CMR has been established as the gold standard noninvasive diagnostic test for myocarditis in general, and it is the preferred non-invasive imaging modality test when evaluating for myocarditis associated with cancer therapy. It should be used alongside serum biomarkers, electrocardiogram, left heart catheterization, coronary computed tomographic angiography, and endomyocardial biopsy

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(EMB) for the assessment of myocarditis associated with cancer therapy..[3, 45, 52-55] CMR interpretation should follow the updated Lake Louise Criteria, [43, 56, 57] with myocarditis diagnosed if at least one increased T2-based measure (T2w or T2 map) identifies myocardial edema and at least one increased T1-based measure (native T1, ECV, LGE) for non-ischemic myocardial injury is present; supporting criteria include the presence of pericarditis or systolic LV dysfunction.[57]

The most widely described cases of myocarditis with cancer treatments are associated with ICI use, and ICI-associated myocarditis shows high rates of major adverse cardiovascular events (MACE).[58-62] LVEF is commonly within the normal range; in the largest published international registry of 103 patients diagnosed with ICI-associated myocarditis with CMR data and histopathological findings[63], 61% of patients had an LVEF ≥50%. LGE was present in <50% of patients, while elevated T2-STIR was present in only 28%. These findings suggested low sensitivity of CMR, possibly due to the dynamic nature of edema and inflammation, with rates of LGE increasing from 21.6% to 72% when CMR was performed on day 4 of admission or later. This suggests that the timing of CMR matters, though it is currently not standardized due to differences among clinical presentations and local diagnostic pathways. CMR findings also did not correlate well with histology: LGE was present in only 35% of patients with pathological fibrosis, and elevated T2-STIR signal in only 26% with lymphocytic infiltration. Over 5 months of follow up, despite overall high MACE rate of 40% (composite of CV death, cardiogenic shock, cardiac arrest, and complete heart block), neither LGE nor T2-STIR was associated with MACE, suggesting that the absence of LGE or increased T2- STIR signal does not exclude ICI-associated myocarditis.[47, 63] Therefore, given the limited negative predictive value of CMR in

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the assessment of ICI-associated myocarditis, a normal CMR does not rule out the presence of clinical ICI-associated myocarditis.[64]

In patients with non-ICI-associated myocarditis, the presence of LGE portends more than double the risk of MACE compared to those without LGE[65], with extracellular volume (ECV) adding incremental prognostic value.[66] T1 mapping appears to have prognostic value in patients with ICI-associated myocarditis, with increasing T1 values associated with higher risk of MACE, although no significant association was found with myocardial T2 values.[67] These findings highlight the importance of quantitative tissue characterization with CMR for a more sensitive diagnosis, especially with techniques sensitive to the detection of myocardial inflammation and edema, potentially before LGE develops in the early evolution of ICI-associated myocarditis.[61, 63] The use of endomyocardial biopsy (EMB) may still be warranted after a normal CMR especially if clinical suspicion remains high and if the results will change management, as a delayed diagnosis may lead to adverse clinical consequences.[63]

Further prospective studies will help to determine the utility of CMR for risk stratification of patients at risk for myocarditis from cancer therapeutics and routine monitoring during treatment. Chimeric antigen receptor (CAR) T cell therapy is another cancer therapeutic that has been shown to cause troponin elevation (among other toxicities), though the role of CMR in evaluating this entity remains an area for further study.[54] In terms of prognostic value, reduced global longitudinal strain by echo in ICI-associated myocarditis is associated with MACE independent of LVEF.[68] Feature tracking CMR strain has also been demonstrated to be abnormal in this patient cohort, even when LVEF was preserved.[69]

Suggested Protocol (for a detailed myocarditis protocol, please see the 2018 updated Lake Louise Criteria)[57]

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- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF, as well as pericardial effusions.
- Native T1 mapping for evaluation of edema or diffuse fibrosis. [a]
- Native T2 mapping (preferred) or T2w STIR imaging for detection of myocardial edema.[a]
- Optional: strain imaging: (feature tracking from bSSFP cines or SENC/DENSE/tagging)
- T1w LGE, ideally with both magnitude and PSIR reconstructions, for detection of fibrosis/scar
- Post-contrast T1 mapping for calculation of ECV.

[a] *Tissue characterization with whole-heart coverage where possible would provide greater sensitivity in detecting myocarditis.*

Vascular Toxicity

Importantly, over 75% of new cancer diagnoses occur in patients over the age of 55 years.[70] This age group coincides with a majority of new coronary artery disease (CAD) diagnoses.[71] Predisposing factors to CAD have also been implicated as risk factors for some oncological processes, such as inflammation, diabetes mellitus, tobacco use, hyperlipidemia, and hypertension.[72] Additionally, some cancer therapies are associated with myocardial infarction (e.g., platinum drugs[73], vascular endothelial growth factor [VEGF] inhibitors[74-76], small molecule tyrosine kinase inhibitors, ICIs), coronary spasm (e.g., fluoropyrimidines such as 5-fluorouracil), thrombotic events, or additional risk of epicardial coronary artery disease (e.g., radiation therapy).[67, 77-81] In oncology patients who present with chest pain syndromes or with significant drops in left ventricular systolic function, atherosclerotic disease—both epicardial and microvascular—should be considered.

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The ability of CMR to characterize anatomy, function, myocardial perfusion and injury lends itself to evaluating the oncology patient in whom there is suspicion for myocardial ischemia, infarction or embolic events. CMR is especially valuable in the setting of elevated troponin to help differentiate between myocarditis and acute coronary syndrome. Ischemia can be assessed via detection of inducible perfusion abnormalities from first pass myocardial perfusion imaging following administration of vasodilator (adenosine or regadenoson) stress and at rest.[82, 83] This can detect epicardial CAD and may be helpful, in the setting of quantitative perfusion methods, in distinguishing patterns of small vessel disease that may be the result of microvascular endothelial dysfunction, as seen with antibody-based inhibitors of vascular endothelial growth factor (VEGF).[84, 85] Newer perfusion methods provide on-the-scanner, in-line quantitative perfusion maps which add objectivity to interpretation of the perfusion study[86], and novel artificial intelligence approaches to interpreting CMR data may also add predictive value.[87] Acquisition and analysis of quantitative myocardial perfusion maps should be in line with the SCMR Expert Consensus Statement.[82]

In patients with troponin elevations and chest pain syndromes, T1 and T2 parametric mapping may identify specific coronary territories and the "area-at-risk"[43], assisting in differentiating an acute coronary syndrome caused by obstructive CAD from an acute stress-induced cardiomyopathy, also known as Takotsubo syndrome, to which patients with cancer are at increased risk.[88] Additionally, distinct patterns of LGE help to separate patterns of fibrosis that are characteristic of myocardial infarction versus patterns of non-ischemic processes such as myocarditis. Dedicated thrombus imaging with a long inversion time (TI) of 600 ms can help identify intracardiac thrombi or source of embolic events. In the context of acute coronary

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events, the presence of microvascular obstruction on LGE imaging offers additional prognostic information related to likely recovery of LV function.

Many systemic anti-cancer therapies including proteasome inhibitors, VEGF inhibitors, small molecule tyrosine kinase inhibitors and platinum-based therapies can also result in extra coronary vascular toxicity including systemic and pulmonary hypertension and thromboembolic events (stroke, transient ischemic attacks, myocardial infarction, angina, and other arterial events).[76, 89] Anthracyclines are also considered to cause negative arterial remodeling, analogous to aging of the vascular system by more than a decade.[90] Acute changes in Pulse Wave Velocity (PWV) and arterial distensibility have been observed in breast cancer and childhood cancer survivors following chemotherapy,[91-94] although measurement is predominantly limited to the research setting at present. Velocity-encoded CMR with phase contrast sequences allows accurate assessment of the blood flow velocity with a sufficient temporal and spatial resolution to study the propagation of the aortic systolic flow wave. Double oblique views of the thoracic aorta from an MR angiogram or 4-D flow sequence allows placement of representative planes for measurement of blood flow velocity in the ascending and descending thoracic aorta. For assessment of PWV, the distance between the slice location of the ascending and descending thoracic flow measurements (L) is measured from the aortogram or aortic cine image, and is divided by the wave foot-to-foot time (ΔT) it takes for that forward wave to reach the end measuring point ($PWV=L/\Delta T$). Future work in the evaluation of vascular toxicity in the setting of CTRCD is warranted to determine clinical application of this emerging imaging modality.

Suggested Protocol

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- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF.
- Native T2 mapping (preferred) or T2w STIR imaging for detection of myocardial edema or area at risk.
- Stress and rest first pass perfusion imaging (for detection of inducible perfusion abnormalities), ideally including quantitative perfusion mapping.
- Post-contrast TI 600 ms imaging for detection of intracardiac thrombi
- T1w LGE, ideally with both magnitude and PSIR reconstructions, for detection of focal fibrosis and infarction, including embolic infarcts
- MRA, if applicable

Iron Overload

Iron overload cardiomyopathy (IOC) describes a secondary form of cardiomyopathy resulting from the accumulation of iron in the myocardium, most commonly because of genetically determined disorders of iron metabolism or multiple transfusions, such as in certain hematologic conditions including myelodysplastic syndromes.[95, 96] There are two main phenotypes: dilated and restrictive,[97] and both may be reversible with timely management, hence early diagnosis and accurate quantification is key.

The mainstay for the quantitative assessment of cardiac iron deposition is currently CMR-derived T2* relaxation time.[98-100] This technique allows for accurate diagnosis and quantification of myocardial and hepatic iron deposition and appropriate tailoring and monitoring of iron chelation therapy, and hence is considered the modality of choice.[101] More recently, myocardial T1 measurement has been shown to be complementary to T2*

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measurement, with potentially increased sensitivity.[102, 103] If LV dysfunction is found to be reduced, then addition of contrast for subsequent LGE imaging should be considered for full cardiomyopathy workup.

Suggested Protocol

- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF.
- T2* weighted imaging for myocardial iron quantification in mid short axis view (+/- axial slice at the level of the liver for liver quantification)
- T1 mapping in the mid short axis (SAx) view
- Optional: T1w LGE, ideally with both magnitude and PSIR reconstructions, if LV or RV dysfunction is present

Cardiac Amyloidosis

Cardiac amyloidosis (CA) is characterized by widespread amyloid infiltration throughout the extracellular space, and the light chain (AL) type can be seen with hematological dyscrasias including multiple myeloma.[104] Alongside assessing for markers of infiltration by detection of left and/or right ventricular thickening, CMR can assess ventricular function (including longitudinal function by strain analysis), atrial dimensions and tissue characterization. Imaging with LGE facilitates detection of cardiac AL amyloidosis with high diagnostic accuracy[105, 106] and tracks infiltration from an early subendocardial distribution to diffuse transmural involvement. With extensive amyloid infiltration there may be reversal of gadolinium kinetics, with nulling of the myocardium prior to blood pool nulling, due to marked expansion of the myocardial ECV from amyloid infiltration. This can make effective nulling of the myocardium challenging and careful attention to the inversion time scout (TI-scout) is required to select the

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optimal inversion time for the LGE sequence. Phase sensitive inversion recovery (PSIR) sequences reduce the requirement to select the correct inversion time during acquisition of LGE images and hence should be used where available.

Both native T1 mapping and calculation of ECV using pre and post contrast T1 values have been extensively validated as indicators of CA infiltration,[107, 108] and provide quantitative surrogate markers of amyloid burden. Elevated native T1 and ECV values can detect early disease with good diagnostic accuracy,[108] and have been associated with poor prognosis.[109, 110] T2 values are also elevated in amyloidosis[111] and predict mortality in the AL form, supporting an independent role for myocardial edema in outcomes.[111] The role of CMR in CA is evolving beyond diagnostic and prognostic utility, to provide a unique tool to monitor amyloid load in the heart serially and hence track response to treatment.[112, 113]

Expert consensus recommendations have recently been established for multimodality imaging in cardiac amyloidosis.[114, 115] These consensus documents detail the diagnostic and prognostic utility of CMR in cardiac amyloidosis, appropriateness of CMR imaging, as well as standardized imaging, interpretation, and reporting techniques. CMR is deemed appropriate for diagnostic screening in patients either with suggestive echocardiograms or bone scintigraphy, with known plasma cell dyscrasias and cardiac symptoms or elevated biomarkers, or with systemic (extra-cardiac) amyloidosis. For patients with proven cardiac amyloidosis, CMR is recommended to quantify amyloid burden at diagnosis, for assessing response to therapy and to investigate new symptoms or signs. Furthermore, ECV demonstrated additional diagnostic and prognostic utility beyond established blood and imaging biomarkers across a spectrum of transthyretin amyloidosis (ATTR) disease that included transthyretin (TTR) variant carriers, patients with extracardiac ATTR amyloidosis, and those with early-stage or overt ATTR

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cardiomyopathy.[116] For patients with renal amyloidosis and significant renal impairment (estimated glomerular filtration rate [eGFR] <15mls/min or dialysis dependent), there have been concerns regarding administration of gadolinium based contrast agents (GBCA), due to concerns regarding nephrogenic systemic fibrosis. Newer recommendations highlight that with Group II GBCA agents the risk is very low, however for anuric patients or those on hemodialysis, contrast-enhanced MRI should be performed as close in time to hemodialysis as possible.[117, 118]

Suggested Protocol

- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF, GLS, atrial volumes and pericardial effusions.
- Native T1 mapping in SAx and four-chamber (4Ch) view (consider native T2 mapping [preferred] or T2w STIR imaging for detection of myocardial edema.)
- T1w EGE images
- TI scout, with attention paid to abnormal myocardial nulling
- T1w LGE for detection of fibrosis/scar, ideally with both magnitude and PSIR reconstructions
- Post-contrast T1 mapping for calculation of ECV.

Pericardial Disease

Acute pericarditis can be seen as a consequence of cancer treatment from certain anti-neoplastic drugs and radiation therapy (RT).[119-122] There are several antineoplastic drugs that have been associated with acute pericarditis, such as busulfan, bleomycin, cytarabine, anthracyclines, cyclophosphamide, some tyrosine kinase inhibitors, and ICIs.[119] Radiation

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therapy to the chest and mediastinum can cause pericarditis, predominantly seen from 5 years to more than 10 years following treatment and can be associated with constriction.[120, 121] Acute presentations of pericarditis during RT are uncommon and usually are related to malignancies invading the pericardium that receive high doses.[122] Imaging with CMR can assess for pericardial diseases in these patients[7, 123] by looking for a pericardial effusion on cine imaging, and for pericardial thickening on both SSFP cine imaging and on T1 weighted (T1w) turbo spin echo (TSE) imaging (with fat suppression). These enable localization of the pericardial thickening, and measurement (with ≥3mm considered abnormal on TSE sequences).[123] T2w imaging can assess for pericardial inflammation and pericardial enhancement with LGE to assess for inflammation and/or fibrosis.[124] For pericardial constriction, septal motion should be assessed, with ventricular interdependence diagnosed with septal shift towards the left ventricle on inspiration during real time cine imaging. Pericardial adhesions can be evaluated with tagging sequences.[124] Simultaneous biventricular real-time free-breathing CMR flow quantification has been shown to be helpful to confirm the diagnosis of constrictive physiology.[125]

Suggested Protocol:

- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF, as well as pericardial effusions.
- T1w-turbo spin echo (TSE) sequences with and without fat suppression to assess pericardial thickness.
- Native T2 mapping for detection of myocardial edema
- T2w STIR imaging for detection of pericardial edema.
- Tagging sequences for detection of tethering or adhesions.

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- Real-time cine with deep inspiratory maneuver for ventricular interdependence (mid SAx view, optional 4-chamber view).
- T1w LGE, ideally with both magnitude and PSIR reconstructions, for detection of pericardial inflammation as well as myocardial fibrosis/scar.

Valvular heart disease

Radiation therapy (RT) to the chest, with or without cancer therapy, increases the risk of valvular heart disease in cancer survivors, albeit often with a long latency period (~20 years).[2, 126-130] Left-sided valves are affected with higher frequency than right-sided valves.[129, 131, 132] Valvular thickening, fibrosis, calcification, and degeneration can lead first to regurgitation and then stenosis, which may be progressive.[126, 133, 134] Aorto-mitral curtain thickening, described by echocardiography, is classic.[135]

Echocardiography is often considered first-line for the diagnosis and surveillance of cancer-therapy related valvulopathy.[136] CMR can be considered as a complementary technique to echocardiography when assessing valvular lesions. CMR can quantify flow directly using phase contrast velocity mapping.[137] In very stenotic valves, the peak jet velocity may be underestimated, but CMR allows direct planimetry of the valve orifice area, which may be a useful technique for quantifying stenosis severity.[137] CMR now has an American College of Cardiology (ACC)/American Heart Association (AHA) Class I indication for assessing the severity of mitral and aortic regurgitation or aortic stenosis, especially when echocardiographic assessments are suboptimal[138], and is particularly useful in assessing right-sided valves, especially the pulmonary valve.[137] CMR has the added advantage of simultaneously assessing the effect on the ventricles with accurate measurements of cardiac structure, function, and tissue characterisation in a single comprehensive examination. Four-dimensional flow imaging (4D

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Flow CMR) permits motion encoding in all three spatial directions alongside the dimension of time and therefore provides additional detail on flow and hemodynamics. Advanced quantification parameters are currently still largely confined to the research arena, and 4D flow acquisition and analysis should follow SCMR expert consensus statement recommendations.[139]

Suggested Protocol

- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF, as well as pericardial effusions.
- Phase contrast velocity mapping through the ascending aorta and main pulmonary artery (PA).
- Optional: Consider dedicated cines to evaluate the valve of interest, e.g. short-axis stack through the aortic valve.
- Optional: Consider through-plane phase contrast of the aortic valve in the setting of aortic stenosis for peak velocity estimation and 2D planimetry.
- T1w LGE for detection of fibrosis/scar if cardiac dysfunction is present.
- Optional: Consider T1 mapping and ECV assessment, particularly in patients with aortic stenosis.

Pulmonary Hypertension

Patients with cancer are at increased risk for pulmonary hypertension (PH), which has been reported to be as high as 15% in cross sectional series.[140] Common cancer therapies have been associated with pulmonary arterial hypertension, including multitargeted kinase inhibitors targeting Breakpoint Cluster Region-Abelson Murine Leukemia Viral Oncogene Homolog 1 (BCR-ABL),, proteasome inhibitors, interferon alpha and alkylating agents.[141] Other factors

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can contribute to PH in this patient population, such as compressive effects of cancer and hypercoagulability with resultant pulmonary embolism or chronic thromboembolic pulmonary hypertension.

Consensus guidelines emphasize the importance of monitoring patients with cancer known to be at increased risk for PH.[80] Right heart structure and function parameters are important for determining prognosis but can be difficult to evaluate accurately by echocardiography given the complex geometry of the RV.[142] Imaging with CMR allows for accurate and reproducible assessment of RV size, morphology and function, as well as flow physiology, and pulmonary arterial (PA) distensibility.[143-145] Findings of LGE, reduced PA distensibility, and retrograde pulmonary artery flow are highly predictive for PH.[146-148] MRA is an alternative method for evaluating pulmonary vasculature that avoids exposure to ionizing radiation, especially in patients for whom CT is contraindicated. Marked RV dilation (end diastolic volume index ≥84 mL/m2), low RV stroke volume (stroke volume index ≤25 ml/m2), decrease in RV transverse shortening and fractional area change by CMR, and reduced LV volume (LV end diastolic volume index ≤40 mL/m2) have each been shown to predict mortality and treatment failure in patients with PH.[149-151]

Suggested Protocol:

- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF, as well as pericardial effusions.
- Optional: Consider native T1 mapping
- Phase contrast velocity mapping at the ascending aorta and main PA for systemic and pulmonic flow (or 4D flow if available), with calculation of pulmonary to systemic flow ratio (Qp:Qs).

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- Three-dimensional (3D) contrast enhanced or non−contrast MRA targeting the pulmonary vasculature.
- T1w LGE, ideally with both magnitude and PSIR reconstructions, for assessment of RV insertion point fibrosis.
- Optional: Consider post-contrast T1 mapping for ECV calculation.

Cardiovascular Complications from Specific Cancer Treatments for Special Consideration: Radiation Therapy and Hematopoietic Stem Cell Transplantation

Radiation therapy (RT) and hematopoietic stem cell transplantation (HSCT) are both associated with cardiovascular complications acutely during therapy, and late effects may arise many years after cancer treatment. Both of these types of cancer therapy can affect a variety of cardiovascular structures (often in combination), meaning that the ability of CMR to interrogate multiple pathologies within a single non-invasive test gives it great utility in this setting.

When exposed to RT, almost all cardiovascular structures can be affected, with accelerated coronary artery disease being one of the more notable toxicities.[152] Effects are dose-dependent: every 1 Gray (Gy) increase in the mean heart dose is associated with a 7.4% increased rate of major coronary events.[152] Stress CMR myocardial perfusion imaging has additional value particularly for symptomatic patients where coronary disease is suspected, but has also been shown to accurately identify high risk patients with coronary artery disease (CAD) in the general population[153] and is as valuable as invasive fractional flow reserve (FFR) as a prognostic marker.[154]

Radiation therapy has also been linked to small vessel endothelial dysfunction,[155] alongside non-vascular effects including heart failure, valvular heart disease, pericarditis, and

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pericardial constriction.[155, 156] For patients with technically difficult echocardiographic studies, CMR can assess for radiation-associated valvular heart disease[157] and pericarditis.[158] Similarly, fibrotic changes to the myocardium may result in restrictive cardiomyopathy and in childhood cancer survivors, T1 mapping and extracellular volume (ECV) values (representing interstitial fibrosis/myocyte atrophy) appear to correlate with exercise capacity in a cohort of anthracycline treated patients, of whom 17% received chest radiation.[159] Left atrial (LA) fibrosis as quantified by LA scar volume using LGE has also been proposed as a marker of myocardial injury due to RT[160]; however, its practical application may be challenging.

HSCT is part of the treatment for many hematological malignancies including certain lymphomas, leukemias, and myeloma.[161] However, HSCT is associated with both short and long term cardiovascular complications, either from direct toxicity of the conditioning chemotherapy or radiation regimen, the cardiometabolic sequelae of immunosuppression, or from graft-versus-host effects.[161-164] Complications including heart failure, myocarditis, supraventricular and ventricular arrhythmias[163, 165], pericarditis, and pericardial effusions have been reported early during treatment, with late effects including coronary artery disease, heart failure and pericardial constriction.[162-164] CMR can assess for multiple pathologies within a single study, but can also exclude other etiologies of heart failure such as myocarditis from opportunistic and atypical infections[57, 166-168] in the setting of immunosuppression with HSCT, amyloidosis in patients with underlying multiple myeloma[111, 115, 169], or iron overload following frequent blood transfusions.[170] Therefore, CMR can provide a very comprehensive imaging assessment for symptomatic (e.g., heart failure, chest pain) patients during transplantation, as well as post transplantation when investigating for late effects. CMR

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protocols for patients with history of RT or HSCT should be tailored to the suspected clinical question, with specific protocols included in the above sections (e.g., valvular disease, coronary artery disease, pericardial disease, myocardial disease).

Pediatric Cancers

CV disease is the second leading cause of morbidity and mortality (after recurrent malignancy) in long–term survivors of childhood cancer.[89, 171-174] Most pediatric cancer protocols continue to rely on cytotoxic therapies, including anthracyclines and RT, although biological and molecular targeted therapies are increasingly being used.[175-179] Chemotherapy regimens are typically high intensity, including increased doses and durations compared to those used in adults. Furthermore, the developing CV system of children and adolescents is particularly vulnerable to the effect of anthracyclines[180] and subclinical changes observed after cancer therapy, coupled with genetic predisposition[174, 181-201] and increased incidence of obesity, diabetes, and hypertension in pediatric cancer survivors, significantly increase the risk of premature CV disease onset.[202-204]

The current CTRCD definition does not take into consideration critical intrinsic differences between children and adults.[205] For children and young adults, reductions in LVEF of more than 10% are clinically significant, even when overall function remains preserved (EF>55%).[136, 205] Echocardiography remains the first-line imaging modality for CTRCD surveillance, however CMR is a second-line or problem-solving tool, used when echo is limited, discordant, or when high diagnostic precision is required for management decisions. Similar to adults, the goal of CMR imaging is to provide early, accurate, and reproducible information regarding global and regional myocardial function and tissue characterization during and after treatment with cardiotoxic chemotherapy in high-risk pediatric patients.[206] The use of robust

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surrogate endpoints for CTRCD assessment is critical for developing effective primary and secondary prevention strategies, including early use of combination heart-failure therapy in this at-risk population.[203] Children's anatomical structures are smaller and heart rates higher, demanding greater spatial and temporal resolution in performing CMR.[207] Also, use of sedation and non-breath-holding imaging strategies also need to be considered for younger and uncooperative patients.[207, 208]

CMR Detection of Early-Onset Cardiotoxicity

CMR surveillance for early-onset cardiotoxicity can be considered for certain high-risk pediatric and young adult patients, such as those on cancer treatment protocols that include cardiotoxic medications associated with RT or HSCT, particularly when anthracyclines are administered.[198, 209-216] [217]

Suggested protocol:

- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF, as well as pericardial effusions.
- (Optional: Strain imaging with feature tracking from bSSFP cines or SENC/DENSE/tagging)
- Native T1 mapping for evaluation of edema or diffuse fibrosis.
- Native T2 mapping (preferred) or T2w STIR imaging for detection of myocardial edema.
- Optional, only in select cases:
    - T1w LGE, ideally with both magnitude and PSIR reconstructions, for detection of fibrosis/scar in patients exposed to RT
    - Post-contrast T1 mapping for calculation of ECV.

Late-Onset Cardiotoxicity

CMR screening of high-risk pediatric and young adult cancer survivors for late-onset cardiotoxicity[174] can be considered at the completion of cardiotoxic chemotherapy, followed

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by CMR at 3-5 year intervals.[206] Adverse cardiac remodeling with myocyte hypertrophy and microscopic fibrosis are associated with the development of heart failure (stage C/D)[203, 218-221] and characterize late onset cardiotoxicity with a five-year survival after onset of clinical heart failure of <50%.[171-174] With respect to RT, although many of the cardiotoxicities, including reduced EF occur >20 years following treatment, evidence of cardiotoxicity as demonstrated by increased ECV values is detected as early as 5-8 years following completion of chemoradiotherapy in patients with esophageal cancer.[222]

Suggested protocol (or imaging sequences to use in evaluation)

- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF, as well as pericardial effusions.
- Strain imaging: (feature tracking from bSSFP cines or SENC/DENSE/tagging)
- Native T1 mapping for evaluation of edema or diffuse fibrosis.
- Native T2 mapping (preferred) or T2w STIR imaging for detection of myocardial edema.
- T1w LGE, ideally with both magnitude and PSIR reconstructions, for detection of fibrosis/scar, especially in patients exposed to RT
- Post-contrast T1 mapping for calculation of ECV.
- Consider use of 4D-Flow CMR for assessment of arterial stiffness.[223]

Cardiac Masses

Cancer confers increased risk for development of cardiac masses – including metastatic neoplasms and thrombi (due to systemic hypercoagulability and/or indwelling central venous catheters). CMR has been shown to differentiate between mass types (normal tissue, thrombus, benign/malignant tumors) with high diagnostic accuracy (98.4%) and is a powerful tool for predicting prognosis, incremental to common clinical risk factors.[224, 225] A detailed review of cardiac masses is outside the scope of this document, but we direct interested readers to previously-published reviews on this topic.[226]

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Imaging with CMR enables multiparametric assessment of cardiac masses and carries a class I indication in the 2020 SCMR Clinical Indications for CMR position paper.[227] CMR aids differentiation between the underlying type of mass using the range of tissue characterization techniques available. It should be emphasized, however, that for masses where there is a high index of suspicion of an underlying neoplastic process, histology should always be obtained for diagnostic confirmation. CMR can also help guide management by determining the location and size of the mass, relationship with adjacent cardiac structures (invasion, vascular or valvular obstruction) and surgical resectability.[228]

Whereas neoplastic masses require vascular supply for tumorigenesis, thrombus is definable based on avascularity. Both resting first pass perfusion and LGE imaging can therefore help differentiate neoplasm from thrombus based on vascular composition[224, 229-232] Complementing LGE-CMR tissue characterization, cine-CMR enables assessment of functional consequences of masses including valvular obstruction and impaired LV contractility.

Advances in CMR technology may further augment cardiac mass assessment. Quantitative T1 mapping and perfusion CMR can assess the magnitude of contrast enhancement to identify highly vascular neoplasms and thrombus chronicity, T2w imaging can assess edema within lesions, and T1w fat saturated imaging can evaluate presence of fat within a mass. Research is ongoing to test the relative utility of parametric mapping and LGE-CMR for diagnostic and prognostic assessment of cardiac masses.

Suggested Protocol

- bSSFP cine (short and long axis planes) for assessment of cardiac morphology, LV and RV volumes, regional contractility, and EF, as well as pericardial effusions.
- T1w TSE through the mass and surrounding structures

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- T2w TSE with fat suppression (optional: without fat suppression) through the mass and surrounding structures as above
- Resting first pass perfusion with slices through the mass
- Repeat T1w TSE spin echo with fat suppression (early after GBCA)
- Optional:
    - Repeat selected bSSFP cine images post-contrast
    - T1w LGE images with TI set to null thrombus (500-600 ms at 1.5T, 850-900 ms at 3T) if thrombus is suspected

General considerations of CMR in cancer survivors

There are several other considerations that should be taken into account when planning, acquiring and reporting CMR in cancer survivors. Cancer treatment decisions regarding choice of treatment or continuation versus interruption/cessation may be at least partly determined by CMR results, hence delays to scheduling and reporting of scans should be minimized. Patients are commonly frail or unwell due either to their cancer or its treatment and hence adjusting protocols to reduce scan duration where possible will aid patient comfort and maximize the likelihood of scan completion. Recent advances in CMR pulse sequence design and application of machine learning have led to the availability of abbreviated protocols for fast acquisitions, free-breathing acquisitions, and extensive capabilities for automated postprocessing and quantitative analysis, including comparisons with prior studies.[233]

Similarly for patients receiving chemotherapy, gaining venous access may be challenging and hence where contrast can be avoided (for example for serial monitoring for CTRCD in patients receiving anthracyclines or HER2 targeted therapies), non-contrast protocols are preferred. Finally, for serial imaging (e.g., surveillance for cardiotoxicity, determining response

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to treatment with amyloid, myocarditis, tumors, etc.), precise quantification demands standardization between scanner, sequences (e.g., no change in T1 mapping sequence), and measurement (e.g., regions of interest assessed). Reports should reference the comparison with prior scans, which should be re-reviewed during reporting. Additional care should also be taken to review the extra-cardiac anatomy carefully for signs of metastases or other complications (such as pulmonary emboli, thrombi on indwelling central venous catheters, etc.). This may require comparison with other cross-sectional imaging (CT/ PET) and discussion with radiology colleagues.

Inclusion of CMR in Current Guidelines and Appropriate Use Criteria

Exponential growth of different classes of targeted cancer therapeutics in the 20th century has revolutionized treatment approaches and continues to transform oncology practice. Although many new anticancer agents have the potential to interfere with cardiac and vascular homeostasis, there has been a gap in understanding of the mechanisms and impact of CV injury. As a result, the development of criteria for CV assessment has been inconsistent and often lacking from oncology regimens.

Current guideline recommendations are based on evidence from early clinical trials of anthracycline and HER-2 targeted therapies in breast cancer where, for the latter, cardiac imaging surveillance was included in regulatory approvals for the drugs.[234] For the vast majority of other cancer therapies, prospective evidence is lacking, with recommendations extrapolated from trastuzumab experience with significant discrepancies. Further, LVEF assessment in older oncology trials has traditionally been done by either nuclear techniques (MUGA) or echocardiography, thus explaining the limited endorsement of CMR even when cardiac imaging is included in guidelines. Many of these papers also nicely highlight the

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advantages and disadvantages of the different cardiac imaging modalities across different clinical scenarios, especially the ACC Cardio-Oncology and Imaging Leadership Councils' document. [136]

In Table 2, we summarize some of the recent professional society guidelines and scientific statements with reference to CMR. For patients presenting with symptoms concerning for CV complications, most society recommendations include cardiac imaging; the differences among them largely reflect the intended audience (oncology vs CV professionals) and design/nature of the publications. Clinical guidelines, for example, predominantly focus on data from randomized clinical trials to guide indications, and patients with cardiac symptoms or active CV diagnoses tend to be excluded from oncology clinical trials. Position papers and consensus statements provide more flexibility with inclusion of populations with limited data and aim to guide practitioners in situations where evidence is lacking.

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Table 2. Professional Society Recommendations for Cardiac Imaging in Cancer Patients and Survivors

Document	**Inclusion of CMR**
2025 ACR Appropriateness Criteria Assessment of Cardiac Function and Baseline Cardiac Risk Stratification in Oncology Patients	• CMR for cardiac risk stratification prior to initiation of oncologic therapy without symptoms • For assessment of cardiac function during oncologic therapy with cardiac symptoms
2025 ESC Guidelines for the management of myocarditis and pericarditis.	(This guideline is focused on all etiologies of myocarditis/pericarditis, but comments specifically on cancer-related and cancer therapy-related cases) • CMR for diagnosis of acute or chronic myocarditis in patients without high risk presentation • For diagnosis of acute pericarditis in unclear cases • For risk stratification in myocarditis or pericarditis • For diagnosis of pericardial constriction • For further characterization of myocarditis in patients with high risk presentation following endomyocardial biopsy • For follow up at 6 months following acute myocarditis

2023 Cardiovascular Imaging in Contemporary Cardio-Oncology: A Scientific Statement from the American Heart Association[10]	• CMR for accurate LVEF or valve assessment before cancer treatment • For unexplained heart failure • For recent myopericarditis • For cardiac mass assessment • For workup of other cardiac conditions as needed (e.g., amyloid)
2023 Systematic review and updated recommendations for cardiomyopathy surveillance for survivors of childhood, adolescent, and young adult cancer from the International Late Effects of Childhood Cancer Guideline Harmonization Group	• CMR might be reasonable for cardiomyopathy surveillance in at-risk survivors of CAYA cancer for whom echocardiography is not technically feasible or optimal • At- risk survivors defined as high dose (≥250mg/m^2 of anthracyclines, ≥30 Gy of chest-directed radiation therapy, ≥100 mg/m^2 of anthracycline in combination with ≥15 Gy of chest-directed radiation) or moderate dose (≥100 to <250 mg/m^2 of anthracycline or ≥15 to <30 Gy of chest radiation
2022 Advances in Multimodality Imaging in Cardio-Oncology: JACC State-of-the-Art Review on behalf of the ACC Cardio-Oncology and ACC Imaging Councils[136]	• CMR for baseline and surveillance with low normal or decreased LVEF on echocardiogram or discrepant results on serial echocardiogram • For workup of symptomatic heart failure, suspected cardiac amyloidosis, or Takotsubo cardiomyopathy • For suspected ICI myocarditis • For workup of cardiac masses
2022 ESC Guidelines on Cardio-Oncology[228]	• CMR for baseline and surveillance if echocardiogram is unavailable or non-diagnostic • For further workup of abnormalities on echocardiogram (e.g., hypertrophic cardiomyopathy)
2020 ESMO Consensus Recommendations on management of Cardiac Disease in Cancer Patients Throughout Oncological Treatment[3]	• CMR (or quantitative 2D/3D echocardiography) recommended for LVEF assessment
2017 ASCO Clinical Guideline on Prevention and Monitoring of Cardiac Dysfunction in Survivors of Adult Cancers[5]	• CMR recommended for surveillance and monitoring of LVEF if echocardiography is not available or poor quality
2016 ESC Position Paper on cancer treatments and cardiovascular toxicity[80]	• As confirmatory test in diagnosis of LV dysfunction or borderline LVEF • In assessment of etiology and prognosis of LV dysfunction (tissue characterization and parametric imaging) • Pericardial disease
2014 ASE/EACVI Expert Consensus for Multimodality Imaging Evaluation of Adult Patients during and after Cancer Therapy[4]	• Recommended as confirmatory tests when LV dysfunction is identified or in patients with inadequate echocardiographic windows • Additive tool in assessment of cardiomyopathy (tissue characterization) valvular and pericardial disease

2D, two-dimensional; 3D, three-dimensional; ACC, American College of Cardiology; ACR, American College of Radiology; ASCO, American Society of Clinical Oncology; ASE,

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American Society of Echocardiography; CAYA, childhood, adolescent, and young adult; CMR, cardiovascular magnetic resonance; EACVI, European Association for Cardiovascular Imaging; ESC, European Society of Cardiology; ESMO, European Society of Medical Oncology; Gy, Gray; ICI, immune checkpoint inhibitor; JACC, Journal of the American College of Cardiology; LV, left ventricular; LVEF, left ventricular ejection fraction.

All statements agree with basing the choice of cardiac imaging modality on local availability and expertise. Once chosen, the same modality should be used with repeated testing, such as surveillance imaging. CV practitioners and imagers are likely to be more familiar with inherent differences between imaging modalities but are likely to see only a fraction of cancer survivors. We therefore recommend collaboration with local oncology teams as well as national oncology societies as a critical step in implementing optimal cardiac imaging techniques for diagnosis and follow-up of patients on specific cancer therapies. This strategy will also facilitate the translation of future practice standards and further development of imaging protocols focused on specific and different CV effects of cancer therapies.

Future Developments and Research Considerations

The increased recognition of the cardiovascular complications of cancer treatments, along with better cancer outcomes and rapid drug development, provide unique opportunities for the use of CMR in cancer survivors. Despite numerous advances, multiple knowledge gaps remain. Ongoing research needs include further defining CMR phenotypes and inclusion of CMR into standard definitions of CV toxicity of cancer therapeutics. For example, imaging findings of cardiac injury related to anthracyclines and HER2-targeted therapies are well characterized[235], but identification of CMR predictors of cardiotoxicity needs further study. In recent years, the application and investigation of CMR to aid the diagnosis of myocarditis as part of immune-related adverse events (IRAEs) in patients treated with ICIs has been growing [63, 67]. However,

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heterogeneity and limitations exist in current data, and the field would benefit from further study, especially as this diagnosis can have implications for possible contraindication to ICI in the future if myocarditis is considered a severe adverse event.[236]

Also needed is the prospective validation of CMR parameters as CV risk predictors cancer survivors. While a significant discourse in the literature has focused on the advantages of different techniques (superior reproducibility, higher accuracy in cardiac function quantification), much larger is the need to transform the overall approach to cardiac imaging in oncology. The traditional single focus, of determining a "safe LVEF value" to proceed with oncology administration, will not be able to improve long term overall outcomes regardless of which technique is used.[235] Current cut-offs for standardized thresholds and normative values for CMR parametric mapping and strain are largely extrapolated from non-oncology cohorts, but further study is needed to establish these values specifically in cardio-oncology populations.[23, 41] Ideally T1 mapping values would be known at baseline, as they can lack specificity for certain disease states; using the same scanner when possible will improve reliability of results.[43]

Additionally, there needs to be inclusion of CMR into clinical trials investigating cardiovascular prevention and treatment strategies in patients with cancer. Small randomized clinical trials investigating primary cardioprevention strategies in.[237-239] breast cancer survivors have demonstrated feasibility and advantages of CMR. In particular, higher accuracy of LVEF assessment has allowed smaller sample sizes compared to studies using LVEF as measured by echocardiography as a primary outcome. These studies have pointed to the common challenges of cardio-oncology trial design, such as lack of high-risk population, low event rate, and the limitations of the intervention itself.[240] Inclusion of under-represented and higher risk

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groups in these trials is also necessary to ensure equity in the field.[241] Innovative treatment strategies, coupled with novel CMR parameters tailored to the individual cancer treatment risk represent research priorities in this category.

Overall, the gap in education and training of CV specialists in cardio-oncology has been recognized, and a growing number of resources have become available.[242] Over the years, the number of publications, dedicated courses, and conferences in the field has significantly increased, in addition to the inclusion of cardio-oncology content in many local, national and international meetings. SCMR annual Scientific Sessions, for example, have consistently included cardio-oncology topics over the last years[243], including joint sessions with the International Cardio-Oncology Society, as well as developed webinars focused on CMR use in this patient population. At the same time, inclusion of CV topics in oncology meetings has created an opportunity to present advances in CMR imaging to the oncology audience.[244] These educational initiatives represent very early steps towards creation of integrated, patient-centered, cardio-oncology care models. The collaboration will need to occur across multiple layers, from coordinated activities between oncology and cardiology health care teams, to aligned professional society engagement and collaborative development of clinical standards. Future growth is needed in dedicated cardio-oncology training with focused instruction on cardiac imaging Importantly, training in advanced cardiac imaging should include special considerations in cancer survivors, as highlighted in the recent Advanced Training Statement on Advanced Cardiovascular Imaging.[245]

Finally, parallel to cardio-oncology, the field of CMR has witnessed major advancements in the last two decades[243], from growth in image quality and data acquisition to development of novel imaging techniques. Improving access to CMR, particularly in low to middle income

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countries, remains an ongoing challenge. Artificial intelligence in CMR has also advanced, potentially resulting in reduced scan time, ability to perform in-line processing, and reduced variability in image acquisition and quality.[246] This can potentially translate to improved access to CMR and improved consistency in image acquisition in patients requiring serial imaging. The ability of CMR to characterize key pathophysiologic processes such as inflammation, edema, fibrosis, and ischemia makes the potential for the use of CMR in cardio-oncology enormous. Coordinated research efforts, described in the steps above, will lead to successful application of CMR technology developments, answer key cardio-oncology questions and improve patient care.

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References

[1] A. Seraphim *et al.*, "Advanced Imaging Modalities to Monitor for Cardiotoxicity," *Curr Treat Options Oncol*, vol. 20, no. 9, p. 73, Aug 8 2019, doi: 10.1007/s11864-019-0672-z.

[2] J. H. Jordan, R. M. Todd, S. Vasu, and W. G. Hundley, "Cardiovascular Magnetic Resonance in the Oncology Patient," *JACC Cardiovasc Imaging*, vol. 11, no. 8, pp. 1150–1172, Aug 2018, doi: 10.1016/j.jcmg.2018.06.004.

[3] G. Curigliano *et al.*, "Management of cardiac disease in cancer patients throughout oncological treatment: ESMO consensus recommendations," *Ann Oncol*, vol. 31, no. 2, pp. 171–190, Feb 2020, doi: 10.1016/j.annonc.2019.10.023.

[4] J. C. Plana *et al.*, "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," *Eur Heart J Cardiovasc Imaging*, vol. 15, no. 10, pp. 1063–93, Oct 2014, doi: 10.1093/ehjci/jeu192.

[5] S. H. Armenian *et al.*, "Prevention and Monitoring of Cardiac Dysfunction in Survivors of Adult Cancers: American Society of Clinical Oncology Clinical Practice Guideline," *J Clin Oncol*, vol. 35, no. 8, pp. 893–911, Mar 10 2017, doi: 10.1200/JCO.2016.70.5400.

[6] M. Aapro *et al.*, "Anthracycline cardiotoxicity in the elderly cancer patient: a SIOG expert position paper," *Ann Oncol*, vol. 22, no. 2, pp. 257–67, Feb 2011, doi: 10.1093/annonc/mdq609.

[7] J. C. Plana, P. Thavendiranathan, C. Bucciarelli-Ducci, and P. Lancellotti, "Multi-Modality Imaging in the Assessment of Cardiovascular Toxicity in the Cancer Patient," *JACC Cardiovasc Imaging*, vol. 11, no. 8, pp. 1173–1186, Aug 2018, doi: 10.1016/j.jcmg.2018.06.003.

[8] E. H. Romond *et al.*, "Trastuzumab plus adjuvant chemotherapy for operable HER2-positive breast cancer," *N Engl J Med*, vol. 353, no. 16, pp. 1673–84, Oct 20 2005, doi: 10.1056/NEJMoa052122.

[9] G. Ezaz, J. B. Long, C. P. Gross, and J. Chen, "Risk prediction model for heart failure and cardiomyopathy after adjuvant trastuzumab therapy for breast cancer," *J Am Heart Assoc*, vol. 3, no. 1, p. e000472, Feb 28 2014, doi: 10.1161/JAHA.113.000472.

[10] D. Addison *et al.*, "Cardiovascular Imaging in Contemporary Cardio-Oncology: A Scientific Statement From the American Heart Association," *Circulation*, vol. 148, no. 16, pp. 1271–1286, Oct 17 2023, doi: 10.1161/CIR.0000000000001174.

[11] S. E. Petersen *et al.*, "Reference ranges for cardiac structure and function using cardiovascular magnetic resonance (CMR) in Caucasians from the UK Biobank population cohort," *J Cardiovasc Magn Reson*, vol. 19, no. 1, p. 18, Feb 3 2017, doi: 10.1186/s12968-017-0327-9.

[12] N. G. Bellenger *et al.*, "Comparison of left ventricular ejection fraction and volumes in heart failure by echocardiography, radionuclide ventriculography and cardiovascular magnetic resonance; are they interchangeable?," *Eur Heart J*, vol. 21, no. 16, pp. 1387–96, Aug 2000, doi: 10.1053/euhj.2000.2011.

[13] G. T. Armstrong *et al.*, "Screening adult survivors of childhood cancer for cardiomyopathy: comparison of echocardiography and cardiac magnetic resonance imaging," *J Clin Oncol*, vol. 30, no. 23, pp. 2876–84, Aug 10 2012, doi: 10.1200/JCO.2011.40.3584.

[14] P. Thavendiranathan, A. D. Grant, T. Negishi, J. C. Plana, Z. B. Popovic, and T. H. Marwick, "Reproducibility of echocardiographic techniques for sequential assessment of

Journal Pre-proof

left ventricular ejection fraction and volumes: application to patients undergoing cancer chemotherapy," *J Am Coll Cardiol,* vol. 61, no. 1, pp. 77–84, Jan 8 2013, doi: 10.1016/j.jacc.2012.09.035.

[15] T. H. Marwick *et al.*, "Strain surveillance during chemotherapy to improve cardiovascular outcomes: the SUCCOUR-MRI trial," *Eur Heart J,* vol. 45, no. 41, pp. 4414–4424, Nov 7 2024, doi: 10.1093/eurheartj/ehae574.

[16] J. Schulz-Menger *et al.*, "Standardized image interpretation and post-processing in cardiovascular magnetic resonance - 2020 update : Society for Cardiovascular Magnetic Resonance (SCMR): Board of Trustees Task Force on Standardized Post-Processing," *J Cardiovasc Magn Reson,* vol. 22, no. 1, p. 19, Mar 12 2020, doi: 10.1186/s12968-020-00610-6.

[17] G. C. Melendez *et al.*, "Frequency of Left Ventricular End-Diastolic Volume-Mediated Declines in Ejection Fraction in Patients Receiving Potentially Cardiotoxic Cancer Treatment," *Am J Cardiol,* vol. 119, no. 10, pp. 1637–1642, May 15 2017, doi: 10.1016/j.amjcard.2017.02.008.

[18] J. H. Jordan, B. Sukpraphrute, G. C. Melendez, M. P. Jolly, R. B. D'Agostino, Jr., and W. G. Hundley, "Early Myocardial Strain Changes During Potentially Cardiotoxic Chemotherapy May Occur as a Result of Reductions in Left Ventricular End-Diastolic Volume: The Need to Interpret Left Ventricular Strain With Volumes," *Circulation,* vol. 135, no. 25, pp. 2575–2577, Jun 20 2017, doi: 10.1161/CIRCULATIONAHA.117.027930.

[19] J. H. Jordan *et al.*, "Left Ventricular Mass Change After Anthracycline Chemotherapy," *Circulation. Heart failure,* vol. 11, no. 7, p. e004560, Jul 2018, doi: 10.1161/CIRCHEARTFAILURE.117.004560.

[20] T. G. Neilan *et al.*, "Left ventricular mass in patients with a cardiomyopathy after treatment with anthracyclines," *Am J Cardiol,* vol. 110, no. 11, pp. 1679–86, Dec 1 2012, doi: 10.1016/j.amjcard.2012.07.040.

[21] E. K. Oikonomou *et al.*, "Assessment of Prognostic Value of Left Ventricular Global Longitudinal Strain for Early Prediction of Chemotherapy-Induced Cardiotoxicity: A Systematic Review and Meta-analysis," *JAMA Cardiol,* vol. 4, no. 10, pp. 1007–1018, Oct 1 2019, doi: 10.1001/jamacardio.2019.2952.

[22] V. O. Puntmann *et al.*, "Society for Cardiovascular Magnetic Resonance (SCMR) expert consensus for CMR imaging endpoints in clinical research: part I - analytical validation and clinical qualification," *J Cardiovasc Magn Reson,* vol. 20, no. 1, p. 67, Sep 20 2018, doi: 10.1186/s12968-018-0484-5.

[23] S. Giusca *et al.*, "Multiparametric Early Detection and Prediction of Cardiotoxicity Using Myocardial Strain, T1 and T2 Mapping, and Biochemical Markers: A Longitudinal Cardiac Resonance Imaging Study During 2 Years of Follow-Up," *Circ Cardiovasc Imaging,* vol. 14, no. 6, p. e012459, Jun 2021, doi: 10.1161/CIRCIMAGING.121.012459.

[24] C. P. Houbois *et al.*, "Serial Cardiovascular Magnetic Resonance Strain Measurements to Identify Cardiotoxicity in Breast Cancer: Comparison With Echocardiography," *JACC Cardiovasc Imaging,* Nov 25 2020, doi: 10.1016/j.jcmg.2020.09.039.

[25] B. C. Drafts *et al.*, "Low to moderate dose anthracycline-based chemotherapy is associated with early noninvasive imaging evidence of subclinical cardiovascular

Journal Pre-proof

disease," *JACC Cardiovasc Imaging,* vol. 6, no. 8, pp. 877–85, Aug 2013, doi: 10.1016/j.jcmg.2012.11.017.
[26] S. Nakano *et al.*, "Cardiac magnetic resonance imaging-based myocardial strain study for evaluation of cardiotoxicity in breast cancer patients treated with trastuzumab: A pilot study to evaluate the feasibility of the method," *Cardiol J,* vol. 23, no. 3, pp. 270–80, 2016, doi: 10.5603/CJ.a2016.0023.
[27] G. Ong *et al.*, "Myocardial strain imaging by cardiac magnetic resonance for detection of subclinical myocardial dysfunction in breast cancer patients receiving trastuzumab and chemotherapy," *Int J Cardiol,* vol. 261, pp. 228–233, Jun 15 2018, doi: 10.1016/j.ijcard.2018.03.041.
[28] M. P. Jolly, J. H. Jordan, G. C. Melendez, G. R. McNeal, R. B. D'Agostino, Jr., and W. G. Hundley, "Automated assessments of circumferential strain from cine CMR correlate with LVEF declines in cancer patients early after receipt of cardio-toxic chemotherapy," *J Cardiovasc Magn Reson,* vol. 19, no. 1, p. 59, Aug 2 2017, doi: 10.1186/s12968-017-0373-3.
[29] I. Y. Gong *et al.*, "Early diastolic strain rate measurements by cardiac MRI in breast cancer patients treated with trastuzumab: a longitudinal study," *Int J Cardiovasc Imaging,* vol. 35, no. 4, pp. 653–662, Apr 2019, doi: 10.1007/s10554-018-1482-2.
[30] M. A. Lunning *et al.*, "Cardiac magnetic resonance imaging for the assessment of the myocadium following doxorubicin-based chemotherapy," *Am J Clin Onc,* 2013.
[31] C. K. Suerken *et al.*, "Simultaneous Left Ventricular Volume and Strain Changes During Chemotherapy Associate With 2-Year Postchemotherapy Measures of Left Ventricular Ejection Fraction," *J Am Heart Assoc,* vol. 9, no. 2, p. e015400, Jan 21 2020, doi: 10.1161/JAHA.119.015400.
[32] S. J. Backhaus *et al.*, "Defining the optimal temporal and spatial resolution for cardiovascular magnetic resonance imaging feature tracking," *J Cardiovasc Magn Reson,* vol. 23, no. 1, p. 60, May 17 2021, doi: 10.1186/s12968-021-00740-5.
[33] M. K. Burrage and V. M. Ferreira, "The use of cardiovascular magnetic resonance as an early non-invasive biomarker for cardiotoxicity in cardio-oncology," *Cardiovasc Diagn Ther,* vol. 10, no. 3, pp. 610–624, Jun 2020, doi: 10.21037/cdt-20-165.
[34] C. M. Kramer, J. Barkhausen, C. Bucciarelli-Ducci, S. D. Flamm, R. J. Kim, and E. Nagel, "Standardized cardiovascular magnetic resonance imaging (CMR) protocols: 2020 update," *J Cardiovasc Magn Reson,* vol. 22, no. 1, p. 17, Feb 24 2020, doi: 10.1186/s12968-020-00607-1.
[35] J. H. Jordan *et al.*, "Anthracycline-Associated T1 Mapping Characteristics Are Elevated Independent of the Presence of Cardiovascular Comorbidities in Cancer Survivors," *Circ Cardiovasc Imaging,* vol. 9, no. 8, Aug 2016, doi: 10.1161/CIRCIMAGING.115.004325.
[36] G. C. Melendez, J. H. Jordan, R. B. D'Agostino, Jr., S. Vasu, C. A. Hamilton, and W. G. Hundley, "Progressive 3-Month Increase in LV Myocardial ECV After Anthracycline-Based Chemotherapy," *JACC Cardiovasc Imaging,* vol. 10, no. 6, pp. 708–709, Jun 2017, doi: 10.1016/j.jcmg.2016.06.006.
[37] J. D. Haslbauer *et al.*, "CMR imaging biosignature of cardiac involvement due to cancer-related treatment by T1 and T2 mapping," *Int J Cardiol,* vol. 275, pp. 179–186, Jan 15 2019, doi: 10.1016/j.ijcard.2018.10.023.

Journal Pre-proof

[38] M. B. Lustberg *et al.*, "Early Detection of Anthracycline-Induced Cardiotoxicity in Breast Cancer Survivors With T2 Cardiac Magnetic Resonance," *Circ Cardiovasc Imaging,* vol. 12, no. 5, p. e008777, May 2019, doi: 10.1161/CIRCIMAGING.118.008777.

[39] C. Galan-Arriola *et al.*, "Serial Magnetic Resonance Imaging to Identify Early Stages of Anthracycline-Induced Cardiotoxicity," *J Am Coll Cardiol,* vol. 73, no. 7, pp. 779–791, Feb 26 2019, doi: 10.1016/j.jacc.2018.11.046.

[40] F. Muehlberg *et al.*, "Native myocardial T1 time can predict development of subsequent anthracycline-induced cardiomyopathy," *ESC Heart Fail,* vol. 5, no. 4, pp. 620–629, Aug 2018, doi: 10.1002/ehf2.12277.

[41] M. A. Altaha *et al.*, "Can Quantitative CMR Tissue Characterization Adequately Identify Cardiotoxicity During Chemotherapy?: Impact of Temporal and Observer Variability," *JACC Cardiovasc Imaging,* vol. 13, no. 4, pp. 951–962, Apr 2020, doi: 10.1016/j.jcmg.2019.10.016.

[42] V. Maestrini *et al.*, "Late Anthracycline-Related Cardiotoxicity in Low-Risk Breast Cancer Patients," *J Am Coll Cardiol,* vol. 69, no. 20, pp. 2573–2575, May 23 2017, doi: 10.1016/j.jacc.2017.03.560.

[43] D. R. Messroghli *et al.*, "Clinical recommendations for cardiovascular magnetic resonance mapping of T1, T2, T2* and extracellular volume: A consensus statement by the Society for Cardiovascular Magnetic Resonance (SCMR) endorsed by the European Association for Cardiovascular Imaging (EACVI)," *J Cardiovasc Magn Reson,* vol. 19, no. 1, p. 75, Oct 9 2017, doi: 10.1186/s12968-017-0389-8.

[44] A. Arbune, A. Soufer, and L. A. Baldassarre, "Clinical and Advanced MRI Techniques for Detection of Checkpoint Inhibitor Associated Myocarditis," *Curr Cardiovasc Imaging Rep,* vol. 13, no. 1, p. 3, 2020/01/14 2020, doi: 10.1007/s12410-019-9521-5.

[45] S. Ganatra and T. G. Neilan, "Immune Checkpoint Inhibitor-Associated Myocarditis," *Oncologist,* vol. 23, no. 8, pp. 879–886, Aug 2018, doi: 10.1634/theoncologist.2018-0130.

[46] N. Palaskas, J. Lopez-Mattei, J. B. Durand, C. Iliescu, and A. Deswal, "Immune Checkpoint Inhibitor Myocarditis: Pathophysiological Characteristics, Diagnosis, and Treatment," *J Am Heart Assoc,* vol. 9, no. 2, p. e013757, Jan 21 2020, doi: 10.1161/JAHA.119.013757.

[47] L. Zhang *et al.*, "Late Gadolinium Enhancement in Patients With Myocarditis from Immune Checkpoint Inhibitors," *J Am Coll Cardiol,* vol. 73, no. 9_Supplement_1, pp. 675–675, 2019, doi: doi:10.1016/S0735-1097(19)31283-5.

[48] J. U. Doherty *et al.*, "ACC/AATS/AHA/ASE/ASNC/HRS/SCAI/SCCT/SCMR/STS 2019 Appropriate Use Criteria for Multimodality Imaging in the Assessment of Cardiac Structure and Function in Nonvalvular Heart Disease: A Report of the American College of Cardiology Appropriate Use Criteria Task Force, American Association for Thoracic Surgery, American Heart Association, American Society of Echocardiography, American Society of Nuclear Cardiology, Heart Rhythm Society, Society for Cardiovascular Angiography and Interventions, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance, and the Society of Thoracic Surgeons," *J Am Coll Cardiol,* vol. 73, no. 4, pp. 488–516, Feb 5 2019, doi: 10.1016/j.jacc.2018.10.038.

Journal Pre-proof

[49] M. Garbi *et al.*, "EACVI appropriateness criteria for the use of cardiovascular imaging in heart failure derived from European National Imaging Societies voting," *Eur Heart J Cardiovasc Imaging*, vol. 17, no. 7, pp. 711–21, Jul 2016, doi: 10.1093/ehjci/jew081.
[50] J. Schulz-Menger *et al.*, "2025 ESC Guidelines for the management of myocarditis and pericarditis," *Eur Heart J*, vol. 46, no. 40, pp. 3952–4041, Oct 22 2025, doi: 10.1093/eurheartj/ehaf192.
[51] B. J. Schneider *et al.*, "Management of Immune-Related Adverse Events in Patients Treated With Immune Checkpoint Inhibitor Therapy: ASCO Guideline Update," *J Clin Oncol*, vol. 39, no. 36, pp. 4073–4126, Dec 20 2021, doi: 10.1200/JCO.21.01440.
[52] M. P. Bonaca *et al.*, "Myocarditis in the Setting of Cancer Therapeutics: Proposed Case Definitions for Emerging Clinical Syndromes in Cardio-Oncology," *Circulation*, vol. 140, no. 2, pp. 80–91, Jul 2 2019, doi: 10.1161/CIRCULATIONAHA.118.034497.
[53] J. Celutkiene *et al.*, "Role of cardiovascular imaging in cancer patients receiving cardiotoxic therapies: a position statement on behalf of the Heart Failure Association (HFA), the European Association of Cardiovascular Imaging (EACVI) and the Cardio-Oncology Council of the European Society of Cardiology (ESC)," *Eur J Heart Fail*, vol. 22, no. 9, pp. 1504–1524, Sep 2020, doi: 10.1002/ejhf.1957.
[54] I. Harries *et al.*, "Magnetic Resonance Imaging to Detect Cardiovascular Effects of Cancer Therapy: JACC CardioOncology State-of-the-Art Review," *JACC CardioOncol*, vol. 2, no. 2, pp. 270–292, Jun 2020, doi: 10.1016/j.jaccao.2020.04.011.
[55] C. E. D. Saunderson, S. Plein, and C. H. Manisty, "Role of cardiovascular magnetic resonance imaging in cardio-oncology," *Eur Heart J Cardiovasc Imaging*, vol. 22, no. 4, pp. 383–396, Mar 22 2021, doi: 10.1093/ehjci/jeaa345.
[56] M. G. Friedrich *et al.*, "Cardiovascular magnetic resonance in myocarditis: A JACC White Paper," *J Am Coll Cardiol*, vol. 53, no. 17, pp. 1475–87, Apr 28 2009, doi: 10.1016/j.jacc.2009.02.007.
[57] V. M. Ferreira *et al.*, "Cardiovascular Magnetic Resonance in Nonischemic Myocardial Inflammation: Expert Recommendations," (in eng), *J Am Coll Cardiol*, vol. 72, no. 24, pp. 3158–3176, Dec 18 2018, doi: 10.1016/j.jacc.2018.09.072.
[58] G. De Velasco *et al.*, "Comprehensive Meta-analysis of Key Immune-Related Adverse Events from CTLA-4 and PD-1/PD-L1 Inhibitors in Cancer Patients," *Cancer Immunol Res*, vol. 5, no. 4, pp. 312–318, Apr 2017, doi: 10.1158/2326-6066.CIR-16-0237.
[59] V. H. Koelzer *et al.*, "Systemic inflammation in a melanoma patient treated with immune checkpoint inhibitors-an autopsy study," *J Immunother Cancer*, vol. 4, p. 13, 2016, doi: 10.1186/s40425-016-0117-1.
[60] D. J. Palmieri and M. S. Carlino, "Immune Checkpoint Inhibitor Toxicity," *Curr Oncol Rep*, vol. 20, no. 9, p. 72, Jul 31 2018, doi: 10.1007/s11912-018-0718-6.
[61] S. S. Mahmood *et al.*, "Myocarditis in Patients Treated With Immune Checkpoint Inhibitors," *J Am Coll Cardiol*, vol. 71, no. 16, pp. 1755–1764, Apr 24 2018, doi: 10.1016/j.jacc.2018.02.037.
[62] J. E. Salem *et al.*, "Cardiovascular toxicities associated with immune checkpoint inhibitors: an observational, retrospective, pharmacovigilance study," *Lancet Oncol*, vol. 19, no. 12, pp. 1579–1589, Dec 2018, doi: 10.1016/S1470-2045(18)30608-9.
[63] L. Zhang *et al.*, "Cardiovascular magnetic resonance in immune checkpoint inhibitor-associated myocarditis," *Eur Heart J*, vol. 41, no. 18, pp. 1733–1743, May 7 2020, doi: 10.1093/eurheartj/ehaa051.

Journal Pre-proof

[64] J. Herrmann *et al.*, "Defining cardiovascular toxicities of cancer therapies: an International Cardio-Oncology Society (IC-OS) consensus statement," *Eur Heart J*, vol. 43, no. 4, pp. 280–299, Jan 31 2022, doi: 10.1093/eurheartj/ehab674.
[65] C. Grani *et al.*, "Prognostic Value of Cardiac Magnetic Resonance Tissue Characterization in Risk Stratifying Patients With Suspected Myocarditis," *J Am Coll Cardiol*, vol. 70, no. 16, pp. 1964–1976, Oct 17 2017, doi: 10.1016/j.jacc.2017.08.050.
[66] C. Grani *et al.*, "Incremental value of extracellular volume assessment by cardiovascular magnetic resonance imaging in risk stratifying patients with suspected myocarditis," *Int J Cardiovasc Imaging*, vol. 35, no. 6, pp. 1067–1078, Jun 2019, doi: 10.1007/s10554-019-01552-6.
[67] P. Thavendiranathan *et al.*, "Myocardial T1 and T2 Mapping by Magnetic Resonance in Patients With Immune Checkpoint Inhibitor-Associated Myocarditis," *J Am Coll Cardiol*, vol. 77, no. 12, pp. 1503–1516, Mar 30 2021, doi: 10.1016/j.jacc.2021.01.050.
[68] M. Awadalla *et al.*, "Global Longitudinal Strain and Cardiac Events in Patients With Immune Checkpoint Inhibitor-Related Myocarditis," *J Am Coll Cardiol*, vol. 75, no. 5, pp. 467–478, Feb 11 2020, doi: 10.1016/j.jacc.2019.11.049.
[69] A. Y. Higgins *et al.*, "Left ventricular myocardial strain and tissue characterization by cardiac magnetic resonance imaging in immune checkpoint inhibitor associated cardiotoxicity," *PLoS One*, vol. 16, no. 2, p. e0246764, 2021, doi: 10.1371/journal.pone.0246764.
[70] National Cancer Institute. "The Surveillance, Epidemiology, and End Results (SEER) Program: Age and Cancer." https://www.cancer.gov/about-cancer/causes-prevention/risk/age (accessed.
[71] S. S. Martin *et al.*, "2025 Heart Disease and Stroke Statistics: A Report of US and Global Data From the American Heart Association," *Circulation*, vol. 151, no. 8, pp. e41–e660, Feb 25 2025, doi: 10.1161/CIR.0000000000001303.
[72] R. J. Koene, A. E. Prizment, A. Blaes, and S. H. Konety, "Shared Risk Factors in Cardiovascular Disease and Cancer," *Circulation*, vol. 133, no. 11, pp. 1104–14, Mar 15 2016, doi: 10.1161/CIRCULATIONAHA.115.020406.
[73] H. S. Haugnes *et al.*, "Cardiovascular risk factors and morbidity in long-term survivors of testicular cancer: a 20-year follow-up study," *J Clin Oncol*, vol. 28, no. 30, pp. 4649–57, Oct 20 2010, doi: 10.1200/JCO.2010.29.9362.
[74] R. J. Motzer *et al.*, "Sunitinib versus interferon alfa in metastatic renal-cell carcinoma," *N Engl J Med*, vol. 356, no. 2, pp. 115–24, Jan 11 2007, doi: 10.1056/NEJMoa065044.
[75] G. D. Demetri *et al.*, "Efficacy and safety of sunitinib in patients with advanced gastrointestinal stromal tumour after failure of imatinib: a randomised controlled trial," *Lancet*, vol. 368, no. 9544, pp. 1329–38, Oct 14 2006, doi: 10.1016/S0140-6736(06)69446-4.
[76] D. Di Lisi *et al.*, "Anticancer therapy-induced vascular toxicity: VEGF inhibition and beyond," *Int J Cardiol*, vol. 227, pp. 11–17, Jan 15 2017, doi: 10.1016/j.ijcard.2016.11.174.
[77] H. M. Chang, T. M. Okwuosa, T. Scarabelli, R. Moudgil, and E. T. H. Yeh, "Cardiovascular Complications of Cancer Therapy: Best Practices in Diagnosis, Prevention, and Management: Part 2," *J Am Coll Cardiol*, vol. 70, no. 20, pp. 2552–2565, Nov 14 2017, doi: 10.1016/j.jacc.2017.09.1095.

Journal Pre-proof

[78] R. M. Touyz and J. Herrmann, "Cardiotoxicity with vascular endothelial growth factor inhibitor therapy," *NPJ Precis Oncol,* vol. 2, p. 13, 2018, doi: 10.1038/s41698-018-0056-z.
[79] E. Gara, K. G. Csiko, Z. Ruzsa, G. Foldes, and B. Merkely, "Anti-cancer drugs-induced arterial injury: risk stratification, prevention, and treatment," *Med Oncol,* vol. 36, no. 8, p. 72, Jul 10 2019, doi: 10.1007/s12032-019-1295-8.
[80] J. L. Zamorano *et al.*, "2016 ESC Position Paper on cancer treatments and cardiovascular toxicity developed under the auspices of the ESC Committee for Practice Guidelines: The Task Force for cancer treatments and cardiovascular toxicity of the European Society of Cardiology (ESC)," *Eur Heart J,* vol. 37, no. 36, pp. 2768–2801, Sep 21 2016, doi: 10.1093/eurheartj/ehw211.
[81] C. Lestuzzi *et al.*, "Effort myocardial ischemia during chemotherapy with 5-fluorouracil: an underestimated risk," *Ann Oncol,* vol. 25, no. 5, pp. 1059–64, May 2014, doi: 10.1093/annonc/mdu055.
[82] A. Chiribiri *et al.*, "SCMR Expert Consensus Statement on Quantitative Myocardial Perfusion Cardiovascular Magnetic Resonance Imaging," *J Cardiovasc Magn Reson,* p. 101940, Aug 8 2025, doi: 10.1016/j.jocmr.2025.101940.
[83] M. Gulati *et al.*, "2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: Executive Summary: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines," *Circulation,* vol. 144, no. 22, pp. e368–e454, Nov 30 2021, doi: 10.1161/CIR.0000000000001030.
[84] K. D. Knott *et al.*, "Quantitative myocardial perfusion in coronary artery disease: A perfusion mapping study," *J Magn Reson Imaging,* vol. 50, no. 3, pp. 756–762, Sep 2019, doi: 10.1002/jmri.26668.
[85] S. J. H. Dobbin *et al.*, "Vascular endothelial growth factor inhibitor-induced cardiotoxicity: prospective multimodality assessment incorporating cardiovascular magnetic resonance imaging," *Heart,* vol. 111, no. 19, pp. 925–932, Sep 11 2025, doi: 10.1136/heartjnl-2024-325535.
[86] T. Kotecha *et al.*, "Automated Pixel-Wise Quantitative Myocardial Perfusion Mapping by CMR to Detect Obstructive Coronary Artery Disease and Coronary Microvascular Dysfunction: Validation Against Invasive Coronary Physiology," *JACC Cardiovasc Imaging,* vol. 12, no. 10, pp. 1958–1969, Oct 2019, doi: 10.1016/j.jcmg.2018.12.022.
[87] K. D. Knott *et al.*, "The Prognostic Significance of Quantitative Myocardial Perfusion: An Artificial Intelligence-Based Approach Using Perfusion Mapping," *Circulation,* vol. 141, no. 16, pp. 1282–1291, Apr 21 2020, doi: 10.1161/CIRCULATIONAHA.119.044666.
[88] A. Desai, A. Noor, S. Joshi, and A. S. Kim, "Takotsubo cardiomyopathy in cancer patients," *Cardiooncology,* vol. 5, p. 7, 2019, doi: 10.1186/s40959-019-0042-9.
[89] C. G. Lenneman and D. B. Sawyer, "Cardio-Oncology: An Update on Cardiotoxicity of Cancer-Related Treatment," *Circ Res,* vol. 118, no. 6, pp. 1008–20, Mar 18 2016, doi: 10.1161/CIRCRESAHA.115.303633.
[90] N. Chaosuwannakit *et al.*, "Aortic stiffness increases upon receipt of anthracycline chemotherapy," *J Clin Oncol,* vol. 28, no. 1, pp. 166–72, Jan 1 2010, doi: 10.1200/JCO.2009.23.8527.

Journal Pre-proof

[91] N. Arnold *et al.*, "Promotion of Arterial Stiffness by Childhood Cancer and Its Characteristics in Adult Long-Term Survivors," *J Am Heart Assoc*, vol. 10, no. 5, p. e015609, Feb 2021, doi: 10.1161/JAHA.119.015609.
[92] J. I. Krystal, M. Reppucci, T. Mayr, J. D. Fish, and C. Sethna, "Arterial stiffness in childhood cancer survivors," *Pediatr Blood Cancer*, vol. 62, no. 10, pp. 1832–7, Oct 2015, doi: 10.1002/pbc.25547.
[93] S. Grover *et al.*, "Early and late changes in markers of aortic stiffness with breast cancer therapy," *Intern Med J*, vol. 45, no. 2, pp. 140–7, Feb 2015, doi: 10.1111/imj.12645.
[94] J. L. Cavalcante, J. A. Lima, A. Redheuil, and M. H. Al-Mallah, "Aortic stiffness: current understanding and future directions," *J Am Coll Cardiol*, vol. 57, no. 14, pp. 1511–22, Apr 5 2011, doi: 10.1016/j.jacc.2010.12.017.
[95] P. Gujja, D. R. Rosing, D. J. Tripodi, and Y. Shizukuda, "Iron overload cardiomyopathy: better understanding of an increasing disorder," *J Am Coll Cardiol*, vol. 56, no. 13, pp. 1001–12, Sep 21 2010, doi: 10.1016/j.jacc.2010.03.083.
[96] C. J. Murphy and G. Y. Oudit, "Iron-overload cardiomyopathy: pathophysiology, diagnosis, and treatment," *Journal of cardiac failure*, vol. 16, no. 11, pp. 888–900, Nov 2010, doi: 10.1016/j.cardfail.2010.05.009.
[97] D. T. Kremastinos and D. Farmakis, "Iron overload cardiomyopathy in clinical practice," *Circulation*, vol. 124, no. 20, pp. 2253–63, Nov 15 2011, doi: 10.1161/CIRCULATIONAHA.111.050773.
[98] J. C. Wood, "History and current impact of cardiac magnetic resonance imaging on the management of iron overload," *Circulation*, vol. 120, no. 20, pp. 1937–9, Nov 17 2009, doi: 10.1161/CIRCULATIONAHA.109.907196.
[99] B. Modell, M. Khan, M. Darlison, M. A. Westwood, D. Ingram, and D. J. Pennell, "Improved survival of thalassaemia major in the UK and relation to T2* cardiovascular magnetic resonance," *J Cardiovasc Magn Reson*, vol. 10, p. 42, Sep 25 2008, doi: 10.1186/1532-429X-10-42.
[100] L. J. Anderson, B. Wonke, E. Prescott, S. Holden, J. M. Walker, and D. J. Pennell, "Comparison of effects of oral deferiprone and subcutaneous desferrioxamine on myocardial iron concentrations and ventricular function in beta-thalassaemia," *Lancet*, vol. 360, no. 9332, pp. 516–20, Aug 17 2002, doi: 10.1016/s0140-6736(02)09740-4.
[101] D. J. Pennell *et al.*, "Cardiovascular function and treatment in beta-thalassemia major: a consensus statement from the American Heart Association," *Circulation*, vol. 128, no. 3, pp. 281–308, Jul 16 2013, doi: 10.1161/CIR.0b013e31829b2be6.
[102] A. Meloni *et al.*, "Myocardial iron overload by cardiovascular magnetic resonance native segmental T1 mapping: a sensitive approach that correlates with cardiac complications," *J Cardiovasc Magn Reson*, vol. 23, no. 1, p. 70, Jun 14 2021, doi: 10.1186/s12968-021-00765-w.
[103] C. Torlasco *et al.*, "Role of T1 mapping as a complementary tool to T2* for non-invasive cardiac iron overload assessment," *PLoS One*, vol. 13, no. 2, p. e0192890, 2018, doi: 10.1371/journal.pone.0192890.
[104] A. D. Wechalekar, J. D. Gillmore, and P. N. Hawkins, "Systemic amyloidosis," *Lancet*, vol. 387, no. 10038, pp. 2641–2654, Jun 25 2016, doi: 10.1016/S0140-6736(15)01274-X.
[105] A. M. Maceira *et al.*, "Cardiovascular magnetic resonance in cardiac amyloidosis," *Circulation*, vol. 111, no. 2, pp. 186–93, Jan 18 2005, doi: 10.1161/01.CIR.0000152819.97857.9D.

Journal Pre-proof

[106] M. Fontana, R. Chung, P. N. Hawkins, and J. C. Moon, "Cardiovascular magnetic resonance for amyloidosis," *Heart Fail Rev,* vol. 20, no. 2, pp. 133–44, Mar 2015, doi: 10.1007/s10741-014-9470-7.
[107] M. Fontana *et al.,* "Native T1 mapping in transthyretin amyloidosis," *JACC Cardiovasc Imaging,* vol. 7, no. 2, pp. 157–65, Feb 2014, doi: 10.1016/j.jcmg.2013.10.008.
[108] T. D. Karamitsos *et al.,* "Noncontrast T1 mapping for the diagnosis of cardiac amyloidosis," *JACC Cardiovasc Imaging,* vol. 6, no. 4, pp. 488–97, Apr 2013, doi: 10.1016/j.jcmg.2012.11.013.
[109] S. M. Banypersad *et al.,* "T1 mapping and survival in systemic light-chain amyloidosis," *Eur Heart J,* vol. 36, no. 4, pp. 244–51, Jan 21 2015, doi: 10.1093/eurheartj/ehu444.
[110] A. Martinez-Naharro *et al.,* "Native T1 and Extracellular Volume in Transthyretin Amyloidosis," *JACC Cardiovasc Imaging,* Mar 12 2018, doi: 10.1016/j.jcmg.2018.02.006.
[111] T. Kotecha *et al.,* "Myocardial Edema and Prognosis in Amyloidosis," (in eng), *J Am Coll Cardiol,* vol. 71, no. 25, pp. 2919–2931, Jun 26 2018, doi: 10.1016/j.jacc.2018.03.536.
[112] A. Martinez-Naharro *et al.,* "CMR-Verified Regression of Cardiac AL Amyloid After Chemotherapy," *JACC Cardiovasc Imaging,* vol. 11, no. 1, pp. 152–154, Jan 2018, doi: 10.1016/j.jcmg.2017.02.012.
[113] M. Fontana *et al.,* "Reduction in CMR Derived Extracellular Volume With Patisiran Indicates Cardiac Amyloid Regression," *JACC Cardiovasc Imaging,* vol. 14, no. 1, pp. 189–199, Jan 2021, doi: 10.1016/j.jcmg.2020.07.043.
[114] S. Dorbala *et al.,* "ASNC/AHA/ASE/EANM/HFSA/ISA/SCMR/SNMMI Expert Consensus Recommendations for Multimodality Imaging in Cardiac Amyloidosis: Part 1 of 2-Evidence Base and Standardized Methods of Imaging," *Journal of cardiac failure,* vol. 25, no. 11, pp. e1–e39, Nov 2019, doi: 10.1016/j.cardfail.2019.08.001.
[115] S. Dorbala *et al.,* "ASNC/AHA/ASE/EANM/HFSA/ISA/SCMR/SNMMI Expert Consensus Recommendations for Multimodality Imaging in Cardiac Amyloidosis: Part 2 of 2-Diagnostic Criteria and Appropriate Utilization," (in eng), *Journal of nuclear cardiology : official publication of the American Society of Nuclear Cardiology,* Aug 29 2019, doi: 10.1007/s12350-019-01761-5.
[116] A. Sheikh *et al.,* "Myocardial Amyloid Burden in Transthyretin Amyloidosis," *J Am Coll Cardiol,* vol. 87, no. 5, pp. 505–518, Feb 10 2026, doi: 10.1016/j.jacc.2025.10.054.
[117] ACR Committee on Drugs and Contrast Media, *ACR Manual on Contrast Media 2025*. 2025.
[118] Society for Cardiovascular Magnetic Resonance. "Gadolinium Contrast Agent Safety and Significance of Recent Data on Brain and Body Deposition." https://scmr.site-ym.com/page/ContrastAgentSafety (accessed June 2, 2026.
[119] C. K. Ala, A. L. Klein, and J. J. Moslehi, "Cancer Treatment-Associated Pericardial Disease: Epidemiology, Clinical Presentation, Diagnosis, and Management," *Curr Cardiol Rep,* journal article vol. 21, no. 12, p. 156, November 25 2019, doi: 10.1007/s11886-019-1225-6.
[120] G. Piovaccari *et al.,* "Cardiac disease after chest irradiation for Hodgkin's disease: incidence in 108 patients with long follow-up," *Int J Cardiol,* vol. 49, no. 1, pp. 39–43, 1995/03/24/ 1995, doi: https://doi.org/10.1016/0167-5273(95)02276-3.

Journal Pre-proof

[121] P. McGale *et al.*, "Incidence of heart disease in 35,000 women treated with radiotherapy for breast cancer in Denmark and Sweden," *Radiother Oncol*, vol. 100, no. 2, pp. 167–175, 2011/08/01/ 2011, doi: https://doi.org/10.1016/j.radonc.2011.06.016.
[122] G. Gagliardi *et al.*, "Radiation dose-volume effects in the heart," *Int J Radiat Oncol Biol Phys*, vol. 76, no. 3 Suppl, pp. S77–85, Mar 1 2010, doi: 10.1016/j.ijrobp.2009.04.093.
[123] Y. Adler *et al.*, "2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC)Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS)," *Eur Heart J*, vol. 36, no. 42, pp. 2921–2964, Nov 7 2015, doi: 10.1093/eurheartj/ehv318.
[124] C. M. Kramer, J. Barkhausen, S. D. Flamm, R. J. Kim, E. Nagel, and P. Society for Cardiovascular Magnetic Resonance Board of Trustees Task Force on Standardized, "Standardized cardiovascular magnetic resonance (CMR) protocols 2013 update," *J Cardiovasc Magn Reson*, vol. 15, p. 91, Oct 8 2013, doi: 10.1186/1532-429X-15-91.
[125] P. Thavendiranathan *et al.*, "Simultaneous right and left heart real-time, free-breathing CMR flow quantification identifies constrictive physiology," *JACC Cardiovasc Imaging*, vol. 5, no. 1, pp. 15–24, Jan 2012, doi: 10.1016/j.jcmg.2011.07.010.
[126] M. H. Crawford, "Chemotherapy-Induced Valvular Heart Disease," *JACC Cardiovasc Imaging*, vol. 9, no. 3, pp. 240–2, Mar 2016, doi: 10.1016/j.jcmg.2015.07.019.
[127] M. H. Stewart, E. Jahangir, and N. M. Polin, "Valvular Heart Disease in Cancer Patients: Etiology, Diagnosis, and Management," *Current treatment options in cardiovascular medicine*, vol. 19, no. 7, p. 53, Jul 2017, doi: 10.1007/s11936-017-0550-6.
[128] K. Murbraech *et al.*, "Valvular Dysfunction in Lymphoma Survivors Treated With Autologous Stem Cell Transplantation: A National Cross-Sectional Study," *JACC Cardiovasc Imaging*, vol. 9, no. 3, pp. 230–9, Mar 2016, doi: 10.1016/j.jcmg.2015.06.028.
[129] M. B. Lund *et al.*, "Increased risk of heart valve regurgitation after mediastinal radiation for Hodgkin's disease: an echocardiographic study," *Heart*, vol. 75, no. 6, pp. 591–595, 1996, doi: 10.1136/hrt.75.6.591.
[130] M. C. Hull, C. G. Morris, C. J. Pepine, and N. P. Mendenhall, "Valvular dysfunction and carotid, subclavian, and coronary artery disease in survivors of hodgkin lymphoma treated with radiation therapy," *JAMA*, vol. 290, no. 21, pp. 2831–7, Dec 3 2003, doi: 10.1001/jama.290.21.2831.
[131] G. Schellong *et al.*, "Late valvular and other cardiac diseases after different doses of mediastinal radiotherapy for Hodgkin disease in children and adolescents: report from the longitudinal GPOH follow-up project of the German-Austrian DAL-HD studies," (in eng), *Pediatr Blood Cancer*, vol. 55, no. 6, pp. 1145–52, Dec 1 2010, doi: 10.1002/pbc.22664.
[132] J. M. Bijl *et al.*, "Assessment of Valvular Disorders in Survivors of Hodgkin's Lymphoma Treated by Mediastinal Radiotherapy +/- Chemotherapy," (in eng), *Am J Cardiol*, vol. 117, no. 4, pp. 691–696, Feb 15 2016, doi: 10.1016/j.amjcard.2015.11.027.
[133] M. Y. Desai *et al.*, "Prevention, Diagnosis, and Management of Radiation-Associated Cardiac Disease: JACC Scientific Expert Panel," (in eng), *J Am Coll Cardiol*, vol. 74, no. 7, pp. 905–927, Aug 20 2019, doi: 10.1016/j.jacc.2019.07.006.

Journal pre-proof

[134] T. Wethal *et al.*, "Valvular dysfunction and left ventricular changes in Hodgkin's lymphoma survivors. A longitudinal study," *Br J Cancer,* vol. 101, no. 4, pp. 575–81, Aug 18 2009, doi: 10.1038/sj.bjc.6605191.
[135] M. Y. Desai *et al.*, "Increased Aorto-Mitral Curtain Thickness Independently Predicts Mortality in Patients With Radiation-Associated Cardiac Disease Undergoing Cardiac Surgery," *Ann Thorac Surg,* vol. 97, no. 4, pp. 1348–1355, 2014/04/01/ 2014, doi: https://doi.org/10.1016/j.athoracsur.2013.12.029.
[136] L. A. Baldassarre *et al.*, "Advances in Multimodality Imaging in Cardio-Oncology: JACC State-of-the-Art Review," *J Am Coll Cardiol,* vol. 80, no. 16, pp. 1560–1578, Oct 18 2022, doi: 10.1016/j.jacc.2022.08.743.
[137] S. G. Myerson, "Heart valve disease: investigation by cardiovascular magnetic resonance," *J Cardiovasc Magn Reson,* vol. 14, 2012.
[138] R. A. Nishimura *et al.*, "2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines," *J Am Coll Cardiol,* vol. 63, no. 22, pp. e57–e185, 2014/06/10/ 2014, doi: https://doi.org/10.1016/j.jacc.2014.02.536.
[139] M. M. Bissell *et al.*, "4D Flow cardiovascular magnetic resonance consensus statement: 2023 update," *J Cardiovasc Magn Reson,* vol. 25, no. 1, p. 40, Jul 20 2023, doi: 10.1186/s12968-023-00942-z.
[140] S. Wieshammer, J. Dreyhaupt, D. Muller, F. Momm, and A. Jakob, "Venous thromboembolism and persistent pulmonary hypertension in cancer patients: a cross-sectional study," *Thromb J,* vol. 14, p. 3, 2016, doi: 10.1186/s12959-016-0077-1.
[141] N. Galie *et al.*, "2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension: The Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS): Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Heart and Lung Transplantation (ISHLT)," *Eur Heart J,* vol. 37, no. 1, pp. 67–119, Jan 1 2016, doi: 10.1093/eurheartj/ehv317.
[142] M. Schneider *et al.*, "Echocardiographic assessment of right ventricular function: current clinical practice," *Int J Cardiovasc Imaging,* vol. 35, no. 1, pp. 49–56, Jan 2019, doi: 10.1007/s10554-018-1428-8.
[143] F. Grothues, J. C. Moon, N. G. Bellenger, G. S. Smith, H. U. Klein, and D. J. Pennell, "Interstudy reproducibility of right ventricular volumes, function, and mass with cardiovascular magnetic resonance," *Am Heart J,* vol. 147, no. 2, pp. 218–23, Feb 2004, doi: 10.1016/j.ahj.2003.10.005.
[144] F. Grothues *et al.*, "Comparison of interstudy reproducibility of cardiovascular magnetic resonance with two-dimensional echocardiography in normal subjects and in patients with heart failure or left ventricular hypertrophy," *Am J Cardiol,* vol. 90, no. 1, pp. 29–34, Jul 1 2002, doi: 10.1016/s0002-9149(02)02381-0.
[145] A. J. Peacock and A. Vonk Noordegraaf, "Cardiac magnetic resonance imaging in pulmonary arterial hypertension," *Eur Respir Rev,* Review vol. 22, pp. 526–534, 2013, doi: 10.1183/09059180.00006313.
[146] B. H. Freed *et al.*, "Late gadolinium enhancement cardiovascular magnetic resonance predicts clinical worsening in patients with pulmonary hypertension," *J Cardiovasc Magn Reson,* vol. 14, p. 11, Feb 1 2012, doi: 10.1186/1532-429X-14-11.

Journal Pre-proof

[147] A. J. Swift *et al.*, "Diagnostic accuracy of cardiovascular magnetic resonance imaging of right ventricular morphology and function in the assessment of suspected pulmonary hypertension results from the ASPIRE registry," *J Cardiovasc Magn Reson,* vol. 14, p. 40, Jun 21 2012, doi: 10.1186/1532-429X-14-40.
[148] A. J. Swift *et al.*, "Noninvasive estimation of PA pressure, flow, and resistance with CMR imaging: derivation and prospective validation study from the ASPIRE registry," *JACC Cardiovasc Imaging,* vol. 6, no. 10, pp. 1036–1047, Oct 2013, doi: 10.1016/j.jcmg.2013.01.013.
[149] S. A. van Wolferen *et al.*, "Prognostic value of right ventricular mass, volume, and function in idiopathic pulmonary arterial hypertension," *Eur Heart J,* vol. 28, no. 10, pp. 1250–7, May 2007, doi: 10.1093/eurheartj/ehl477.
[150] G. J. Mauritz *et al.*, "Progressive changes in right ventricular geometric shortening and long-term survival in pulmonary arterial hypertension," *Chest,* vol. 141, no. 4, pp. 935–943, Apr 2012, doi: 10.1378/chest.10-3277.
[151] J. Kochav, L. Simprini, and J. W. Weinsaft, "Imaging of the right heart--CT and CMR," *Echocardiography,* vol. 32 Suppl 1, pp. S53–68, Jan 2015, doi: 10.1111/echo.12212.
[152] S. C. Darby *et al.*, "Risk of Ischemic Heart Disease in Women after Radiotherapy for Breast Cancer," *N Engl J Med,* vol. 368, no. 11, pp. 987–998, 2013, doi: 10.1056/NEJMoa1209825.
[153] J. P. Greenwood *et al.*, "Effect of Care Guided by Cardiovascular Magnetic Resonance, Myocardial Perfusion Scintigraphy, or NICE Guidelines on Subsequent Unnecessary Angiography Rates: The CE-MARC 2 Randomized Clinical Trial," *JAMA,* vol. 316, no. 10, pp. 1051–1060, 2016, doi: 10.1001/jama.2016.12680.
[154] E. Nagel *et al.*, "Magnetic Resonance Perfusion or Fractional Flow Reserve in Coronary Disease," *N Engl J Med,* vol. 380, no. 25, pp. 2418–2428, 2019, doi: 10.1056/NEJMoa1716734.
[155] M. Y. Desai, C. L. Jellis, R. Kotecha, D. R. Johnston, and B. P. Griffin, "Radiation-Associated Cardiac Disease: A Practical Approach to Diagnosis and Management," *JACC Cardiovasc Imaging,* vol. 11, no. 8, pp. 1132–1149, Aug 2018, doi: 10.1016/j.jcmg.2018.04.028.
[156] J. Wilson, C. Jun Hua, N. Aziminia, and C. Manisty, "Imaging of the Acute and Chronic Cardiovascular Complications of Radiation Therapy," *Circ Cardiovasc Imaging,* vol. 18, no. 3, p. e017454, Mar 2025, doi: 10.1161/CIRCIMAGING.124.017454.
[157] H. C. Baumgartner *et al.*, "Recommendations on the echocardiographic assessment of aortic valve stenosis: a focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography," *Eur Heart J Cardiovasc Imaging,* vol. 18, no. 3, pp. 254–275, Mar 1 2017, doi: 10.1093/ehjci/jew335.
[158] J. C. Lopez-Mattei and D. J. Shah, "The role of cardiac magnetic resonance in valvular heart disease," *Methodist DeBakey cardiovascular journal,* vol. 9, no. 3, pp. 142–8, Jul–Sep 2013, doi: 10.14797/mdcj-9-3-142.
[159] E. B. Tham *et al.*, "Diffuse myocardial fibrosis by T1-mapping in children with subclinical anthracycline cardiotoxicity: relationship to exercise capacity, cumulative dose and remodeling," *J Cardiovasc Magn Reson,* vol. 15, no. 1, p. 48, 2013/06/10 2013, doi: 10.1186/1532-429X-15-48.

Journal Pre-proof

[160] Y. J. Huang *et al.*, "Detection of late radiation damage on left atrial fibrosis using cardiac late gadolinium enhancement magnetic resonance imaging," *Adv Radiat Oncol,* vol. 1, no. 2, pp. 106–114, 2016/04/01/ 2016, doi: https://doi.org/10.1016/j.adro.2016.04.002.
[161] A. C. Society. "Stem Cell Transplant for Cancer." https://www.cancer.org/treatment/treatments-and-side-effects/treatment-types/stem-cell-transplant/why-stem-cell-transplants-are-used.html (accessed.
[162] D. G. Gent, M. Saif, R. Dobson, and D. J. Wright, "Cardiovascular Disease After Hematopoietic Stem Cell Transplantation in Adults: JACC: CardioOncology State-of-the-Art Review," *JACC CardioOncol,* vol. 6, no. 4, pp. 475–495, Aug 2024, doi: 10.1016/j.jaccao.2024.06.004.
[163] E. Peres *et al.*, "Cardiac complications in patients undergoing a reduced-intensity conditioning hematopoietic stem cell transplantation," (in eng), *Bone marrow transplantation,* vol. 45, no. 1, pp. 149–52, Jan 2010, doi: 10.1038/bmt.2009.97.
[164] T. Murdych and D. J. Weisdorf, "Serious cardiac complications during bone marrow transplantation at the University of Minnesota, 1977-1997," (in eng), *Bone marrow transplantation,* vol. 28, no. 3, pp. 283–7, Aug 2001, doi: 10.1038/sj.bmt.1703133.
[165] A. Singla *et al.*, "Incidence of supraventricular arrhythmias during autologous peripheral blood stem cell transplantation," (in eng), *Biology of blood and marrow transplantation : journal of the American Society for Blood and Marrow Transplantation,* vol. 19, no. 8, pp. 1233–7, Aug 2013, doi: 10.1016/j.bbmt.2013.05.019.
[166] K. Kataoka, T. Takahashi, H. Iwata, A. Hangaishi, K. Kumano, and M. Kurokawa, "Fulminant myocarditis after allogeneic bone marrow transplantation: successful cytomegalovirus therapy and mechanical circulatory support for bridge to recovery," (in eng), *Biology of blood and marrow transplantation : journal of the American Society for Blood and Marrow Transplantation,* vol. 16, no. 1, pp. 129–30, Jan 2010, doi: 10.1016/j.bbmt.2009.05.008.
[167] U. Jehn *et al.*, "Lethal cardiac and cerebral toxoplasmosis in a patient with acute myeloid leukemia after successful allogeneic bone marrow transplantation," (in eng), *Transplantation,* vol. 38, no. 4, pp. 430–3, Oct 1984.
[168] J. Hadem *et al.*, "One day from dyspnea to death--unsuccessful application of extracorporeal membrane oxygenation in toxoplasma myocarditis following bone marrow transplantation," (in eng), *Clin Res Cardiol,* vol. 95, no. 9, pp. 477–81, Sep 2006, doi: 10.1007/s00392-006-0402-y.
[169] A. M. Agha *et al.*, "Role of cardiovascular imaging for the diagnosis and prognosis of cardiac amyloidosis," (in eng), *Open Heart,* vol. 5, no. 2, p. e000881, 2018, doi: 10.1136/openhrt-2018-000881.
[170] P. Kirk *et al.*, "Cardiac T2* Magnetic Resonance for Prediction of Cardiac Complications in Thalassemia Major," *Circulation,* vol. 120, no. 20, pp. 1961–1968, 2009, doi: doi:10.1161/CIRCULATIONAHA.109.874487.
[171] A. C. Mertens *et al.*, "Cause-specific late mortality among 5-year survivors of childhood cancer: the Childhood Cancer Survivor Study," *J Natl Cancer Inst,* vol. 100, no. 19, pp. 1368–79, Oct 1 2008, doi: 10.1093/jnci/djn310.
[172] M. Tukenova *et al.*, "Role of cancer treatment in long-term overall and cardiovascular mortality after childhood cancer," *J Clin Oncol.,* vol. 28, no. 8, pp. 1308–1315, 2010. [Online]. Available: PM:20142603.

Journal Pre-proof

[173] R. C. Reulen *et al.*, "Long-term cause-specific mortality among survivors of childhood cancer," *JAMA*, vol. 304, no. 2, pp. 172–9, Jul 14 2010, doi: 10.1001/jama.2010.923.
[174] O. H. Toro-Salazar *et al.*, "Effect of myocardial dysfunction in cardiac morbidity and all cause mortality in childhood cancer subjects treated with anthracycline therapy," *Cardio-Oncology*, journal article vol. 1, no. 1, pp. 1–9, 2015, doi: 10.1186/s40959-015-0005-8.
[175] S. E. G. Burdach, M. A. Westhoff, M. F. Steinhauser, and K. M. Debatin, "Precision medicine in pediatric oncology," *Molecular and cellular pediatrics*, vol. 5, no. 1, p. 6, Aug 31 2018, doi: 10.1186/s40348-018-0084-3.
[176] V. B. Pai and M. C. Nahata, "Cardiotoxicity of chemotherapeutic agents: incidence, treatment and prevention," *Drug safety*, vol. 22, no. 4, pp. 263–302, Apr 2000, doi: 10.2165/00002018-200022040-00002.
[177] I. B. Rhea and G. H. Oliveira, "Cardiotoxicity of Novel Targeted Chemotherapeutic Agents," *Current treatment options in cardiovascular medicine*, vol. 20, no. 7, p. 53, Jun 19 2018, doi: 10.1007/s11936-018-0649-4.
[178] R. Moudgil and E. T. Yeh, "Mechanisms of Cardiotoxicity of Cancer Chemotherapeutic Agents: Cardiomyopathy and Beyond," *The Canadian journal of cardiology*, vol. 32, no. 7, pp. 863–870 e5, Jul 2016, doi: 10.1016/j.cjca.2016.01.027.
[179] D. J. Grieve and S. M. Davidson, "New insights into cardiotoxicity caused by chemotherapeutic agents," *British journal of pharmacology*, vol. 174, no. 21, pp. 3675–3676, Nov 2017, doi: 10.1111/bph.14025.
[180] C. Huang *et al.*, "Juvenile exposure to anthracyclines impairs cardiac progenitor cell function and vascularization resulting in greater susceptibility to stress-induced myocardial injury in adult mice," *Circulation*, vol. 121, no. 5, pp. 675–83, Feb 9 2010, doi: 10.1161/CIRCULATIONAHA.109.902221.
[181] F. Aminkeng *et al.*, "A coding variant in RARG confers susceptibility to anthracycline-induced cardiotoxicity in childhood cancer," *Nature genetics*, vol. 47, no. 9, pp. 1079–84, Sep 2015, doi: 10.1038/ng.3374.
[182] M. Krajinovic *et al.*, "Polymorphisms of ABCC5 and NOS3 genes influence doxorubicin cardiotoxicity in survivors of childhood acute lymphoblastic leukemia," (in eng), *The pharmacogenomics journal*, vol. 17, no. 1, p. 107, Jan 2017, doi: 10.1038/tpj.2016.86.
[183] H. Visscher *et al.*, "Pharmacogenomic prediction of anthracycline-induced cardiotoxicity in children," *J Clin Oncol*, vol. 30, no. 13, pp. 1422–8, May 1 2012, doi: 10.1200/JCO.2010.34.3467.
[184] J. G. Blanco *et al.*, "Anthracycline-Related Cardiomyopathy After Childhood Cancer: Role of Polymorphisms in Carbonyl Reductase Genes--A report From the Children's Oncology Group," *J Clin Oncol.*, JCO.2011.34.8987 pii ;10.1200/JCO.2011.34.8987 doi 2011. [Online]. Available: PM:22124095.
[185] S. H. Armenian *et al.*, "Genetic susceptibility to anthracycline-related congestive heart failure in survivors of haematopoietic cell transplantation," *British journal of haematology*, vol. 163, no. 2, pp. 205–13, Oct 2013, doi: 10.1111/bjh.12516.
[186] D. L. Hertz *et al.*, "Evidence for association of SNPs in ABCB1 and CBR3, but not RAC2, NCF4, SLC28A3 or TOP2B, with chronic cardiotoxicity in a cohort of breast cancer patients treated with anthracyclines," *Pharmacogenomics*, vol. 17, no. 3, pp. 231–40, Feb 2016, doi: 10.2217/pgs.15.162.
[187] J. G. Blanco *et al.*, "Genetic polymorphisms in the carbonyl reductase 3 gene CBR3 and the NAD(P)H:quinone oxidoreductase 1 gene NQO1 in patients who developed

Journal Pre-proof

anthracycline-related congestive heart failure after childhood cancer," *Cancer,* vol. 112, no. 12, pp. 2789–95, Jun 15 2008, doi: 10.1002/cncr.23534.
[188] A. Reichwagen *et al.,* "Association of NADPH oxidase polymorphisms with anthracycline-induced cardiotoxicity in the RICOVER-60 trial of patients with aggressive CD20(+) B-cell lymphoma," *Pharmacogenomics,* vol. 16, no. 4, pp. 361–72, 2015, doi: 10.2217/pgs.14.179.
[189] A. F. Semsei *et al.,* "ABCC1 polymorphisms in anthracycline-induced cardiotoxicity in childhood acute lymphoblastic leukaemia," *Cell biology international,* vol. 36, no. 1, pp. 79–86, Jan 2012, doi: 10.1042/CBI20110264.
[190] L. Wojnowski *et al.,* "NAD(P)H oxidase and multidrug resistance protein genetic polymorphisms are associated with doxorubicin-induced cardiotoxicity," *Circulation,* vol. 112, no. 24, pp. 3754–62, Dec 13 2005, doi: 10.1161/CIRCULATIONAHA.105.576850.
[191] D. Vivenza, M. Feola, O. Garrone, M. Monteverde, M. Merlano, and C. Lo Nigro, "Role of the renin-angiotensin-aldosterone system and the glutathione S-transferase Mu, Pi and Theta gene polymorphisms in cardiotoxicity after anthracycline chemotherapy for breast carcinoma," *The International journal of biological markers,* vol. 28, no. 4, pp. e336–47, Dec 17 2013, doi: 10.5301/jbm.5000041.
[192] H. Visscher *et al.,* "Genetic variants in SLC22A17 and SLC22A7 are associated with anthracycline-induced cardiotoxicity in children," *Pharmacogenomics,* vol. 16, no. 10, pp. 1065–76, 2015, doi: 10.2217/pgs.15.61.
[193] S. J. Morrissy, "Regulation of anti-oxidant and anti-apoptotic genes by progesterone in cardiomyocytes," Doctor of Philosophy Dissertation, Pharmacology and Toxicology, University of Arizona, Arizona, 2007. [Online]. Available: https://search.proquest.com/openview/1fe2140be71bcb7a7c643e8804d1780f/1?pq-origsite=gscholar&cbl=18750&diss=y
[194] C. Vulsteke *et al.,* "Clinical and genetic risk factors for epirubicin-induced cardiac toxicity in early breast cancer patients," *Breast cancer research and treatment,* vol. 152, no. 1, pp. 67–76, Jul 2015, doi: 10.1007/s10549-015-3437-9.
[195] Y. Shuai *et al.,* "Global gene expression profiles of MT knockout and wild-type mice in the condition of doxorubicin-induced cardiomyopathy," *Toxicology letters,* vol. 200, no. 1-2, pp. 77–87, Jan 15 2011, doi: 10.1016/j.toxlet.2010.10.017.
[196] X. Wang *et al.,* "CELF4 Variant and Anthracycline-Related Cardiomyopathy: A Children's Oncology Group Genome-Wide Association Study," *J Clin Oncol,* vol. 34, no. 8, pp. 863–70, Mar 10 2016, doi: 10.1200/JCO.2015.63.4550.
[197] X. Wang *et al.,* "Hyaluronan synthase 3 variant and anthracycline-related cardiomyopathy: a report from the children's oncology group," *J Clin Oncol,* vol. 32, no. 7, pp. 647–53, Mar 1 2014, doi: 10.1200/JCO.2013.50.3557.
[198] S. E. Lipshultz *et al.,* "Female sex and drug dose as risk factors for late cardiotoxic effects of doxorubicin therapy for childhood cancer," *N Engl J Med,* vol. 332, no. 26, pp. 1738–1743, 1995. [Online]. Available: PM:7760889.
[199] J. P. Krischer, S. Epstein, D. D. Cuthbertson, A. M. Goorin, M. L. Epstein, and S. E. Lipshultz, "Clinical cardiotoxicity following anthracycline treatment for childhood cancer: the Pediatric Oncology Group experience," *J Clin Oncol,* vol. 15, no. 4, pp. 1544–1552, 1997. [Online]. Available: PM:9193351.

Journal Pre-proof

[200] P. Kong, P. Christia, and N. G. Frangogiannis, "The pathogenesis of cardiac fibrosis," *Cellular and molecular life sciences : CMLS,* vol. 71, no. 4, pp. 549–74, Feb 2014, doi: 10.1007/s00018-013-1349-6.
[201] D. A. Knowles *et al.*, "Determining the genetic basis of anthracycline-cardiotoxicity by molecular response QTL mapping in induced cardiomyocytes," *eLife,* vol. 7, May 8 2018, doi: 10.7554/eLife.33480.
[202] V. T. Truong *et al.*, "Cardiac magnetic resonance tissue tracking in right ventricle: Feasibility and normal values," *Magnetic resonance imaging,* vol. 38, pp. 189–195, May 2017, doi: 10.1016/j.mri.2017.01.007.
[203] S. H. Armenian, S. K. Gelehrter, and E. J. Chow, "Strategies to prevent anthracycline-related congestive heart failure in survivors of childhood cancer," *Cardiology research and practice,* vol. 2012, p. 713294, 2012, doi: 10.1155/2012/713294.
[204] S. E. Lipshultz and M. J. Adams, "Cardiotoxicity after childhood cancer: beginning with the end in mind," *J Clin Oncol,* vol. 28, no. 8, pp. 1276–81, Mar 10 2010, doi: 10.1200/JCO.2009.26.5751.
[205] S. H. Armenian *et al.*, "Screening for cardiac dysfunction in anthracycline-exposed childhood cancer survivors," *Clinical cancer research : an official journal of the American Association for Cancer Research,* vol. 20, no. 24, pp. 6314–23, Dec 15 2014, doi: 10.1158/1078-0432.CCR-13-3490.
[206] M. J. Ehrhardt *et al.*, "Systematic review and updated recommendations for cardiomyopathy surveillance for survivors of childhood, adolescent, and young adult cancer from the International Late Effects of Childhood Cancer Guideline Harmonization Group," *Lancet Oncol,* vol. 24, no. 3, pp. e108–e120, Mar 2023, doi: 10.1016/S1470-2045(23)00012-8.
[207] O. P. Simonetti and S. Cook, "Technical aspects of pediatric CMR," *J Cardiovasc Magn Reson,* vol. 8, no. 4, pp. 581–93, 2006, doi: 10.1080/10976640600713715.
[208] H. N. Ntsinjana, M. L. Hughes, and A. M. Taylor, "The role of cardiovascular magnetic resonance in pediatric congenital heart disease," *J Cardiovasc Magn Reson,* vol. 13, p. 51, Sep 21 2011, doi: 10.1186/1532-429X-13-51.
[209] M. J. Adams, L. S. Constine, and S. E. Lipshultz, "Late effects of therapy for Hodgkin's lymphoma," *Curr Hematol Malig Rep,* 10.1007/s11899-007-0020-4 doi vol. 2, no. 3, pp. 143–150, 2007. [Online]. Available: PM:20425363.
[210] J. Akam-Venkata, V. I. Franco, and S. E. Lipshultz, "Late Cardiotoxicity: Issues for Childhood Cancer Survivors," (in eng), *Current treatment options in cardiovascular medicine,* vol. 18, no. 7, p. 47, Jul 2016, doi: 10.1007/s11936-016-0466-6.
[211] A. M. Goorin, A. R. Chauvenet, A. R. Perez-Atayde, J. Cruz, R. McKone, and S. E. Lipshultz, "Initial congestive heart failure, six to ten years after doxorubicin chemotherapy for childhood cancer," *J Pediatr,* vol. 116, no. 1, pp. 144–147, 1990. [Online]. Available: PM:2295956.
[212] S. E. Lipshultz, S. D. Colan, R. D. Gelber, A. R. Perez-Atayde, S. E. Sallan, and S. P. Sanders, "Late cardiac effects of doxorubicin therapy for acute lymphoblastic leukemia in childhood," *N Engl J Med,* 10.1056/NEJM199103213241205 doi vol. 324, no. 12, pp. 808–815, 1991. [Online]. Available: PM:1997853.
[213] M. J. Adams, S. E. Lipshultz, C. Schwartz, L. F. Fajardo, V. Coen, and L. S. Constine, "Radiation-associated cardiovascular disease: manifestations and management," *Semin*

Journal Pre-proof

Radiat Oncol, S1053-4296(03)00026-2 pii ;10.1016/S1053-4296(03)00026-2 doi vol. 13, no. 3, pp. 346–356, 2003. [Online]. Available: PM:12903022.

[214] M. J. Adams and S. E. Lipshultz, "Pathophysiology of anthracycline- and radiation-associated cardiomyopathies: implications for screening and prevention," *Pediatr Blood Cancer,* 10.1002/pbc.20352 doi vol. 44, no. 7, pp. 600–606, 2005. [Online]. Available: PM:15856486.

[215] L. Gianni, E. H. Herman, S. E. Lipshultz, G. Minotti, N. Sarvazyan, and D. B. Sawyer, "Anthracycline cardiotoxicity: from bench to bedside," *J Clin Oncol,* 26/22/3777 pii ;10.1200/JCO.2007.14.9401 doi vol. 26, no. 22, pp. 3777–3784, 2008. [Online]. Available: PM:18669466.

[216] S. Bhatia *et al.*, "Collaborative Research in Childhood Cancer Survivorship: The Current Landscape," *J Clin Oncol,* vol. 33, no. 27, pp. 3055–64, Sep 20 2015, doi: 10.1200/JCO.2014.59.8052.

[217] V. J. Ferrans, J. R. Clark, J. Zhang, Z. X. Yu, and E. H. Herman, "Pathogenesis and prevention of doxorubicin cardiomyopathy," *Tsitologiia,* vol. 39, no. 10, pp. 928–937, 1997. [Online]. Available: PM:9505340.

[218] A. M. Segura, R. Radovancevic, Z. T. Demirozu, O. H. Frazier, and L. M. Buja, "Anthracycline treatment and ventricular remodeling in left ventricular assist device patients," *Texas Heart Institute journal,* vol. 42, no. 2, pp. 124–30, Apr 2015, doi: 10.14503/THIJ-14-4509.

[219] O. Lencova-Popelova *et al.*, "Molecular remodeling of left and right ventricular myocardium in chronic anthracycline cardiotoxicity and post-treatment follow up," *PLoS One,* vol. 9, no. 5, p. e96055, 2014, doi: 10.1371/journal.pone.0096055.

[220] C. J. Zeiss *et al.*, "Doxorubicin-Induced Cardiotoxicity in Collaborative Cross (CC) Mice Recapitulates Individual Cardiotoxicity in Humans," *G3,* Jul 1 2019, doi: 10.1534/g3.119.400232.

[221] V. J. Ferrans, "Overview of cardiac pathology in relation to anthracycline cardiotoxicity," *Cancer Treat Rep,* vol. 62, no. 6, pp. 955–961, 1978. [Online]. Available: PM:352510.

[222] A. Omidi, E. Weiss, C. R. Trankle, M. Rosu-Bubulac, and J. S. Wilson, "Quantitative assessment of radiotherapy-induced myocardial damage using MRI: a systematic review," *Cardiooncology,* vol. 9, no. 1, p. 24, May 18 2023, doi: 10.1186/s40959-023-00175-0.

[223] O. Yersal, U. Eryilmaz, H. Akdam, N. Meydan, and S. Barutca, "Arterial Stiffness in Breast Cancer Patients Treated with Anthracycline and Trastuzumab-Based Regimens," *Cardiology research and practice,* vol. 2018, p. 5352914, 2018, doi: 10.1155/2018/5352914.

[224] A. T. Chan *et al.*, "Prognostic utility of differential tissue characterization of cardiac neoplasm and thrombus via late gadolinium enhancement cardiovascular magnetic resonance among patients with advanced systemic cancer," *J Cardiovasc Magn Reson,* vol. 19, p. 76, 10/12
03/15/received
10/04/accepted 2017, doi: 10.1186/s12968-017-0390-2.

[225] C. Shenoy *et al.*, "Cardiovascular magnetic resonance imaging in suspected cardiac tumour: a multicentre outcomes study," *Eur Heart J,* vol. 43, no. 1, pp. 71–80, Dec 28 2021, doi: 10.1093/eurheartj/ehab635.

Journal Pre-proof

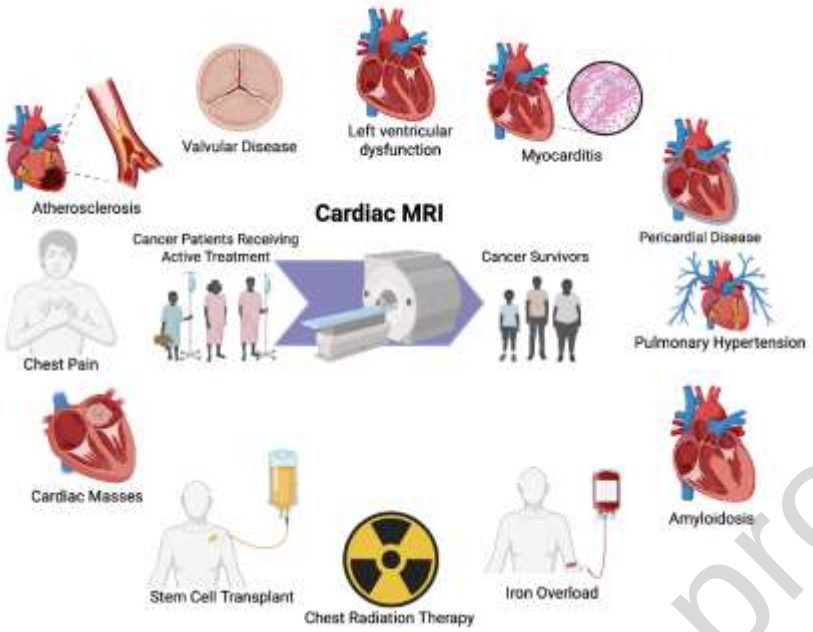
[226] C. Basso, S. Rizzo, M. Valente, and G. Thiene, "Cardiac masses and tumours," *Heart*, vol. 102, no. 15, pp. 1230–45, Aug 1 2016, doi: 10.1136/heartjnl-2014-306364.
[227] T. Leiner *et al.*, "SCMR Position Paper (2020) on clinical indications for cardiovascular magnetic resonance," *J Cardiovasc Magn Reson*, vol. 22, no. 1, p. 76, Nov 9 2020, doi: 10.1186/s12968-020-00682-4.
[228] A. R. Lyon *et al.*, "2022 ESC Guidelines on cardio-oncology developed in collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO) and the International Cardio-Oncology Society (IC-OS)," *Eur Heart J*, Aug 26 2022, doi: 10.1093/eurheartj/ehac244.
[229] S. C. Pun *et al.*, "Pattern and Prognostic Implications of Cardiac Metastases Among Patients With Advanced Systemic Cancer Assessed With Cardiac Magnetic Resonance Imaging," (in eng), *J Am Heart Assoc*, vol. 5, no. 5, May 2016, doi: 10.1161/jaha.116.003368.
[230] P. Pazos-Lopez *et al.*, "Value of CMR for the differential diagnosis of cardiac masses," (in eng), *JACC Cardiovasc Imaging*, vol. 7, no. 9, pp. 896–905, Sep 2014, doi: 10.1016/j.jcmg.2014.05.009.
[231] N. Mousavi *et al.*, "Assessment of Cardiac Masses by Cardiac Magnetic Resonance Imaging: Histological Correlation and Clinical Outcomes," *J Am Heart Assoc*, vol. 8, no. 1, p. e007829, Jan 8 2019. [Online]. Available: https://www.ncbi.nlm.nih.gov/pubmed/30616453.
[232] J. W. Weinsaft *et al.*, "Detection of left ventricular thrombus by delayed-enhancement cardiovascular magnetic resonance prevalence and markers in patients with systolic dysfunction," *J Am Coll Cardiol*, vol. 52, no. 2, pp. 148–57, Jul 8 2008, doi: 10.1016/j.jacc.2008.03.041.
[233] T. Leiner *et al.*, "Machine learning in cardiovascular magnetic resonance: basic concepts and applications," *J Cardiovasc Magn Reson*, vol. 21, no. 1, p. 61, Oct 7 2019, doi: 10.1186/s12968-019-0575-y.
[234] P. Collier, M. Hussain, Z. B. Popovic, and B. P. Griffin, "Cardiac surveillance for anti-HER2 chemotherapy," *Cleve Clin J Med*, vol. 88, no. 2, pp. 110–116, Feb 1 2021, doi: 10.3949/ccjm.88a.19150.
[235] B. Kenigsberg, A. Wellstein, and A. Barac, "Left Ventricular Dysfunction in Cancer Treatment: Is it Relevant?," *JACC. Heart failure*, vol. 6, no. 2, pp. 87–95, Feb 2018, doi: 10.1016/j.jchf.2017.08.024.
[236] National Cancer Institute; Cancer Therapy Evaluation Program. "Common Terminology Criteria for Adverse Events (CTCAE), Version 5.0." https://ctep.cancer.gov/protocoldevelopment/electronic_applications/docs/ctcae_v5_quick_reference_5x7.pdf (accessed November 26, 2021).
[237] G. Gulati *et al.*, "Prevention of cardiac dysfunction during adjuvant breast cancer therapy (PRADA): a 2 x 2 factorial, randomized, placebo-controlled, double-blind clinical trial of candesartan and metoprolol," *Eur Heart J*, vol. 37, no. 21, pp. 1671–80, Jun 1 2016, doi: 10.1093/eurheartj/ehw022.
[238] S. L. Heck *et al.*, "Prevention of Cardiac Dysfunction During Adjuvant Breast Cancer Therapy (PRADA): Extended Follow-Up of a 2x2 Factorial, Randomized, Placebo-Controlled, Double-Blind Clinical Trial of Candesartan and Metoprolol," *Circulation*, vol. 143, no. 25, pp. 2431–2440, Jun 22 2021, doi: 10.1161/CIRCULATIONAHA.121.054698.

Journal Pre-proof

[239] E. Pituskin *et al.*, "Multidisciplinary Approach to Novel Therapies in Cardio-Oncology Research (MANTICORE 101-Breast): A Randomized Trial for the Prevention of Trastuzumab-Associated Cardiotoxicity," *J Clin Oncol*, vol. 35, no. 8, pp. 870–877, Mar 10 2017, doi: 10.1200/JCO.2016.68.7830.
[240] A. Barac, A. Blaes, and F. Lynce, "Lessons From Primary Cardiac Prevention Trials During Trastuzumab Therapy: End of One Size Fits All," *J Am Coll Cardiol*, vol. 73, no. 22, pp. 2869–2871, Jun 11 2019, doi: 10.1016/j.jacc.2019.04.006.
[241] D. Addison *et al.*, "Equity in Cardio-Oncology Care and Research: A Scientific Statement From the American Heart Association," *Circulation*, vol. 148, no. 3, pp. 297–308, Jul 18 2023, doi: 10.1161/CIR.0000000000001158.
[242] J. A. Alvarez-Cardona *et al.*, "Cardio-Oncology Education and Training: JACC Council Perspectives," *J Am Coll Cardiol*, vol. 76, no. 19, pp. 2267–2281, Nov 10 2020, doi: 10.1016/j.jacc.2020.08.079.
[243] D. C. Lee *et al.*, "The growth and evolution of cardiovascular magnetic resonance: a 20-year history of the Society for Cardiovascular Magnetic Resonance (SCMR) annual scientific sessions," *J Cardiovasc Magn Reson*, vol. 20, no. 1, p. 8, Jan 31 2018, doi: 10.1186/s12968-018-0429-z.
[244] S. F. Dent *et al.*, "Optimizing Cardiovascular Health in Patients With Cancer: A Practical Review of Risk Assessment, Monitoring, and Prevention of Cancer Treatment-Related Cardiovascular Toxicity," *Am Soc Clin Oncol Educ Book*, vol. 40, pp. 1–15, Mar 2020, doi: 10.1200/EDBK_286019.
[245] L. A. Baldassarre *et al.*, "2025 ACC/AHA/ASE/ASNC/SCCT/SCMR Advanced Training Statement on Advanced Cardiovascular Imaging: A Report of the ACC Competency Management Committee," *J Am Coll Cardiol*, vol. 87, no. 5, pp. 570–632, Feb 10 2026, doi: 10.1016/j.jacc.2025.07.036.
[246] Y. Hua *et al.*, "Deep learning reconstruction for fast cardiovascular magnetic resonance imaging protocol: A comparative study with conventional cardiovascular magnetic resonance," *J Cardiovasc Magn Reson*, vol. 28, no. 1, p. 102017, Nov 28 2025, doi: 10.1016/j.jocmr.2025.102017.

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