



Review article

Cardiac magnetic resonance parameters associated with myocarditis in patients treated with immunotherapy: a scoping review

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Received: December 10, 2025.
Accepted: February 26, 2026.
Online: March 6, 2026.

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Funding

Self-funded.

Conflicts of interest

None.

Cite as

Quispe-Silvestre E, Gamonal-Moran KV, Dragonetti LC, Almeida Arostegui N. Cardiac magnetic resonance parameters associated with myocarditis in patients treated with immunotherapy: a scoping review. Arch Peru Cardiol Cir Cardiovasc. 2026;7(1). doi: 10.47487/apcyccv.v7i1.586.



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ABSTRACT

This scoping review, conducted in accordance with the PRISMA-ScR methodology, maps the available scientific evidence on cardiac magnetic resonance (CMR) abnormalities associated with immune checkpoint inhibitor-induced myocarditis (ICI-M). Twenty studies published between 2019 and 2025 were analysed, highlighting alterations in left ventricular function (left ventricular ejection fraction [LVEF]), myocardial oedema detected by T2-weighted sequences, and fibrosis identified as late gadolinium enhancement (LGE). Key diagnostic criteria included elevated extracellular volume (ECV), impaired global longitudinal strain (GLS), and abnormal T1 and T2 relaxation times. Of the included studies, most were retrospective, followed by case reports and prospective studies. The therapies most frequently associated with ICI-M involved combinations of anti-PD1 and anti-CTLA4 agents. Cardiac magnetic resonance (CMR) has proven to be a highly sensitive, non-invasive tool for detecting myocardial involvement, even before the elevation of biomarkers such as troponins. The importance of using advanced tools like CMR to assess cardiovascular complications associated with myocarditis is highlighted. Parameters such as LVEF, GLS, LGE, and ECV provide valuable information on ventricular function and structural alterations related to myocarditis. These findings allow us to identify knowledge gaps and guide future research on the use of CMR in myocarditis.

Keywords: Cardio-Oncology; Biomarkers; Immunotherapy; Diagnostic Imaging (Source: MeSH-NLM).

RESUMEN

Parámetros de resonancia magnética cardiaca asociados a miocarditis por inmunoterapia en pacientes oncológicos: una revisión de alcance

Esta revisión de alcance, basada en la metodología PRISMA-ScR, mapea la evidencia científica disponible sobre parámetros alterados en la resonancia magnética cardíaca (RMC) asociados con miocarditis inducida por inhibidores del punto de control inmunitario (ICI-M). Se analizaron veinte estudios publicados entre 2019 y 2025, destacando alteraciones en la función ventricular izquierda (FEVI), edema miocárdico detectado por secuencias ponderadas en T2 y fibrosis identificada como realce tardío de gadolinio (RTG). Los principales criterios diagnósticos incluyeron parámetros como volumen extracelular (VEC) elevado, strain longitudinal global (SLG) alterado y tiempos de relajación T1/T2 anormales. Del total de estudios revisados, la mayoría fueron retrospectivos, seguidos por informes de casos y estudios prospectivos. Las terapias más frecuentemente asociadas con ICI-M involucraron combinaciones de agentes anti-PD1 y anti-CTLA4. La RMC ha demostrado ser una herramienta altamente sensible y no invasiva para detectar la afectación miocárdica, incluso en ausencia de alteraciones significativas en biomarcadores en algunos casos reportados. Se destaca la importancia de utilizar herramientas avanzadas como la RMC para evaluar las complicaciones cardiovasculares asociadas con la miocarditis. Parámetros como la FEVI, el SLG, el RTG y el VEC proporcionan información valiosa sobre la función ventricular y las alteraciones estructurales relacionadas con la miocarditis. Estos hallazgos nos permiten identificar lagunas en el conocimiento y orientar la investigación futura sobre el uso de la RMC en la miocarditis.

Palabras clave: Cardiooncología; Biomarcadores; Inmunoterapia Activa; Diagnóstico por Imagen (Fuente: DeCS-BIREME).

Introduction

Myocarditis associated with immunotherapy, particularly with immune checkpoint inhibitors (ICIs), represents a serious adverse event, with a reported incidence ranging from 0.06% to 2.4% according to multicentre studies ⁽¹⁾. As the number of patients receiving ICI therapies increases each year, so does the risk of severe complications. Most patients treated with ICIs develop some degree of immune-related toxicity, which may affect virtually any organ. In combination PD-1/CTLA-4 therapies, adverse events occur in nearly all patients, with severe toxicities (grade ≥ 3) in approximately 40-50%, whereas with anti-PD-1 monotherapy, severe toxicities affect around 20% and mild to moderate events occur in 60-70% of cases ⁽²⁾. The underlying pathophysiology involves loss of immune tolerance, whereby T cells activated by ICIs attack not only tumour cells but also healthy tissues such as the myocardium. It is characterised by predominantly lymphocytic myocardial infiltration, involving CD8+ and CD4+ T cells and PD-L1+ macrophages. This infiltrate may trigger arrhythmias, heart failure, and sudden death ⁽³⁾, underscoring the importance of early diagnosis and appropriate management.

Clinical presentation is highly variable, ranging from asymptomatic cases detected through electrocardiographic abnormalities to severe presentations with chest pain, dyspnoea, or acute heart failure. It may also manifest with arrhythmias, syncope, or even sudden death. Diagnosis is based on clinical suspicion supported by investigations such as electrocardiography, biomarker analysis (troponin, NT-proBNP), and echocardiography, although endomyocardial biopsy remains the gold standard for confirming inflammation and non-ischaemic necrosis ⁽⁴⁾.

In this context, cardiac magnetic resonance (CMR) has emerged as a key non-invasive diagnostic tool. CMR enables comprehensive assessment of cardiac structure and function, as well as detailed tissue characterisation, allowing detection of myocardial inflammation and fibrosis ^(2,5). Several sequences and specific parameters are used, including myocardial oedema (T2); the presence of oedema, indicated by increased signal intensity on T2-weighted sequences such as STIR and T2 mapping, suggests inflammation and is an early marker of myocarditis. In addition, late gadolinium enhancement (LGE) allows identification of areas of replacement fibrosis or myocardial necrosis, which appear as regions of increased myocardial signal intensity on these sequences ⁽⁶⁾. The enhancement pattern may help differentiate myocarditis from other cardiomyopathies. Another parameter is extracellular volume (ECV), which quantifies expansion of the extracellular space and may be increased in cases of inflammation or fibrosis; this parameter is particularly useful for detecting subtle changes in the extracellular matrix ⁽⁶⁾.

Beyond its diagnostic utility, CMR also plays a crucial role in patient follow-up, enabling monitoring of treatment response and disease progression. Despite its advantages, CMR has limitations, including limited availability in some medical

centres, long acquisition times, high cost, claustrophobia, and the need for specialised personnel for acquisition and interpretation. Its use may also be restricted in patients with implanted cardiac devices or contraindications to gadolinium administration ⁽⁷⁾.

Eichhorn *et al.* ⁽²⁾ note that myocarditis assessment with CMR includes evaluation of cardiac chamber dimensions and function, T2-weighted imaging, LGE, T1 and T2 mapping, and calculation of ECV fraction. Emerging techniques such as CMR feature-tracking, texture analysis, and artificial intelligence are being developed to improve diagnosis and prognosis. This review highlights the value of CMR in clinical decision-making, monitoring, and risk stratification across different stages of this condition.

Similarly, Arcari *et al.* ⁽⁸⁾ examined the use of CMR in ICI-induced myocarditis. Their analysis showed that CMR findings included preserved left ventricular ejection fraction, with myocardial oedema prevalence ranging from 9% to 60% and late gadolinium enhancement from 23% to 83%. T1 and T2 mapping, performed in 108 and 104 patients respectively, showed partial concordance with endomyocardial biopsy, with native T1 mapping emerging as the imaging biomarker with the highest concordance. CMR findings in ICI-induced myocarditis included myocardial dysfunction, oedema, and fibrosis, although these were less pronounced than in classical forms of myocarditis, and native T1 mapping demonstrated significant prognostic value.

A scoping review approach was selected given that the evidence on CMR in ICI-induced myocarditis is emerging, heterogeneous, and encompasses different study designs. This type of review allows mapping of study types, imaging parameters used, and knowledge gaps without restricting inclusion based on methodological quality. Accordingly, the objective was to map the available scientific evidence on commonly altered or reported parameters in CMR studies among patients receiving immunotherapy.

Methods

This review was conducted in accordance with the PRISMA-ScR guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews), which provide a structured framework for describing the existing literature on a given topic (**Figure 1**). This design was selected due to the heterogeneity of the studies and the exploratory nature of the review. Two independent reviewers performed the initial study selection and assessment, and the Rayyan platform was used for the screening process and resolution of discrepancies.

Eligibility criteria and information sources

Eligibility criteria were defined according to the PCC framework (Population, Concept, Context), ensuring a relevant selection of studies. The population included adult oncology

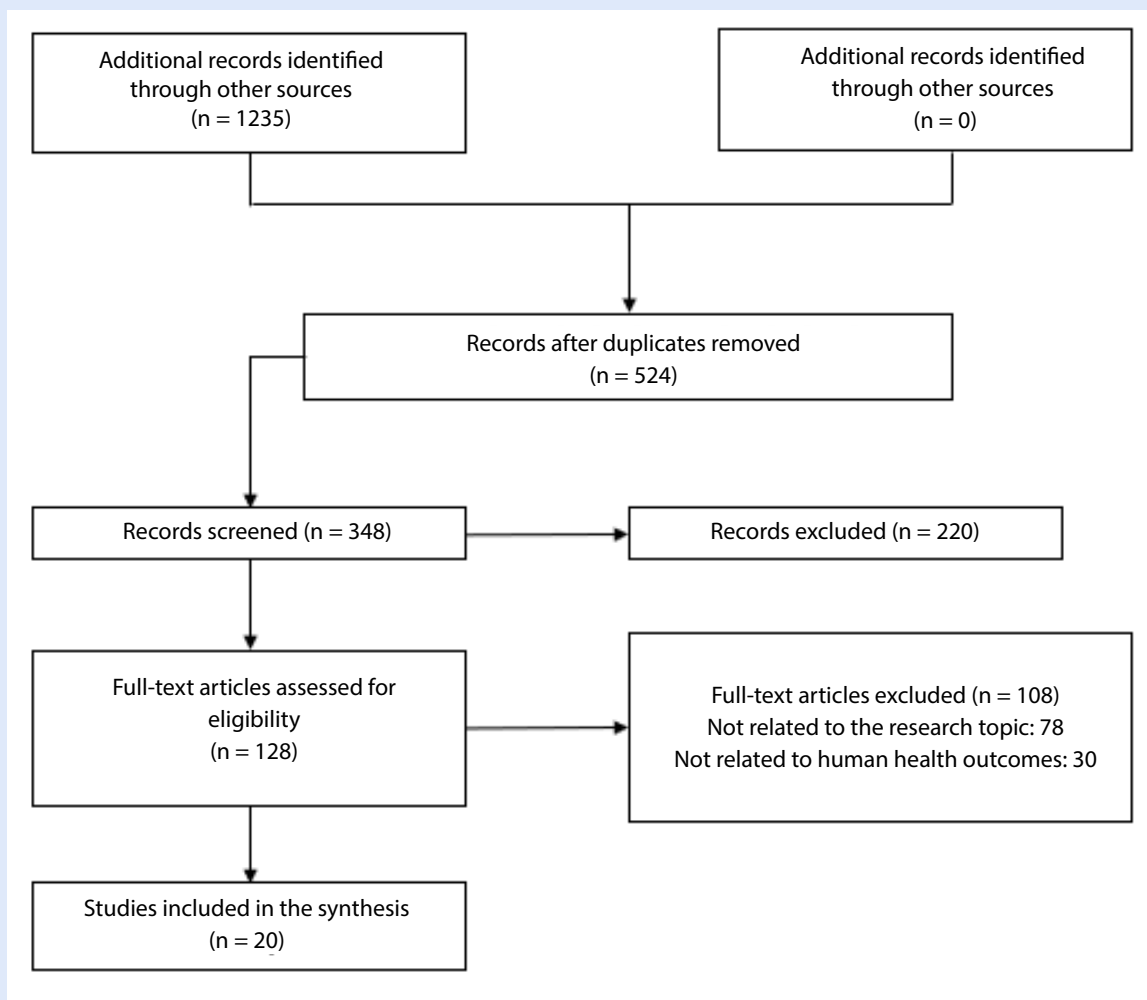


Figure 1. Flow diagram of the study selection process according to PRISMA-ScR.

patients receiving immunotherapy with suspected and/or confirmed myocarditis associated with treatment. The concept encompassed CMR parameters used for the evaluation or diagnosis of immunotherapy-associated myocarditis. The context included studies conducted in any clinical setting in which CMR was used to assess immunotherapy-related cardiotoxicity.

Inclusion criteria comprised adult patients diagnosed with myocarditis by CMR who had received immunotherapy for any type of cancer. Observational studies, case reports, or clinical trials published between 2019 and 2025, in Spanish or English, were included. Exclusion criteria were patients younger than 18 years, experimental studies using animal models, cases of myocarditis diagnosed without the use of CMR, or myocarditis caused by chemotherapy.

The literature search was conducted in three specialised databases: Scopus, PubMed, and SciELO. These databases were selected due to their relevance for biomedical publications in oncology, cardiology, and imaging, and are considered reliable, high-impact sources. PubMed represents a primary

source of evidence in health sciences, Scopus provides broad international multidisciplinary coverage, and SciELO enables inclusion of regional Latin American literature.

Search strategy and selection process

A search strategy was developed using MeSH terms and keywords, including myocarditis, diagnostic imaging, CMR, immunotherapy, late contrast enhancement, myocardial injury biomarkers, and immune checkpoint inhibitors. During the identification phase, duplicates and studies not meeting inclusion criteria were removed, reducing the initial 1,235 records to 348 selected based on titles and abstracts. In the screening stage, study objectives, design, abstracts, imaging techniques, type of immunotherapy, specific focus on ICI-related myocarditis, and reported altered parameters were assessed. This process allowed exclusion of non-relevant studies, reviews, and publications with insufficient data. Finally, after applying eligibility criteria and removing duplicates, 20 articles met the inclusion criteria for this review. No formal methodological quality assessment was performed, as the aim

of the study was to map the available evidence rather than to assess internal validity.

Results

Publication trends and geographical distribution

All included studies addressed immune checkpoint inhibitor-associated myocarditis (ICI-M), with three specifically focusing on lymphocytic myocarditis induced by ICIs. The ICIs evaluated included anti-PD-1, anti-PD-L1, and anti-CTLA-4 agents, predominantly in combination regimens of anti-PD-1 (nivolumab, pembrolizumab) and anti-CTLA-4 (ipilimumab). Of the 20 studies, 60% were retrospective, 35% were case reports, and only one study (5%) was prospective.

The temporal distribution of publications was heterogeneous: 25% in 2024; 20% in 2020 and 2021; 10% in 2019, 2022, and 2025; and 5% in 2023. Most studies originated from Germany (20%), followed by the United States and France (15% each), and China (10%). Other countries, including Japan, Ireland, Spain, Italy, the United Kingdom, Brazil, Australia, and Finland, each contributed 5%, corresponding to one study per country (Table 1)⁽⁹⁻²⁸⁾.

Types of parameters investigated

Among the 20 studies reviewed, nine reported normal left ventricular ejection fraction (LVEF >50%) in all evaluated patients^(12-14,16-18,22,23,28). One study observed a slight improvement in LVEF compared with baseline measurements, increasing from values close to the lower limit of normal to a range of 51-55% (15). Two studies reported a mild reduction in LVEF relative to baseline values, although still above 50%^(10,26); additionally, three studies reported heart failure, defined as LVEF <50% (20,21,24). Finally, in three studies, LVEF was either not assessed or no abnormalities were identified^(9,11,25).

Although quantitative data were not provided in several studies, reductions in LVEF in some cohorts suggest impaired myocardial contractility. This indicates that ventricular function is compromised in certain patients, which may be associated with different cardiac pathologies. LVEF remains a key parameter for evaluating ventricular function and classifying heart failure. However, variability in its assessment and reporting may limit comparability across studies.

The next parameter, global longitudinal strain (GLS), was not evaluated in 15 of the included studies. However, several studies provided relevant data. Faron *et al.*⁽¹⁰⁾ reported a baseline GLS value of $-23.4\% \pm 4.8$, which decreased to $-19.6\% \pm 5.1$ after intervention, with a statistically significant difference ($p = 0.005$). Similarly, Hichem *et al.*⁽¹⁹⁾ reported normal values (-19.9%) followed by abnormal findings with apical sparing (-9.8%). These findings suggest a reduction

in myocardial function following intervention, indicating a negative impact on myocardial performance.

The absence of GLS assessment in most studies suggests that this parameter is not widely used or reported in the available literature. This may be related to challenges in its measurement, the lack of standardised interpretation criteria, or the requirement for specialised software for analysis, which increases costs. Nevertheless, studies that evaluated GLS may provide valuable insights into underlying myocardial function.

Higgins *et al.*⁽¹⁷⁾ showed that patients with abnormal GLS had a mean value of $-9.8\% \pm 4.2$, whereas those with normal LVEF exhibited a GLS of $-12.3\% \pm 2.4$. These findings suggest the presence of subclinical myocardial dysfunction detectable by GLS, even when LVEF remains within normal ranges.

A significant negative correlation between GLS and LVEF was also observed ($r_s = -0.64$, $p < 0.002$), indicating that myocardial dysfunction identified by GLS may precede reductions in LVEF, highlighting its potential role in the early detection of cardiotoxicity. Li *et al.*⁽²⁶⁾ evaluated GLS across different myocardial layers. For the subendocardial layer (GLSendo), values were -14.61 ± 2.67 in the corticosteroid-refractory ICI-M group (CR) and -18.50 ± 2.53 in the corticosteroid-sensitive group (CS). For the subepicardial layer (GLSepsi), values were -14.75 ± 2.53 in the CR group and -16.68 ± 2.05 in the CS group. Compared with the control group, which had a GLSepsi of -19.31 ± 1.80 , the CS group showed impairment in this parameter. These results suggest layer-specific differences in myocardial function, with greater impairment observed in the CS group compared with controls.

Regarding alterations in contractility, several studies did not explicitly report these as independent outcomes but rather inferred them from previously described parameters. Although quantitative data were not always provided, impaired contractility can be inferred in certain groups, particularly in the corticosteroid-refractory group, based on reductions in LVEF.

Types of structural alterations and oedema assessed

Among the included studies, twelve did not report the presence of pericardial effusion. However, Faron *et al.*⁽¹⁰⁾ observed a significant increase in the incidence of pericardial effusion, from one patient (5%) at baseline to ten patients (45%) after the study, which was statistically significant ($p = 0.004$). Boussoar *et al.*⁽¹⁵⁾ and Ederhy *et al.*⁽¹⁸⁾ reported minimal or mild pericardial effusion. Tong *et al.*⁽¹⁶⁾ identified pericardial effusion in five patients, classified as trivial to moderate. Hichem *et al.*⁽¹⁹⁾ and Gallegos *et al.*⁽²¹⁾ reported the presence of pericardial effusion either before or after their respective studies. In contrast, Sato *et al.*⁽²⁰⁾ and Solano *et al.*⁽²⁷⁾ did not detect pericardial effusion.

Table 1. List of included studies.

N.º	Author	Year	Country	Objetivo
1	Wang <i>et al.</i> ⁽⁹⁾	2024	Germany	To generate knowledge on pathogenesis and evaluate outcomes in oncology centres using intensified management of patients with ICI-M.
2	Faron <i>et al.</i> ⁽¹⁰⁾	2021	Germany	To explore subclinical cardiac effects related to ICI therapy, specifically inflammation, fibrosis, and systolic dysfunction, using CMR.
3	Cadour <i>et al.</i> ⁽¹¹⁾	2022	France	To analyse CMR characteristics in ICI-M and explore their prognostic value for MACE.
4	Giblin <i>et al.</i> ⁽¹²⁾	2021	Ireland	To investigate the incidence, management, and prognosis of subclinical myocarditis associated with combined ICI therapy in patients with metastatic melanoma.
5	Lerchner <i>et al.</i> ⁽¹³⁾	2025	Germany	To evaluate the role of CMR in assessing ICI-M in a real-world clinical setting.
6	Zhang <i>et al.</i> ⁽¹⁴⁾	2020	USA	To characterise CMR findings in patients with ICI-M and assess their association with MACE.
7	Boussouar <i>et al.</i> ⁽¹⁵⁾	2025	Italy	To monitor ICI-related myotoxicity using a comprehensive CMR-based approach.
8	Tong <i>et al.</i> ⁽¹⁶⁾	2024	United Kingdom	To determine concordance between CMR and 18F-FDG-PET in patients with suspected ICI-M.
9	Higgins <i>et al.</i> ⁽¹⁷⁾	2021	Brazil	To evaluate the value of CMR in patients with suspected ICI-induced cardiotoxicity, particularly in detecting myocarditis.
10	Ederhy <i>et al.</i> ⁽¹⁸⁾	2020	France	To evaluate the diagnosis of ICI-M in patients with normal CMR findings, highlighting the importance of EMB for confirmation.
11	Hichem <i>et al.</i> ⁽¹⁹⁾	2024	France	To illustrate diagnostic and management challenges of ICI-M, particularly in atypical presentations mimicking other cardiomyopathies such as infiltrative cardiomyopathy.
12	Sato <i>et al.</i> ⁽²⁰⁾	2020	Japan	To monitor the evolution of ICI-M using CMR, assessing longitudinal changes in myocardial tissue and their correlation with histological and clinical findings.
13	Gallegos <i>et al.</i> ⁽²¹⁾	2019	USA	To describe a case of ICI-M (nivolumab and ipilimumab) and correlate CMR findings with post-mortem histopathology to highlight the potential role of CMR in early diagnosis.
14	Ko <i>et al.</i> ⁽²²⁾	2024	USA	To describe an atypical case of ICI-M (e.g., pembrolizumab) and its diagnosis and management.
15	Arponen & Skytta ⁽²³⁾	2020	Japan	To investigate the incidence of myocarditis in patients receiving combined nivolumab and ipilimumab as first-line treatment for advanced renal cell carcinoma.
16	Thavendiranathan <i>et al.</i> ⁽²⁴⁾	2021	USA	To evaluate the diagnostic and prognostic value of CMR using T1 and T2 mapping in patients with ICI-M.
17	Treiber <i>et al.</i> ⁽²⁵⁾	2023	Germany	To investigate whether regional T1, T2, and ECV values within LGE-positive regions improve differentiation between cardiac sarcoidosis and myocarditis compared with global values.
18	Li <i>et al.</i> ⁽²⁶⁾	2024	China	To evaluate imaging characteristics of corticosteroid-sensitive and corticosteroid-refractory ICI-M using CMR and identify parameters for early detection of refractory cases.
19	Solano <i>et al.</i> ⁽²⁷⁾	2019	Spain	To describe CMR findings in a series of patients diagnosed with immune-mediated myocarditis associated with ICIs.
20	Zhao <i>et al.</i> ⁽²⁸⁾	2022	China	To evaluate quantitative CMR parameters in detecting myocardial injury associated with ICI-induced myocarditis.

CMR: cardiac magnetic resonance. ICI: immune checkpoint inhibitors. MACE: major adverse cardiovascular events. FDG-PET: fluorodeoxyglucose positron emission tomography. EMB: endomyocardial biopsy. ECV: extracellular volume. ICI-M: immune checkpoint inhibitor-associated myocarditis. T1: longitudinal relaxation time. T2: transverse relaxation time.

Regarding myocardial oedema, nine studies did not evaluate this parameter, reported no evidence, or described its absence. Ederhy *et al.*⁽¹⁸⁾ and Solano *et al.*⁽²⁷⁾ reported normal findings. Gallegos *et al.*⁽²¹⁾ were unable to assess this variable due to the patient's inability to perform breath-holding during imaging. Wang *et al.*⁽⁹⁾ reported the presence of myocardial oedema, as did Lerchner *et al.*⁽¹³⁾, although the latter described it as rare and not associated with ICI-M. Hichem *et al.*⁽¹⁹⁾ reported diffuse myocardial oedema.

Positive findings of myocardial oedema were reported by Zhang *et al.*⁽¹⁴⁾ in 28% of patients and by Tong *et al.*⁽¹⁶⁾ in three patients (25%). Higgins *et al.*⁽¹⁷⁾ assessed myocardial oedema using the T2 myocardial-to-skeletal muscle ratio, considering a value ≥ 2 as positive. Thavendiranathan *et al.*⁽²⁴⁾ applied the modified Lake Louise criteria, finding that 53% of patients met criteria for myocardial oedema. Zhao *et al.*⁽²⁸⁾ evaluated oedema using T2-weighted imaging and an oedema ratio (ER ≥ 2.0), reporting abnormal findings in 52% of patients based on STIR sequences.

T2 mapping was not reported in eight studies. Among those that assessed it, findings showed considerable variability depending on study conditions and methodological parameters (Table 2).

With respect to fibrosis, four studies did not identify macroscopic fibrosis. Three studies reported its presence without providing quantitative data^(9,21,25). Giblin *et al.*⁽¹²⁾ highlighted its absence, while Ederhy *et al.*⁽¹⁸⁾ reported non-

significant findings. Other studies reported more relevant findings, which are detailed in Table 3.

Regarding ECV, thirteen studies did not report this parameter, suggesting it is not widely assessed or reported. Nevertheless, ECV is relevant for understanding myocardial tissue composition and detecting conditions such as fibrosis.

Among studies that evaluated ECV, findings were heterogeneous. Cadour *et al.*⁽¹¹⁾ reported values of 2.59 ± 1.97 in one group and 1.34 ± 0.57 in another ($p = 0.03$). Boussouar *et al.*⁽¹⁵⁾ reported values of 29% and 24% in their respective groups. Ederhy *et al.*⁽¹⁸⁾ reported an ECV of 26% derived from native T1 values and haematocrit. Hichem *et al.*⁽¹⁹⁾ did not provide specific values but reported a generalised increase in 45-50% of patients. Treiber *et al.*⁽²⁵⁾ focused on regions with LGE without reporting quantitative ECV values. Solano *et al.*⁽²⁷⁾ reported normal ECV in one patient, increased values in another, and segmental increases in a third. Zhao *et al.*⁽²⁸⁾ reported a mean ECV of $30.26 \pm 5.01\%$, which was elevated compared with normal values.

Among the studies analysed, 40% did not report results using T1 mapping. This lack of reporting in a substantial proportion of studies limits understanding of its potential diagnostic value. However, when applied, T1 mapping proved to be a valuable tool for myocardial tissue characterisation, enabling detection of fibrosis and microstructural alterations. Studies with significant findings are summarised in Table 4.

Overall, 20% of studies did not report macroscopic fibrosis. The absence of such reporting may indicate that it

Table 2. T2-mapping results by author.

Author	T2-Mapping	Result
Faron <i>et al.</i> ⁽¹⁰⁾	54 ± 3 ms (baseline); 58 ± 4 ms (post); $p < 0.001$	Significant increase.
Cadour <i>et al.</i> ⁽¹¹⁾	0.88 ± 1.96 (comparison); pre-ICI (-0.76 ± 1.41); $p = 0.002$	Significant differences.
Boussouar <i>et al.</i> ⁽¹⁵⁾	T2 = 56 ms. T2 = 47 ms (complete oedema resolution).	Moderate and diffuse values (local reference T2 = 48 ± 5 ms).
Tong <i>et al.</i> ⁽¹⁶⁾	Mean T2 = 54 ± 5 ms (<55 ms)	Normal values.
Higgins <i>et al.</i> ⁽¹⁷⁾	T2 >52 ms	Any value >52 ms considered abnormal.
Ederhy <i>et al.</i> ⁽¹⁸⁾	Global T2: 42±3 ms. Regional T2 (lateral wall): 41±2 ms. Regional T2 (septal wall): 43±1 ms	Normal values.
Hichem <i>et al.</i> ⁽¹⁹⁾	Borderline values around 57 ms in the septum.	Increased T2.
Sato <i>et al.</i> ⁽²⁰⁾	79 ms (normal value: 46±4 ms). 70 ms (persistent elevation).	Elevated values.
Thavendiranathan <i>et al.</i> ⁽²⁴⁾	T2 values were 56.2 ± 4.9 ms compared with reference values of 49.8 ± 2.2 ms ($p < 0.001$).	Abnormal in 43% of evaluated patients.
Treiber <i>et al.</i> ⁽²⁵⁾	Global values.	No difference.
Solano <i>et al.</i> ⁽²⁷⁾	Not specified.	Normal, elevated, and segmental elevation.
Zhao <i>et al.</i> ⁽²⁸⁾	Reference normal value: 50.98 ± 1.31 ms. Mean value in patients: not numerically specified, but elevated in 92% of cases. Abnormality rate: 92%, significantly higher than other qualitative methods such as ER and LGE	Significantly elevated in patients with ICI-M.

ICI: immune checkpoint inhibitors. ER: oedema ratio. LGE: late gadolinium enhancement. ICI-M: immune checkpoint inhibitor-associated myocarditis. T2: transverse relaxation time.

Table 3. LGE findings by author.

Author	LGE
Faron <i>et al.</i> ⁽¹⁰⁾	Not available (baseline); 2 (9%) post-treatment
Cadour <i>et al.</i> ⁽¹¹⁾	82% (ICI-M); pre-ICI 10%; p = 0.001
Lerchner <i>et al.</i> ⁽¹³⁾	ICI-M (56.0%); 26.7% in non-M; p = 0.03
Zhang <i>et al.</i> ⁽¹⁴⁾	Present in 48% of patients. subendocardial/transmural, subepicardial, mid-myocardial, and diffuse patterns.
Tong <i>et al.</i> ⁽¹⁶⁾	Present in eight patients (67%); patterns: mid-myocardial (4/8), subendocardial (3/8), subepicardial (1/8).
Higgins <i>et al.</i> ⁽¹⁷⁾	Present in 80% of patients (16 patients): 14 non-ischaeamic pattern, 2 ischaemic; no correlation with LVEF (rs = -0.29, p = 0.22) or GLS (rs = 0.10, p = 0.67)
Hichem <i>et al.</i> ⁽¹⁹⁾	Not present (baseline). present post-treatment, predominantly nodular in the lateral wall.
Sato <i>et al.</i> ⁽²⁰⁾	Patchy involvement in inferolateral wall. Diffuse subendocardial fibrosis.
Li <i>et al.</i> ⁽²⁶⁾	Present in 22 patients (62.86%) with ICI-M; no significant differences between CS and CR groups.
Solano <i>et al.</i> ⁽²⁷⁾	Equivocal, normal, normal. Presence: detected in 68% of patients. Mean extent: 10.38 ± 9.64%. Main patterns: Mid-myocardial (27%). Subepicardial (25%). Diffuse (9%). No subendocardial pattern detected. Correlation: Positive correlation with native T1 value (r = 0.28, p<0.04). Positive correlation with ECV (r = 0.50, p=0.001).
Zhao <i>et al.</i> ⁽²⁸⁾	

LGE: late gadolinium enhancement. ICI: immune checkpoint inhibitors. ICI-M: immune checkpoint inhibitor-associated myocarditis. LVEF: left ventricular ejection fraction. GLS: global longitudinal strain. ECV: extracellular volume. T1: longitudinal relaxation time

was not consistently assessed or that it was not detected in the populations studied.

Most sensitive parameters and prognostic utility

Among the evaluated parameters, GLS and native T1 mapping emerged as the most sensitive for the early detection of myocardial dysfunction. GLS abnormalities were identified even in the presence of preserved LVEF, as reported by Faron *et al.* ⁽¹⁰⁾ and Higgins *et al.* ⁽¹⁷⁾, suggesting earlier detection of functional impairment. Native T1 mapping, in turn, was associated with microstructural myocardial changes and showed a high prevalence of abnormal findings, demonstrating greater sensitivity compared with traditional parameters such as LGE or T2-based oedema assessment.

Parameters such as LGE, T1 mapping, and ECV have shown associations with major adverse cardiovascular events (MACE) and therefore possess significant prognostic value. Thavendiranathan *et al.* ⁽²⁴⁾ demonstrated that elevated native T1 was associated with increased cardiovascular risk, with an area under the curve (AUC) of 0.91 for predicting MACE. Additionally, the extent of LGE correlated with fibrosis and ECV, indicating its utility as a marker of irreversible structural damage. In contrast, some parameters, such as right ventricular GLS, did not demonstrate independent prognostic value after adjustment for other factors ⁽²⁹⁾.

Discussion

ICIs have revolutionised cancer treatment; however, they are associated with immune-related toxicities, including myocarditis, characterised by infiltration of T cells (CD3+, CD4+, CD8+) and macrophages (CD68+). Diagnosis remains challenging and is based on clinical presentation, electrocardiography, biomarkers, and imaging. Endomyocardial biopsy is essential, whereas CMR has certain limitations. Nevertheless, medical imaging plays a key role in improving the management of ICI-M, encompassing diagnosis, prognosis, and therapeutic decision-making ⁽³⁰⁾.

In this study, LVEF was one of the most frequently assessed parameters across the reviewed studies. In 45% of cases, normal values (>50%) were reported, whereas 15% of studies documented heart failure with LVEF <50%. Additionally, 10% reported mild reductions in LVEF, although still above the critical threshold of 50%. These findings indicate that LVEF remains an essential indicator for evaluating ventricular function and classifying heart failure in patients with ICI-M. However, variability in its assessment and reporting may limit comparability across studies.

Table 4. T1-mapping results by author.

Author	T1-Mapping
Faron <i>et al.</i> ⁽¹⁰⁾	972 ± 26 ms (baseline); 1006 ± 36 ms (post); p<0.001 (significant difference)
Cadour <i>et al.</i> ⁽¹¹⁾	1.79 ± 1.93 (baseline); 0.03 ± 0.85 (post); p=0.001 (significant difference)
Boussouar <i>et al.</i> ⁽¹⁵⁾	985 ms (normal value)
Tong <i>et al.</i> ⁽¹⁶⁾	Myocardial T1 values between CMR-negative and CMR-positive cases (1065 ± 19 ms vs. 1086 ± 19 ms; p = 0.03) (significant difference)
Ederhy <i>et al.</i> ⁽¹⁸⁾	Global native T1: 1296 ± 34 ms; lateral wall: 1313 ± 46 ms; Regional native T1 (septal wall): 1311 ± 32 ms. Normal values.
Hichem <i>et al.</i> ⁽¹⁹⁾	No T1 elevation (baseline). Increased T1 (post-treatment).
Sato <i>et al.</i> ⁽²⁰⁾	1505 ms (normal: 1314 ± 29 ms). 1431 ms (post-treatment). Partial improvement.
Thavendiranathan <i>et al.</i> ⁽²⁴⁾	Native T1 values were 1079.0 ± 55.5 ms compared with reference values of 1000.3 ± 22.1 ms (p<0.001). They showed excellent discriminatory value for MACE (AUC = 0.91; 95% CI: 0.84–0.98). Abnormal T1 values in 78% of patients.
Treiber <i>et al.</i> ⁽²⁵⁾	Regional (in LGE-positive areas) Increased values.
Solano <i>et al.</i> ⁽²⁷⁾	Normal, elevated, and segmental elevation.
Zhao <i>et al.</i> ⁽²⁸⁾	Reference normal value: 1020.37±17.21 ms. Mean value in patients. Abnormality rate: detected as abnormal in 73% of patients. Significantly elevated.

CMR: cardiac magnetic resonance. MACE: major adverse cardiovascular events. AUC: area under the curve. CI: confidence Interval. LGE: late gadolinium enhancement. T1: longitudinal relaxation time.

LGE was reported in several studies as a key tool for identifying macroscopic fibrosis. Higgins *et al.* ⁽¹⁷⁾ identified LGE in 80% of patients, predominantly with non-ischaeamic patterns. Additionally, Zhao *et al.* ⁽²⁸⁾ reported positive correlations between LGE and parameters such as ECV, reinforcing its utility for assessing structural damage. With respect to ECV, although not all studies evaluated this parameter, those that did reported elevated values in patients with ICI-M, including a mean value of 30.26%, significantly higher than normal values, consistent with diffuse fibrosis.

The pattern of LGE described in the reviewed studies was predominantly non-ischaeamic, characterised by subepicardial or mid-myocardial distribution and non-territorial involvement, consistent with the inflammatory aetiology of ICI-M. Arcari *et al.* ⁽⁸⁾ evaluated the role of CMR in ICI-M and found that, although LVEF is preserved in many cases, the prevalence of oedema and fibrosis detected by LGE is lower than expected. However, advanced techniques such as T1 and T2 mapping provide more precise assessment, with native T1 mapping showing the highest concordance with endomyocardial biopsy and significant prognostic value.

Benz *et al.* ⁽³¹⁾ analysed the role of CMR biomarkers as surrogate endpoints in clinical trials of myocardial disease. CMR allows accurate monitoring of disease activity and

quantification of response to novel therapies through assessment of cardiac volumes, mass, and function, as well as myocardial tissue characterisation. Despite its advantages, access to CMR remains limited, and susceptibility to artefacts requires rigorous standardisation in clinical trials. Although CMR biomarkers are promising, they do not replace the need for adequate sample sizes to evaluate clinical efficacy and safety, nor do they fully meet regulatory requirements for the approval of new treatments.

Popa *et al.* ⁽³²⁾ reported that the most significant predictors of MACE are LGE, myocardial oedema, and prolonged native T1. LGE does not necessarily represent established fibrosis in the presence of oedema and may resolve over time. LGE without oedema may indicate fibrosis, whereas persistent oedema reflects ongoing inflammation and may be associated with the potential for recovery. CMR should be performed in all patients with acute myocarditis at 6 months to assess progression and prognosis.

Díaz-Navarro *et al.* ⁽³³⁾ evaluated CMR findings in patients with acute myocarditis initially diagnosed as ST-segment elevation myocardial infarction. Their results demonstrated myocardial oedema and LGE in all patients. Oedema involved nearly half of the left ventricular mass, whereas LGE was less extensive. These findings highlight CMR as a key diagnostic

tool for differentiating acute myocarditis from myocardial infarction, particularly when myocarditis mimics infarction.

The presence of myocardial oedema was variable across studies. While some reported absence or normal findings, others documented significant rates of oedema using techniques such as T2 mapping and modified Lake Louise criteria. For example, Zhao *et al.* ⁽²⁸⁾ reported elevated oedema indices in 52% of patients, indicating a high prevalence in ICI-M. Regarding pericardial effusion, although not common, some studies, such as Faron *et al.* ⁽¹⁰⁾, reported a significant increase following ICI treatment (from one patient at baseline to ten patients post-treatment; $p = 0.004$), suggesting a potential association between ICIs and pericardial inflammatory changes.

With respect to T2 mapping, Faron *et al.* ⁽¹⁰⁾ reported a significant increase in mean T2 values following treatment (from 54 ms to 58 ms; $p < 0.001$). Similarly, T1 mapping proved to be a valuable tool for detecting microscopic fibrosis and stratifying the risk of MACE. Thavendiranathan *et al.* ⁽²⁴⁾ reported abnormal T1 values in 78% of evaluated patients.

Brendel *et al.* ⁽³⁴⁾ assessed the ability of CMR to detect active inflammation and differentiate chronic from healed myocarditis. Their findings showed that T2 mapping was the most accurate parameter for identifying chronic myocarditis, particularly when abnormal in three or more segments. Combining T2 mapping with biomarkers such as troponin or C-reactive protein improved specificity. Multiparametric CMR demonstrated greater accuracy in distinguishing chronic from healed myocarditis compared with T1 mapping, ECV fraction, or LGE alone.

Bernhard *et al.* ⁽²⁹⁾, in a cohort of patients with suspected myocarditis, found that abnormal right ventricular GLS (RV GLS) was present in 21.9% of cases. Although RV GLS impairment was associated with MACE, this association was no longer significant after adjustment for right ventricular ejection fraction, LVEF, left ventricular GLS, and LGE extent. These findings suggest that RV GLS has limited prognostic value, as it does not provide independent information once ventricular function and tissue characteristics are considered.

Ferreira *et al.* ⁽⁵⁾ reported that CMR provides robust evidence of myocardial inflammation when myocardial oedema is demonstrated in combination with other markers of inflammatory myocardial injury. This is based on at least one T2-based criterion together with at least one T1-based criterion, an approach aimed at improving diagnostic accuracy in the detection of myocardial inflammation.

Kirkpatrick *et al.* ⁽³⁵⁾ highlighted the evolution and increasing importance of CMR in the diagnosis and management of myocarditis, particularly in the context of the SARS-CoV-2 pandemic and COVID-19 vaccination. Initially, CMR faced challenges in establishing its role due to

factors such as professional boundaries between radiology and cardiology, reimbursement issues, and competition from cardiac computed tomography. However, technological advances, including accelerated imaging sequences and T1 and T2 mapping techniques, have improved efficiency and tissue characterisation, expanding its clinical applicability. CMR is now considered an essential tool for evaluating patients with non-ischaemic cardiomyopathies and chest pain in the absence of coronary artery disease.

GLS was assessed in only 25% of the included studies, reflecting limited adoption of this parameter in the reviewed literature. Nevertheless, available evidence suggests that GLS abnormalities may precede reductions in LVEF, supporting its role as an early marker of myocardial dysfunction. Faron *et al.* ⁽¹⁰⁾ reported a significant decrease in GLS following intervention (from -23.4% to -19.6%, $p = 0.005$), while Hichem *et al.* ⁽¹⁹⁾ identified specific patterns such as apical sparing. The subepicardial strain impairment observed in some cases of myocarditis may be explained by the underlying pathophysiology of the inflammatory process. In myocarditis, inflammation typically begins in the epicardium and subsequently extends towards the mid-myocardial layers, initially sparing the subendocardium. These findings underscore the importance of GLS for the early detection of cardiotoxicity.

In conclusion, the reviewed studies highlight the value of CMR as an advanced tool for assessing cardiovascular complications associated with ICIs. Parameters such as LVEF, GLS, LGE, ECV, and native T1 mapping provide essential information on ventricular function, structural alterations, and the risk of major adverse events. However, the limited implementation of sensitive techniques such as GLS and T1/T2 mapping reflects a technological and training gap across many clinical centres.

Imaging biomarkers face several limitations in clinical application. The lack of standardisation in the acquisition and interpretation of GLS, T1/T2 mapping, and ECV values limits comparability across centres and studies. In addition, limited access to high-resolution CMR and its susceptibility to artefacts ⁽³¹⁾ represent barriers to routine use. Therefore, the development of standardised protocols and improved accessibility to these techniques are essential to enable broader and more reliable implementation in clinical practice.

This scoping review has several limitations. The available evidence was heterogeneous in terms of study design, diagnostic criteria, and CMR protocols, which limits comparability across studies. As a scoping review, no formal assessment of methodological quality was performed; therefore, findings should be interpreted as a descriptive mapping of the current state of knowledge rather than a critical synthesis. The search was restricted to articles in English

and Spanish, which may introduce language bias, and grey literature was not comprehensively included. Finally, given the rapid evolution of this field, recent studies may not have been captured in the latest search.

Future research should focus on validating consensus-based diagnostic approaches, promoting the incorporation of these parameters into international clinical guidelines, and establishing native T1 mapping as a key biomarker in

the follow-up of oncology patients receiving ICIs. In addition, prospective studies and controlled clinical trials are needed to establish robust causal associations between ICI therapy and myocardial alterations detected by multiparametric CMR.

Author contributions

EQS, KGM: conceptualisation, methodology, validation, formal analysis, investigation, writing, review. **NAA, LCD:** eview, editing, and supervision.

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