

## INVITED REVIEW

# Cardiac Magnetic Resonance Imaging in Immune Checkpoint Inhibitor-Related Myocarditis

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## ABSTRACT

Immune checkpoint inhibitors (ICIs) have revolutionized oncologic treatment, offering a novel and effective approach to personalized cancer immunotherapy. By targeting immune tolerance pathways, ICIs enhance T-cell-mediated tumor cytotoxicity. Despite their therapeutic efficacy, ICIs are associated with immune-related adverse events (irAEs), including severe cardiovascular complications like myocarditis, pericarditis, and vasculitis. ICI-related myocarditis, although uncommon, carries a high mortality rate of up to 50%, particularly in patients receiving combination therapies. This review examines the mechanisms of ICIs, highlights the clinical presentation of cardiac irAEs, and underscores the utility of cardiac magnetic resonance imaging (CMR) in the diagnosis of ICI-associated myocarditis. Through case studies, we illustrate the diagnostic and therapeutic strategies for ICI-related cardiac complications, highlighting the importance of multidisciplinary collaboration in mitigating morbidity and mortality.

## 1 | Introduction

Immune checkpoint inhibitors (ICIs) are antibody and receptor-based immune modulators, which represent a novel approach to personalized cancer treatment. These agents have rapidly become the standard of care for cancer immunotherapy and comprise an exciting frontier for current and future research endeavors. With the rapidly increasing prevalence of ICI prescription, there has been a concordant rise in immune-related adverse events (irAEs). In this review, we will summarize the mechanisms of action and uses of ICIs, associated cardiac irAEs, and the utilization of cardiac magnetic resonance imaging (CMR) for patients with treatment-related myocarditis.

### 1.1 | ICI Background

ICIs represent a growing field of molecular-based modulation of immune tolerance in the context of malignancy. Immune

checkpoints are a group of cell-membrane proteins that facilitate bonding between healthy cells and circulating immune cells within the innate (e.g., natural killer cells) and adaptive (e.g., B and T cells) pathways [1]. These protein pairs promote immune tolerance by downregulating T-cell activation. Common pathways include PD-1, PD-L1, CTLA-4, and LAG-3 [2]. Tumor cells, especially those with a high mitotic rate, skillfully exploit these pathways by upregulating PD-L1 expression, binding to PD-1, and thus downregulating T-cell activation [1]. The production of PD-L1 is enhanced in inflammatory settings involving interferon gamma, thus promoting tumor propagation [1].

The first FDA-approved ICI in the United States was ipilimumab, a CTLA-4 inhibitor, which was authorized in 2011 for metastatic melanoma [3]. Since then, 11 ICIs have been approved for over 20 cancer types as of January 2024, including hematologic malignancies and solid tumors [4]. The most common indications for ICI treatment include melanoma and non-small cell lung

cancer, with pembrolizumab and nivolumab, antibodies to the T-cell PD-1 receptor, representing the most prescribed medications [5, 6]. These immune modulators inhibit PD-L1 binding, thus promoting T-cell activation and tumor cell cytotoxicity. As tumor cells express higher levels of PD-L1, they are more likely to bind to the medication, causing a localized effect. However, as a majority of healthy cells also produce PD-L1, these medications can lead to a variety of irAEs.

## 1.2 | Cardiac Immune-Related Adverse Events

ICIs have been associated with a wide variety of irAEs, with particularly severe reactions involving the cardiovascular system. Examples include myocarditis, pericardial disease, and vasculitis. Major risk factors for irAEs are combination ICI therapy, which involves treatment with both CTLA-4 and PD-1 inhibition, and high-dose monotherapy [7, 8].

The reported incidence of myocarditis in individuals treated with ICIs is 0.3%–1.1%, with a 4.7 times higher chance of developing and experiencing a more severe event after combination immunotherapy [5, 7]. Fatal outcomes were noted in 50% of patients, again more frequent with combination immunotherapy [9]. In contrast, approximately 75% of patients with non-ICI-related myocarditis experience an uncomplicated hospital course, and among the other 25%, there was a maximum fatality rate of 28% [10].

Pericardial disease includes pericarditis, pericardial effusion, and myopericarditis. The reported incidence is approximately 1.57 events per 100 person years, with a 21.1% fatality rate [8, 9, 11]. Vasculitis was seen in less than 1% of patients, with a fatality rate of approximately 6.1% [9]. Giant cell arteritis was the dominant diagnosis, with one study reporting vision loss in 28% of affected patients [12]. For myocarditis and pericardial disease, the median time to onset was approximately 30 days (after 1–2 cycles of ICI therapy), while for vasculitis, the median time to onset was approximately 60 days (after three cycles) [9].

## 2 | Diagnosis of ICI Myocarditis

### 2.1 | Clinical Background and Presentation

Clinical manifestations of ICI-related myocarditis typically appear early in the treatment course, often within the first few weeks of therapy, although clinical presentation is variable. While other forms of myocarditis may follow a more protracted course, ICI-related myocarditis often progresses with marked severity, demanding heightened clinical vigilance and prompt intervention. Notably, ICI-related myocarditis can only be diagnosed after excluding all other possible etiologies of myocardial injury, including ischemia and myocardial oxygen supply and demand mismatch [5, 7, 13]. The clinical presentation of this condition ranges from entirely asymptomatic to life-threatening cardiac compromise. Moreover, ICI myocarditis is often associated with myositis and myasthenia gravis as an overlap syndrome, further complicating its clinical presentation. The American Society of Clinical Oncology (ASCO) clinical practice guidelines for irAEs

propose a classification system delineating this spectrum of severity (Figure 1) [14].

Asymptomatic patients (Grade 1) may display only subtle abnormalities, such as mild elevations in cardiac biomarkers or electrocardiographic changes, without overt clinical symptoms. Progression to Grade 2 disease is often marked by mild but more noticeable cardiac symptoms (e.g., syncope, exertional dyspnea, chest discomfort present in approximately 90% of patients), accompanied by abnormal biomarkers and ECG findings [10]. These include conduction disease (PR interval prolongation, second- and third-degree AV block), and ventricular ectopy (including nonsustained and sustained ventricular tachycardia). Patients in this category may also present with nonspecific complaints that can easily be attributed to other causes, emphasizing the need for a high index of suspicion and routine cardiac surveillance in patients receiving ICIs [7, 13–15].

More pronounced clinical and imaging abnormalities define moderate (Grade 3) disease, often featuring left ventricular systolic dysfunction (LVEF < 50%) or regional wall motion abnormalities (WMAs) detected on transthoracic echocardiography. CMR findings at this stage may be diagnostic or highly suggestive of myocarditis, demonstrating myocardial edema, hyperemia, or late-gadolinium enhancement (LGE), which are indicative of inflammatory infiltration. As severity escalates to Grade 4, patients may experience fulminant, life-threatening disease with cardiogenic shock, or severe arrhythmias [7, 13–15].

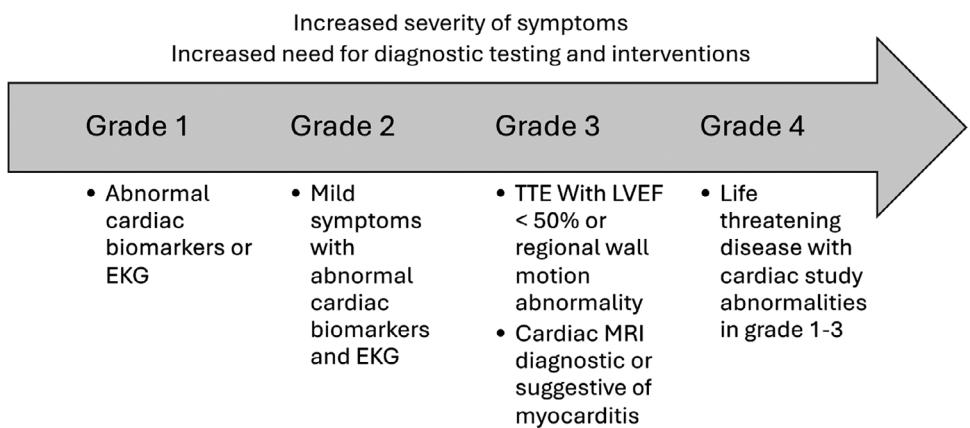
Diagnostic evaluation is multifactorial, involving the clinical picture, biomarker assessment, and imaging modalities. Even a modest elevation in troponin may serve as an early indicator of active disease. While echocardiography may reveal left ventricular systolic dysfunction or regional WMAs, it is often normal, especially in mild cases. Consequently, a presumptive diagnosis based on clinical and noninvasive findings frequently guides therapy [5, 13, 16].

### 2.2 | Clinical Imaging

Nonischemic cardiomyopathy (NICM) is typically classified into the following five groups: hypertrophic, restrictive/infiltrative, dilated/unclassified, arrhythmogenic, and inflammatory (expert panel, 2021) [17]. ICI-related NICM is a subset of inflammatory NICM. Clinical presentation varies, but myocarditis remains a key clinical concern among patients on ICIs given its association with significant fatality rates. The gold standard for diagnosis of myocarditis is endomyocardial biopsy and CMR. CMR is preferred as it is a noninvasive modality and has been found to be more sensitive for myocarditis, particularly in the early stages when echocardiogram findings may be negative [10]. Per the American College of Radiology, appropriate imaging workup for inflammatory NICM may include CMR with and without contrast in conjunction with transthoracic echocardiography.

CMR offers insight into underlying structural, functional, and myocardial tissue changes. Modern techniques utilize 1.5T or 3T scanners with electrocardiographic-gating software that allows for retrospective, prospective, and triggered gating. Required pulse sequences include cine-balanced steady-state free precess-

## ICI Related Myocarditis Severity



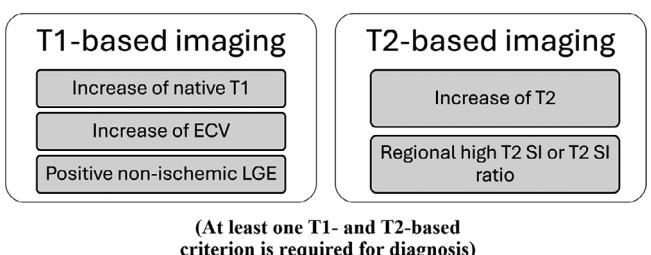
**FIGURE 1** | Proposed four-tier grading system for myocarditis severity, illustrating a stepwise increase in symptoms, diagnostic abnormalities, and need for intervention from Grade 1 through Grade 4 based on The American Society of Clinical Oncology (ASCO) guidelines on managing irAEs.

sion (bSSFP), rapid multislice myocardial perfusion imaging, LGE, phase contrast flow quantification, and 3D contrast-enhanced angiographic imaging [18].

Many etiologies for NICM can be comprehensively evaluated with CMR. Cine sequences provide information regarding wall motion and cardiac structure, while phase-contrast sequences quantitatively evaluate flow dynamics. Ejection fractions, forward flow volumes, ventricular volumes, and wall kinetics can be calculated using these sequences [18]. Tissue characterization is key in the diagnosis of myocarditis. Native T1 mapping and extracellular volume (ECV) can be used to evaluate myocardial integrity in the setting of deposition disease. Elevated values are observed with tissue deposition (e.g., amyloidosis or sarcoidosis), diffuse edema, or significant valvular disease. Conversely, decreased values are seen in the setting of hemochromatosis and fat deposition. Post-contrast sequences allow clinicians to evaluate abnormal enhancement of the myocardium. Processes that disrupt the interstitium, such as fibrosis, protein deposition, or acute myocardial injury cause abnormal accumulation of gadolinium-based intravenous contrast, which shortens the T1 time of the tissue and appears hyperintense on delayed imaging. The extent of LGE has been correlated with the development of malignant arrhythmia and heart failure (HF) [19]. T2-weighted imaging and T2 mapping can assess myocardial edema, which is a sign of acute inflammation, such as in myocarditis [20, 21].

In 2018, the JACC scientific expert panel published an update to the Lake Louise Criteria on the use of CMR with parametric mapping for patients with suspected myocarditis or inflammatory cardiac changes. This update concluded that myocarditis can be suspected when patients exhibit at least one clinical criterion (e.g., chest pain or dyspnea) and one diagnostic criterion (e.g., EKG changes, biomarker elevation, or cardiac imaging finding). For asymptomatic patients, at least two diagnostic criteria are required [22]. On CMR, the main criteria are T1- and T2-based findings (Figure 2). Additional supportive criteria include pericarditis and systolic left ventricular dysfunction. Subsequent studies found that these updated guidelines were 87.5% sensitive and 96.2% specific for the diagnosis of myocarditis [23].

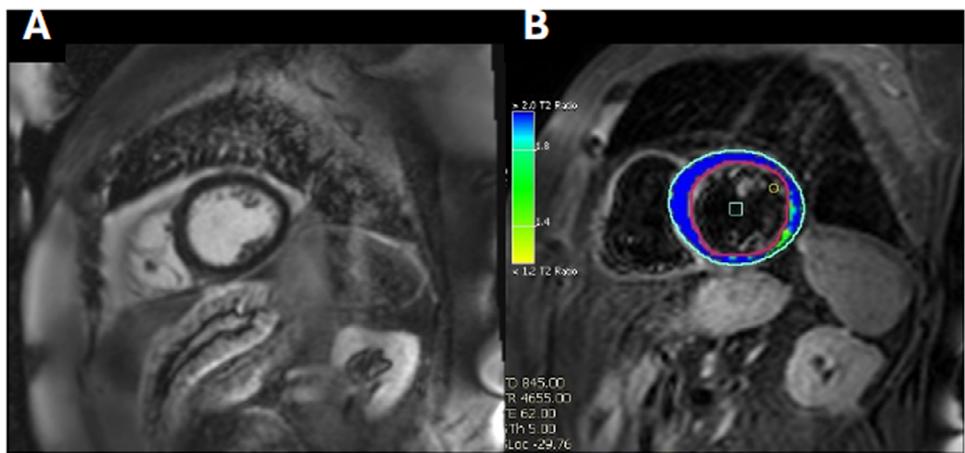
## 2018 Lake Louise Criteria for Myocarditis



**FIGURE 2** | Updated (2018) Lake Louise CMR criteria for myocarditis. T1-based indices include increased native T1, elevated extracellular volume (ECV), and nonischemic patterns of LGE, while T2-based indices include elevated T2 and regional increases in T2 signal intensity or T2 signal intensity ratio.

## 2.3 | Treatment

All patients with suspected ICI-related myocarditis, regardless of severity, should discontinue immunotherapy and undergo urgent clinical evaluation. Hospital admission for close monitoring and prompt cardio-oncology consultation is recommended. Initial management includes early administration of high-dose oral corticosteroids (1–2 mg/kg of prednisone). Transfer to a cardiac intensive care unit is recommended for patients with significant ventricular ectopy, conduction abnormalities, or worsening HF symptoms, given the risk of rapid clinical deterioration. In patients who do not exhibit a prompt biomarker response to standard-dose oral corticosteroids, escalation (3–5 days of IV methylprednisolone) may be warranted, accompanied by adjunctive immunosuppressive therapies such as mycophenolate, infliximab, tofacitinib, abatacept, and/or intravenous immunoglobulin. After discharge, an early evaluation of cardiac biomarkers (within 1 week) and follow-up with a cardio-oncologist (within 2–4 weeks) is recommended. In selected mild cases, following a thorough risk–benefit discussion with the patient, cardio-oncology, and oncology teams, ICI therapy may be considered for resumption [7, 13, 16].



**FIGURE 3** | (A) Short-axis post-contrast imaging shows no LGE. (B) T2 weighted imaging in the mid-left ventricle shows diffuse myocardial edema within the left ventricle. The blue coloration reflects a signal intensity that is twice that of normal skeletal muscle, consistent with diffuse myocardial edema.

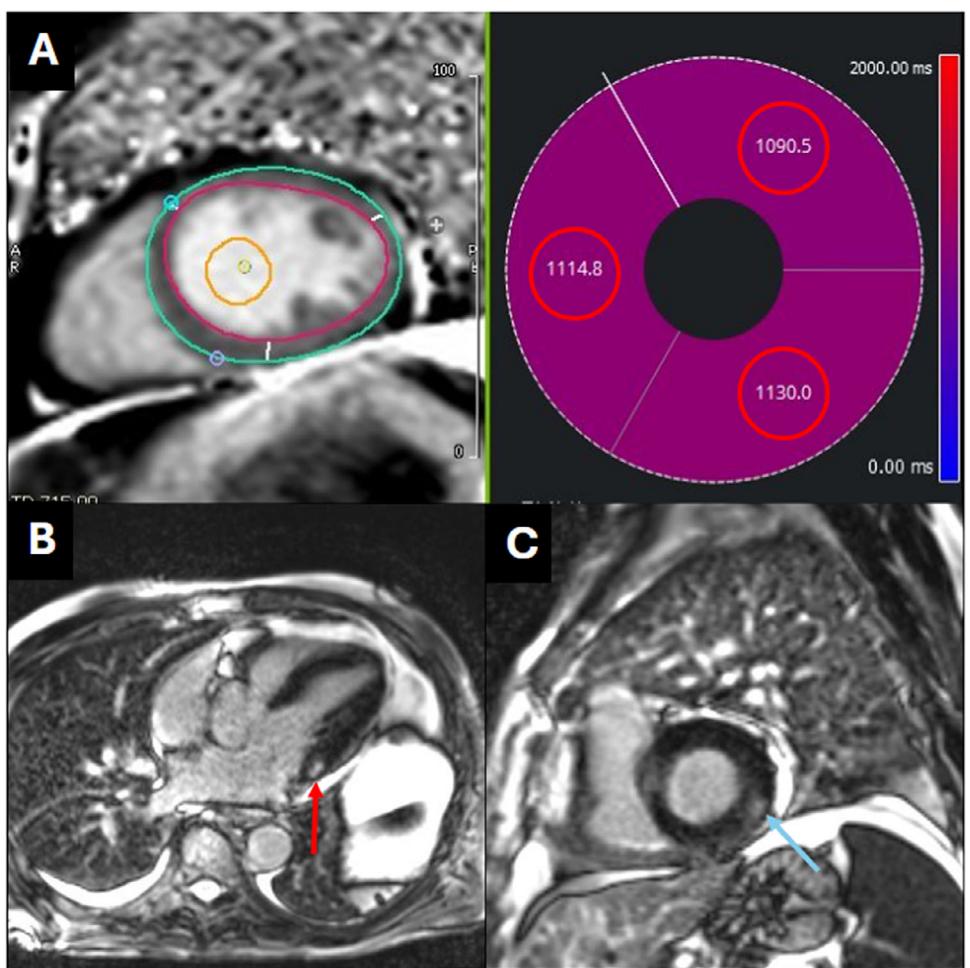
### 3 | Cases

**Case 1:** Eighty-seven-year-old patient with left cheek squamous cell carcinoma. Prior to surgical debulking, the patient received neoadjuvant immunotherapy with cemiplimab, a PD-1 ICI. Approximately 4 months after starting treatment (having completed seven rounds), the patient developed lower extremity swelling with decreasing oxygen saturation. Pertinent labs included high-sensitivity troponin T (hsTrop) of 12 (normal < 12), N-terminal pro-B-type natriuretic peptide (NT-proBNP) of 686 (normal < 450 pg/mL). EKG was consistent with atrial fibrillation, and transthoracic echocardiogram (TTE) demonstrated preserved systolic left ventricular function. Three months later, the patient was admitted with worsening left lower extremity swelling secondary to a left external iliac vein DVT extending to the popliteal vein and was found to have uptrending hsTrop with a peak of 179. EKG remained unchanged. Repeat echocardiography demonstrated hypokinesis of the mid to basal inferior wall and septum with mildly decreased LVEF of 52% and a moderate diastolic dysfunction. CMR showed mildly decreased LVEF and diffuse T2 signal hyperintensity consistent with acute myocardial edema (Figure 3). The absence of LGE likely reflected the timing of imaging relative to the onset of myocarditis, with scar formation often requiring a longer timeframe before manifestation on imaging. After a diagnosis of ICI-related myocarditis, the immunotherapy was discontinued, and a tapering regimen of prednisone was initiated. Three months after discharge, the patient's troponin levels had normalized; a repeat CMR examination demonstrated complete normalization of left ventricular systolic function as well as resolution of T2 signal abnormalities. No LGE was seen.

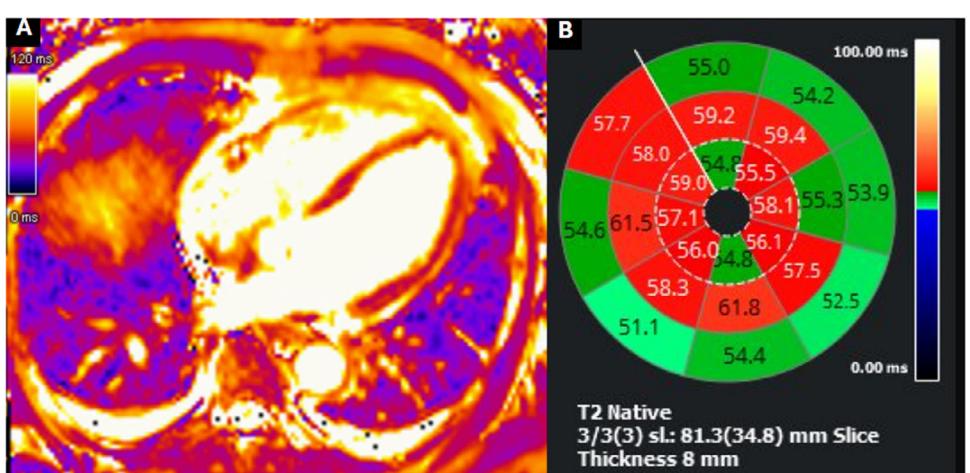
**Case 2:** Sixty-one-year-old patient with esophageal cancer with metastasis to the liver. Given the extent of the disease, the patient enrolled in a clinical trial that included treatment with FOLFOX (folinic acid, 5-fluorouracil, and oxaliplatin) and nivolumab, a PD-1 ICI. Approximately 2 months after starting immunotherapy (completing two cycles), the patient presented with shortness of breath. Labs demonstrated elevated lactic acid to 6.4, rising hsTrop peaking to 440, and elevated NT-proBNP to 4700. Initial chest x-ray showed pulmonary edema. EKG and telemetry were

negative for arrhythmias. Echocardiography showed basal and mid inferior as well as inferoseptal hypokinesis with preserved LVEF of 65%. CTA coronary showed nonobstructive coronary artery disease. CMR revealed elevated T2 and T1 values in the left ventricular mid-to-apical segments, increased ECV, and LGE in the basal-to-mid inferior and inferolateral walls—findings consistent with acute myocarditis (Figures 4 and 5). After discontinuing nivolumab, the patient was transitioned to a nonimmune-checkpoint-inhibitor chemotherapy regimen and initiated on a tapering course of prednisone. Three months after discharge, his troponin levels had normalized. On follow-up CMR, T2-weighted images revealed no significant edema or inflammation, and T1 mapping values were within normal limits; however, there was persistent, patchy, mid-myocardial, and subepicardial LGE in the basal lateral wall, similar to previous findings.

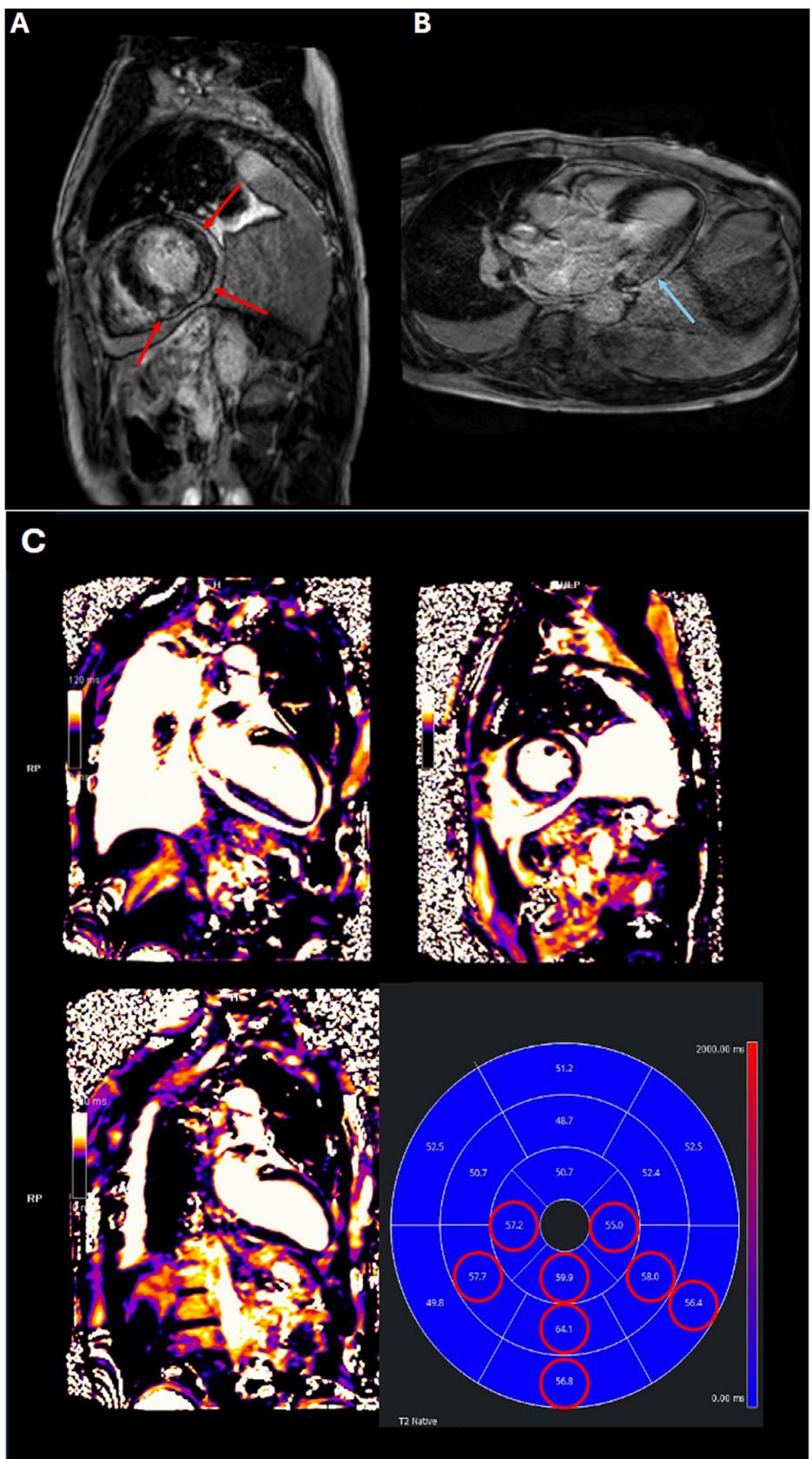
**Case 3:** Seventy-two-year-old patient with a remote history of non-Hodgkin's lymphoma status post-chest radiotherapy and renal cell carcinoma status post-partial nephrectomy. Approximately two decades after initial treatment, the patient developed dysphagia and was found to have a biopsy-proven esophageal cancer at the gastroesophageal junction. This patient was initially treated with FOLFOX, radiation therapy, and nivolumab immunotherapy. After one treatment cycle, the patient developed shortness of breath, lower extremity edema, and dyspnea on exertion. Pertinent lab values included rising hsTrop to a peak of 125 and NT-proBNP of 7200. EKG showed a new incomplete left bundle branch block. Echocardiography showed mildly reduced LVEF of 45% without WMAs and a new small pericardial effusion. CMR findings were concordant with the echocardiography results demonstrating a globally decreased LVEF and a small pericardial effusion. Additionally, there was T2 hyperintensity along the mid inferior, inferoseptal, and inferolateral segments, consistent with myocardial edema, as well as abnormal T1 mapping, ECV values and LGE in these regions (Figure 6). The constellation of findings was consistent with ICI-induced myocarditis. Consequently, the ICI regimen was discontinued, a different chemotherapy regimen was initiated, and the patient was placed on a prednisone taper. Post-discharge CMR showed marked improvement in myocardial edema and LGE, although the distribution remained similar to previous findings.



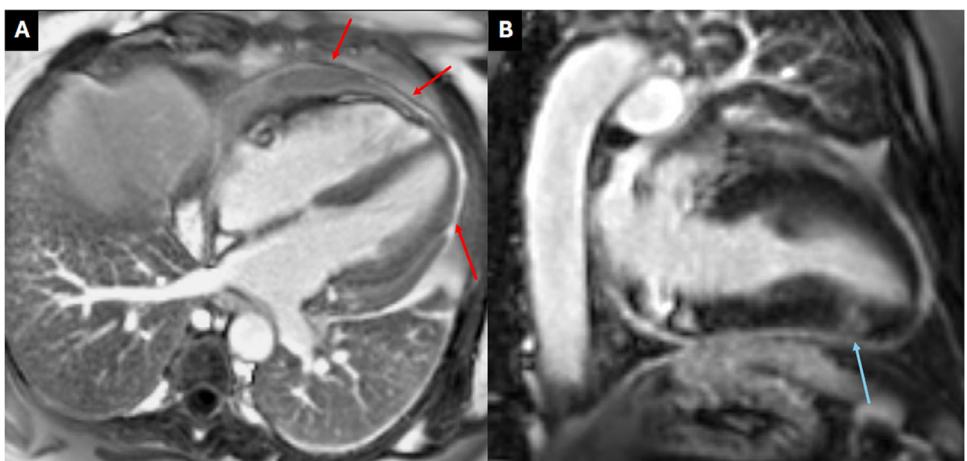
**FIGURE 4** | (A) Short axis native T1 weighted mapping of the mid myocardium shows diffusely increased T1 signal (normal range 880–1080 ms), consistent with diffuse inflammation (red circles). (B) Four-chamber view post-contrast imaging shows midmyocardial LGE within the basal inferolateral wall (red arrow). (C) Two-chamber view post-contrast imaging shows midmyocardial LGE within the basal inferolateral walls (blue arrow).



**FIGURE 5** | (A) Four-chamber view T2 imaging demonstrates increased signal intensity in the septal and apical segments, consistent with myocardial edema. (B) Short-axis view T2 mapping reveals elevated signals predominantly in the mid and apical segments, supporting the presence of myocardial edema.



**FIGURE 6** | (A) Two-chamber view post-contrast imaging shows midmyocardial LGE within the anterolateral, inferolateral, and inferior walls (red arrows). (B) Four-chamber view post-contrast imaging shows midmyocardial LGE within the basal inferolateral wall (blue arrow). (C) T2 mapping (normal < 55 ms) shows diffuse myocardial edema in the inferior segments (red circles).



**FIGURE 7** | (A) Four-chamber view, fat-saturated, T2 weighted imaging shows nodular thickening of the pericardium along the anterior right ventricle with a small pericardial effusion (red arrows). (B) Sagittal view, fat-saturated, T2 weighted imaging shows pericardial thickening with a small pericardial effusion as well as midmyocardial to epicardial LGE along the inferior mid-to-apical inferior wall (blue arrow).

**Case 4:** Sixty-three-year-old patient with a history of clear cell renal cell carcinoma, who received neoadjuvant combination immunotherapy with ipilimumab and nivolumab. After three cycles of treatment, the patient presented to the hospital with right-sided pleuritic chest pain that radiated to the back. HsTrop and pro-BNP were negative. EKG showed sinus tachycardia, and CTA chest demonstrated a small pericardial effusion. The findings were suspicious for ICI-related pericarditis. The immunotherapy was stopped, and the patient was treated with a steroid taper. A follow-up CMR with contrast, approximately 1 month after steroid initiation, showed normal LVEF with a small-to-moderate pericardial effusion. Post-contrast images revealed circumferential pericardial enhancement and midmyocardial LGE in the mid-to-apical inferior wall (Figure 7). T1 mapping, T2 mapping, and ECV were within normal limits, which may be secondary to the delayed acquisition of imaging in relation to the initiation of steroids. Although biomarkers were within normal limits at initial presentation, it was uncertain whether the case was caught early in the disease course. Thus, the differential included ICI-related pericarditis in the setting of NICM versus myopericarditis. Subsequent imaging after continued steroid administration showed significant improvement in the pericardial effusion with decreased LGE.

#### 4 | Conclusion

ICIs represent a promising and rapidly expanding approach to oncologic treatment. They are used for a wide range of solid tumors and hematologic malignancies. As their use continues to increase, clinicians must be aware of their major adverse effects, especially ones carrying substantial morbidity and mortality. Although ICI-related myocarditis occurs in only a small proportion of treated individuals, mortality rates can reach up to 50%, with higher rates of incidence in patients treated with high-dose monotherapy or combination therapy. If there is clinical suspicion of myocarditis, multiple laboratory markers and imaging modalities can be utilized, including EKG/telemetry, cardiac biomarkers, echocardiography, and CMR, which has become a mainstay of the diagnostic pathway. When CMR is

performed, it is important to include native and post-contrast T1 mapping as well as T2 mapping in addition to structural and functional analysis. Used in conjunction with echocardiography, CMR can enhance the timeliness and accuracy of diagnosis, thereby improving outcomes for ICI-related myocarditis patients through early initiation of targeted therapy.

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The authors have nothing to report.

#### Conflicts of Interest

The authors declare no conflicts of interest.

#### Data Availability Statement

No data were analyzed for or in support of this review.

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