Myocardial inflammation is an important cause of myocardial injury, which often results from the host immune response, and can be triggered by infection, autoimmune diseases, ischemic injury, or toxins. Myocarditis is a more specific term, defined as a nonischemic inflammatory disease of the myocardium. Traditionally, diagnosis has relied on histologic evaluation of the myocardium showing inflammation and myocyte damage (1). Recently, there has been heightened awareness of myocarditis due to reports of myocardial inflammation after COVID-19 illness and vaccination. Cardiac MRI plays an important role in the assessment of suspected myocarditis, as timely identification can affect patient management and prognosis (2). The aim of this review is to provide an overview of the role of cardiac MRI and typical findings in patients with nonischemic myocardial inflammation, with a focus on emerging data in the setting of acute myocarditis after COVID-19 vaccination.

Incidence and Pathophysiology of Myocarditis

The incidence of myocarditis is difficult to establish, as clinical symptoms are nonspecific, including chest pain and shortness of breath, and endomyocardial biopsy is not frequently performed for definitive diagnosis. Approximately one-third of patients presenting with acute coronary syndrome without substantial coronary artery disease are ultimately diagnosed with acute myocarditis (3).

Even across diverse causes (Table 1), myocarditis is ultimately driven by an immune response directed at cardiomyocytes. In acute myocarditis, the initial trigger is either direct myocardial injury or immune dysregulation that induces inflammation by activating an innate or adaptive immune response. Myocardial injury can manifest across a spectrum of clinical severity—from subclinical disease, to myocarditis with preserved cardiac function, to more severe cases that result in reduced systolic or diastolic function, arrhythmia, and rarely hemodynamic collapse and cardiogenic shock. In most patients, the immune response is self-limited and downregulates with clearance of the initial trigger. However, depending on the degree of myocardial injury, patients may have residual myocardial dysfunction and fibrosis. In a minority of patients, the inflammatory response can persist or recur, leading to chronic myocarditis. Most patients recover completely after acute myocarditis, but a small proportion, estimated at less than 5%, will progress to dilated cardiomyopathy due to myocardial remodeling (Fig 1) (4).

The most common trigger for myocarditis in developed countries is viral infection (5,6). Although traditional serologic studies, viral cultures, and molecular techniques can be used to identify viral pathogens in the setting of myocarditis, these techniques lack both sensitivity and specificity (7). Myocardial injury is also associated with COVID-19 illness, with elevated troponin levels in more than 60% of hospitalized patients (8). Although SARS-CoV-2 can infect cardiomyocytes by binding to the angiotensin-converting enzyme 2, indirect myocardial inflammation due to immune dysregulation may be a more prominent mechanism of myocardial injury (8).
Noninfectious causes of myocardial inflammation include autoimmune and immune-mediated disorders such as vasculitides, connective tissue disorders such as systemic lupus erythematosus, and granulomatous diseases such as giant cell myocarditis. Several drugs and medications are associated with myocarditis, including amphetamines and immune checkpoint inhibitors. Myocarditis is an uncommon adverse event after immunization (9). However, there is emerging evidence that COVID-19 vaccination is associated with myocarditis in a minority of patients.

Diagnosis

Establishing a diagnosis of acute myocarditis is important, as timely recognition can impact patient management and outcomes (2). Myocarditis is an important cause of sudden cardiac death in young adults, accounting for up to 12% of sudden cardiac death cases, according to postmortem analysis (10). Due to increased risk of sudden cardiac death, particularly when performing exercise, avoidance of competitive sports is typically recommended for at least 3 months in patients with acute myocarditis (11).

Endomyocardial biopsy is still considered the reference standard for definitive diagnosis of myocarditis; however, it is not frequently performed due to the invasive nature of the procedure and associated risks, as well as low sensitivity compared with cardiac explant at autopsy (12). Endomyocardial biopsy is usually only indicated if there is clinical evidence that the results will have a meaningful effect on therapeutic decisions (13). When endomyocardial biopsy is performed, the Dallas criteria are commonly used, which require histologic evidence of inflammatory infiltrates within the myocardium associated with myocyte damage and/or necrosis of nonischemic origin for definitive diagnosis (Fig 2) (7). Newer proposed criteria rely on immunohistochemical techniques, which may be more sensitive (14).

In clinical practice, diagnostic criteria for suspected myocarditis that are based on expert consensus are more commonly employed. Acute myocarditis is considered clinically suspected if at least one clinical criterion and at least one diagnostic criterion are met (15). Clinical criteria include acute chest pain, new onset dyspnea, palpitations, unexplained arrhythmia symptoms, syncope, aborted sudden cardiac death, and unexplained cardiogenic shock. Diagnostic criteria include electrocardiographic, Holter monitor, or stress test abnormalities; elevated troponin levels; functional and structural abnormalities at cardiac imaging; and typical tissue characterization features of edema and/or late gadolinium enhancement (LGE) at cardiac MRI. Cardiac MRI can be used to meet either of the latter two criteria, highlighting the important role of imaging for diagnosis in acute myocarditis (15). Imaging findings can also be useful in identifying or excluding other potential diagnoses that may have a similar clinical presentation, including acute coronary syndrome or stress-induced cardiomyopathy. In some circumstances, imaging findings may suggest a specific potential cause for myocardial injury, although there is substantial overlap in imaging findings between different causes of myocarditis.

Imaging Myocardial Inflammation

The American Heart Association recommends testing for patients with signs consistent with myocarditis, using one or more cardiac imaging techniques, such as echocardiography or cardiac MRI (16).

Echocardiography

Echocardiography is often the first imaging modality used in patients with suspected myocarditis, as it is widely available and allows for relatively rapid assessment of cardiac size and function. Typical findings, including increased myocardial wall thickness and echogenicity, impaired global systolic function and strain, regional wall motion abnormalities, and ventricular dilatation, are relatively nonspecific (17). However, echocardiography provides important prognostic information, as increased left ventricular (LV) size and impaired function are predictors of poor outcomes (18).

CT Imaging

Coronary CT angiography is a noninvasive imaging modality that may be useful in excluding obstructive coronary artery disease in patients presenting with acute chest pain and elevated troponin levels, due to its high negative predictive value. Late iodine enhancement may be useful in evaluating myocardial damage, particularly in patients with a contraindication to MRI, although there are limited data specifically in acute myocarditis (19).

PET Imaging

Fluorodeoxyglucose PET is well established in the evaluation of active myocardial inflammation in the setting of cardiac sarcoidosis. Limited data available demonstrate that fluoro-
Sanchez Tijmes et al recently combined PET/MRI scanners have become available, which could provide complementary information from both modalities in patients with myocarditis (21).

Deoxyglucose PET can also identify inflammation in the setting of acute myocarditis (20). PET is typically performed in conjunction with CT for anatomic localization, although more recently combined PET/MRI scanners have become available, which could provide complementary information from both modalities in patients with myocarditis (21).

| Table 1: Causes of Myocardial Inflammation and Typical MRI Findings |
|---------------------------------------------------------------|
| **Cause** | **Specific Cause or Mechanism** | **Key MRI Finding** |
| Infection | Infectious agents can induce cardiac injury by directly infecting cardiomyocytes or through cellular or humoral immune activation | Viral myocarditis: Linear subepicardial or midwall LGE, commonly involving the basal inferolateral wall, basal anterior septum, mid inferolateral wall, and basal to mid inferior wall, with corresponding T2 hyperintensity or high T2 |
| Viral: Enteroviruses, coronaviruses, adenoviruses, parvovirus B19, herpesviridae 6, CMV, EBV, HIV, influenza; SARS-CoV-2 can infect cardiomyocytes by binding to the ACE2 receptor, although immune dysregulation is likely a more prominent mechanism of myocardial injury | Chagas disease: LGE present in up to 70% of patients, most commonly at the left ventricular apex, apical inferior and lateral wall, and basal to mid inferolateral wall; LGE is usually midwall or subepicardial and less commonly subendocardial or transmural with apical aneurysms |
| Bacterial: *Borrelia burgdorferi* (Lyme disease), *Treponema pallidum*, group A Streptococcus (likely postinfectious) | Bacterial and parasitic myocarditis: Limited data on MRI findings with no specific pattern |
| Protozoal: *Trypanosoma cruzi* (Chagas disease), *Toxoplasma gondii* | COVID-19: Findings may be similar to non-COVID viral myocarditis, although some studies have indicated a higher prevalence of diffuse myocardial edema, with global elevation of T1 and T2 mapping values |
| Parasitic: *Echinococcus granulosus*, *Trichinella spiralis* | mRNA COVID-19 vaccination: There are currently limited MRI data, mostly from case series to date; MRI findings appear to be typical for viral myocarditis, although the severity and extent of MRI abnormalities reported have been relatively mild; axillary lymphadenopathy ipsilateral to the vaccination site may be present and may be a useful clue, particularly if a history of recent vaccine administration is not provided |
| Postvaccination mRNA COVID-19 vaccines: Proposed mechanisms include immune activation and dysregulation and molecular mimicry between viral spike protein and an unknown cardiac protein | mRNA COVID-19 vaccination: There are currently limited MRI data, mostly from case series to date; MRI findings appear to be typical for viral myocarditis, although the severity and extent of MRI abnormalities reported have been relatively mild; axillary lymphadenopathy ipsilateral to the vaccination site may be present and may be a useful clue, particularly if a history of recent vaccine administration is not provided |
| Systemic disease | Several systemic diseases are associated with myocardial inflammation | EGPA: MRI findings include patchy midwall and subepicardial LGE with corresponding T2 hyperintensity and subendocardial apical LGE with or without apical thrombus; concomitant pulmonary opacities might be present |
| Vasculitides: EGPA, Kawasaki disease | SLE: Patchy or linear midwall and subepicardial LGE in one-third of patients; elevated T1 and T2 value decrease following anti-inflammatory treatment; higher prevalence of pericardial and pleural effusion and thickening than in other causes of myocarditis |
| Connective tissue disorders: Systemic sclerosis, SLE, rheumatoid arthritis, dermatomyositis | Sarcoidosis: Patchy and nodular LGE with associated high T2, most common at the basal septum and basal inferolateral segment; associated findings include mediastinal and hilar lymphadenopathy and pulmonary opacities |
| Granulomatous disease: Sarcoidosis | Hypereosinophilic syndrome, cocaine, postradiation injury, thyrotoxicosis, giant cell myocarditis |
| Drug related Hypersensitivity reactions: Penicillin, cephalosporins, benzodiazepines, tricyclic antidepressants | ICI-related myocarditis: Diffusely elevated T1 and T2 values in 78% and 43% of patients, respectively; in one study, only 48% of patients met both T1 and T2 modified Lake Louise criteria; LGE present in 48% of patients, most commonly subepicardial or midmyocardial, and predominating in the basal and mid inferior and inferolateral segments |
| Toxic reactions: Anthracyclines, amphetamines, cyclophosphamide | ICI-related myocarditis: Diffusely elevated T1 and T2 values in 78% and 43% of patients, respectively; in one study, only 48% of patients met both T1 and T2 modified Lake Louise criteria; LGE present in 48% of patients, most commonly subepicardial or midmyocardial, and predominating in the basal and mid inferior and inferolateral segments |
| Immune activation or dysregulation: ICI-related myocarditis | Hypereosinophilic syndrome: Similar MRI findings to EGPA, with higher prevalence of subendocardial LGE |
| Other | Hypereosinophilic syndrome, cocaine, postradiation injury, thyrotoxicosis, giant cell myocarditis | Giant cell myocarditis: MRI appearance is similar to cardiac sarcoidosis, although LGE tends to be more extensive and right ventricular involvement more common |

Note.—ACE2 = angiotensin-converting enzyme 2, CMV = cytomegalovirus, EBV = Epstein-Barr virus, EGPA = eosinophilic granulomatosis with polyangiitis, ICI = immune checkpoint inhibitor, LGE = late gadolinium enhancement, SLE = systemic lupus erythematosus.
Cardiac MRI Assessment of Myocarditis Including after COVID-19 Vaccination

Cardiac MRI

Cardiac MRI is the most important noninvasive cardiac imaging modality for the diagnosis, follow-up, and risk stratification of patients with nonischemic myocardial inflammation, with unparalleled ability to characterize myocardial tissue. According to the 2021 American Heart Association/American College of Cardiology/American Society of Echocardiography/American College of Chest Physicians/Society for Academic Emergency Medicine/Society of Cardiovascular Computed Tomography/Society for Cardiovascular Magnetic Resonance Guideline for the Evaluation and Diagnosis of Chest Pain, cardiac MRI is useful in distinguishing myocarditis from other causes of acute chest pain in patients with myocardial injury who have nonobstructive coronary arteries at anatomic testing. Cardiac MRI is also useful in patients with suspected myocarditis or myopericarditis if there is diagnostic uncertainty or to determine the presence and extent of myocardial or pericardial inflammation and fibrosis (22).

Updated Lake Louise Criteria

MRI findings of myocardial inflammation are commonly assessed using expert consensus guidelines, the Lake Louise criteria (LLC), initially published in 2009. These criteria were broadly used in clinical practice, although evaluation was limited due to subjectivity in qualitative assessment and moderate diagnostic sensitivity (23). The LLC were revised in 2018 to incorporate parametric mapping, which allows for quantitative assessment of regional and global myocardial T1 and T2 relaxation times and extracellular volume (ECV) (24). In comparison to the original LLC, the revised criteria have significantly higher sensitivity (88% vs 73%) while maintaining very high specificity (96%) (25). According to the revised criteria, cardiac MRI provides strong evidence of acute myocardial inflammation in patients with high clinical pretest probability if at least one criterion in each of the following two categories is positive: a T2-based marker of myocardial edema and a T1-based marker of myocardial damage (Fig 3). The presence of only one marker may still support the diagnosis of myocardial inflammation in the appropriate clinical context, although with lower specificity. Importantly, these criteria were intended to be applied in patients with clinically suspected myocardial inflammation and not applied broadly as a screening test for myocardial injury in asymptomatic patients.

T2-based Criteria for Myocardial Edema

Tissue edema is a hallmark of inflammation that is often focal in the setting of myocarditis, although diffuse edema can also be identified (26). T2-based criteria for myocardial edema include regional high T2 signal intensity, global T2 signal intensity ratio equal to or greater than 2.0 on T2-weighted images, or regional or global increase of myocardial T2 relaxation time.

Assessment of myocardial edema at cardiac MRI was previously reliant on T2-weighted imaging, which has high diagnostic accuracy for focal edema, although image quality can be degraded by artifact and signal inhomogeneity, limiting reproducibility (27). T2 mapping allows for direct quantification of T2 relaxation times and is particularly useful for ruling out active inflammation given its very high sensitivity (89%) (28). High T2 signal is specific for increased tissue water and therefore can discriminate between active and healed myocarditis (29).

Figure 1: Pathophysiology of myocarditis. (Reprinted, with permission, from Valentina Sanchez Tijmes).
LGE is present both in the setting of acute inflammation (with myocyte necrosis and hyperemia) and in the setting of fibrosis (due to expansion of the extracellular space) and therefore cannot reliably differentiate between acute and healed myocarditis (6,24). Over time, the extent of LGE usually decreases as inflammation resolves and scar contracts. T1 and ECV are elevated in the setting of interstitial and replacement myocardial fibrosis. Native T1 is a composite measurement reflecting signal from both the intracellular (mainly myocytes) and extracellular (mainly interstitial) myocardial compartments, while ECV is an estimate of the proportion of the extracellular space only. These parametric mapping techniques may have incremental diagnostic and prognostic value beyond LGE, particularly in the setting of diffuse inflammation, given the ability for direct quantification of myocardial tissue changes.

T1 and ECV are also elevated in the setting of myocardial edema, although unlike elevated T2, these changes are not specific for acute inflammation (29). Given the complementary information provided by T1 and T2 mapping, it is useful to interpret these values together. For example, in a patient with suspected myocarditis, corresponding elevated T2, T1, and ECV values in-
Cardiac MRI Assessment of Myocarditis Including after COVID-19 Vaccination

**REVISITED LAKE LOUISE CRITERIA FOR MYOCARDITIS**

| MAIN CRITERIA | SUPPORTIVE CRITERIA |
|---------------|---------------------|
| ![T2 Criteria (Edema)](image) | ![Syodolic Dysfunction](image) |
| HIGH T2-Signal Intensity | REGIONAL OR GLOBAL HYPOKINESIA |
| ![T1 Criteria (Injury)](image) | PERICARDIAL ENHANCEMENT |
| NON-ISCHEMIC LGE | PERICARDITIS |
| ![High T1 or ECV](image) | |

In patients with high clinical pre-test probability of myocardial inflammation:
- Fulfilment of any T2-criteria AND any T1-criteria → Strong evidence of myocardial inflammation
- Fulfilment of any T2-criteria OR any T1-criteria → Possible evidence of myocardial inflammation
- Left ventricular systolic dysfunction and pericarditis are supportive but are not required for diagnosis

**Figure 3:** Summary of revised Lake Louise criteria for myocarditis. ECV = extracellular volume, LGE = late gadolinium enhancement.

LV Dysfunction

In more severe cases of myocarditis, regional wall motion abnormalities and systolic LV dysfunction can be identified at MRI. Systolic LV dysfunction (either regional or global) is a supportive criterion for myocarditis but is not required to make the diagnosis according to the revised LLC. After an acute episode of myocarditis, global systolic function often improves rapidly and, in most cases, returns to normal. Systolic dysfunction is typically more severe in fulminant myocarditis, and despite frequent improvement in the acute phase, LV function remains lower on average compared with nonfulminant cases at long-term follow-up (30). Myocardial strain quantification may increase the sensitivity for subtle wall motion abnormalities but has not been routinely implemented in clinical practice to date (24).

**Pericardial Inflammation**

Findings of pericardial inflammation are also considered to be supportive for the diagnosis of myocarditis, including pericardial enhancement, high T1 or T2 mapping values, or the presence of a pericardial effusion. When present, concomitant pericarditis is most commonly observed involving the pericardium adjacent to areas of inflamed myocardium, although it can also be diffuse.

**Adverse Risk Markers at MRI**

LGE is a strong, independent predictor of cardiac and all-cause mortality in patients with myocarditis (31). The risk of major adverse cardiovascular events increases by approximately 79% for every 10% increase in quantitative LGE extent (32). Of note, the presence of LGE with concomitant T2 hyperintensity is associated with better prognosis compared with isolated LGE without T2 hyperintensity. This is most likely due to the fact that LGE without associated edema typically reflects fibrosis, which is irreversible, while LGE in the context of T2 hyper-
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intensity confers the possibility of at least partial recovery as edema improves over time (33). Other important adverse prognostic MRI markers include global systolic dysfunction (LV ejection fraction < 40%) and higher T1 and ECV (32,34). In patients with acute myocarditis with evidence of myocardial edema and/or LV dysfunction, follow-up cardiac MRI may be considered 3 to 6 months after the baseline study to assess for functional recovery and the possibility of residual scarring.

Cardiac MRI Protocol and Postprocessing

In the setting of suspected myocardial inflammation, the MRI protocol should include short- and long-axis cine sequences for assessment of ventricular volumes and function, T2-based imaging (black blood T2-weighted imaging and/or T2 parametric maps), and T1-based imaging (LGE and/or pre- and post-contrast-enhancement T1 mapping) (Table 2).

One important consideration with respect to the evaluation of parametric maps is that values vary substantially on the basis of technical and patient-specific factors, including field strength. T2 values are higher at 1.5 T compared with 3 T, while T1 values are substantially higher at 3 T compared with 1.5 T. Therefore, mapping values should be compared with local reference ranges (35). Maps should be assessed visually as well as quantitatively, including global assessment of diffuse tissue changes along with focal evaluation in myocardial segments that are visually abnormal or demonstrate regional wall motion abnormalities.

For highest diagnostic performance, MRI should ideally be performed in the acute phase. Cardiac MRI markers of myocardial inflammation typically demonstrate rapid and continuous improvement during the first few weeks after the onset of symptoms (36). The sensitivity for detection of myocardial edema in particular is much lower if patients are imaged weeks after the initial clinical presentation. Establishing a diagnosis of nonacute myocarditis is particularly challenging, as findings are often nonspecific.

Cardiac MRI in Specific Causes of Myocarditis

Cardiac MRI findings demonstrate substantial overlap between different causes of myocarditis, and therefore, it is imperative that clinical features are taken into consideration. Clinical and cardiac MRI findings in specific causes of nonischemic myocardial inflammation are summarized in Table 1. Given the recent focus on the role of cardiac imaging in patients with COVID-19 and in patients with suspected myocarditis after COVID-19 vaccination, specific cardiac MRI findings in these settings are highlighted below.

COVID-19

Several cardiac MRI studies have evaluated myocardial damage in patients who recovered from COVID-19, although estimates of myocardial abnormalities have ranged widely, likely reflecting differences in patient populations, including baseline cardiac risk factors and the severity of COVID-19 illness, as well as the timing of imaging after the initial infection. A recent study found that T1 and T2 values were more commonly diffusely elevated in patients recently recovered from COVID-19 compared with patients with non–COVID-19 myocarditis (37). However, other
Table 2: Suggested Cardiac MRI Protocol for Acute Myocarditis

| Sequence          | Target                                      | Acquisition                  | Strength                          | Limitation                                                                 | Key Analysis and Reporting Point                                      |
|-------------------|---------------------------------------------|------------------------------|-----------------------------------|-----------------------------------------------------------------------------|------------------------------------------------------------------------|
| **Core Protocol** |                                             |                              |                                   |                                                                             |                                                                        |
| Cine SSFP         | Global and regional systolic function       | Short-axis stack base to apex | Accurate quantification of cardiac volumes, function, and mass                | Artifacts may degrade images and impact the accuracy of quantified values | Biventricular size, ejection fraction, and mass                         |
|                   |                                             | Long-axis (two-, three-, and four-chamber) images | Low LVEF strong predictor of adverse outcomes |                                                                             | Presence or absence of regional wall motion abnormalities               |
| LGE               | Necrosis and fibrosis                        | Short-axis stack base to apex | Identifies myocardial and pericardial inflammation and fibrosis               | Cannot reliably differentiate acute from chronic myocarditis                | Optional feature tracking strain analysis                               |
|                   |                                             | Long-axis (two-, three-, and four-chamber) images | May be useful in differentiating myocarditis from other diagnoses             |                                                                             | Presence or absence, pattern, distribution, and intensity of myocardial LGE |
|                   |                                             |                              | Strong predictor of adverse outcomes                                          |                                                                             | Presence or absence and distribution of pericardial enhancement        |
|                   |                                             |                              |                                   |                                                                             | Optional quantification of myocardial LGE extent                        |
| T2-weighted Imaging | Edema                                       | Short-axis stack base to apex | Visual evaluation of focal edema                                               | Susceptible to artifact and signal inhomogeneity                            | Presence or absence, pattern, and distribution of focal myocardial edema on the basis of visual evaluation |
|                   |                                             | Optional long-axis images     |                                   | Challenging to identify global edema                                         | Presence of global edema on the basis of increased T2 signal intensity ratio greater than or equal to 2 |
| Native T1 mapping* | Edema and fibrosis                           | Short-axis sections (base, mid, and apex) | Quantification of myocardial tissue changes (regional and diffuse), including both intracellular and extracellular processes Included in the revised LLC T1 criteria for myocardial damage | Sequences may not be available at all centers Local scanner-specific reference values are needed for interpretation Does not differentiate edema from fibrosis | Presence or absence of elevated native T1 values (regional and global) Values should be interpreted in the context of local scanner– and field strength–specific reference ranges |
| T2 mapping*       | Edema                                       | Short-axis sections (base, mid, and apex) | Quantification of myocardial edema (regional and diffuse) Specific for edema Included in the revised LLC T2 criteria for myocardial edema | Sequences may not be available at all centers Local scanner-specific reference values are needed for interpretation Does not detect fibrosis | Presence or absence of elevated native T2 values (regional and global) Values should be interpreted in the context of local scanner– and field strength–specific reference ranges |
| Optional sequence |                                             |                              |                                   |                                                                             |                                                                        |
| EGE               | Hyperemia and capillary leak                 | Short-axis stack base to apex | Assessment of hyperemia Included in the original LLC | Requires administration of contrast agent Not included in the revised LLC | Presence of regional or global EGE on the basis of EGE ratio greater than or equal to 4 |

studies have reported more focal MRI abnormalities typical of non-COVID myocarditis in patients who have recovered from COVID-19, including subepicardial LGE (Fig 5) (38). Data regarding MRI findings in COVID-19–related myocardial injury continue to evolve, with multiple large studies currently underway.
Myocarditis after COVID-19 Vaccination

Myocarditis has been reported in a minority of people following administration of mRNA-based COVID-19 vaccines, including mRNA-1273 (Moderna) and BNT162b2 mRNA (Pfizer-BioNTech), with symptom onset typically within a few days of vaccination (median, 2–3 days). Myocarditis is three to five times more frequent after the second dose compared with the first, although patients with prior history of COVID-19 are at higher risk after the first dose. The U.S. Vaccine Adverse Event Reporting System (VAERS) received 1903 reports of myopericarditis among people who received at least one dose of a COVID-19 vaccine as of August 18, 2021 (9), in the context of nearly 360 million total doses administered. As of June 2021, there were approximately 40.6 cases of myocarditis reported per million second doses administered to males aged 12–29 years and 2.4 cases reported per million second doses in men aged 30 years or older (39); for females, reported rates were 4.2 and 1.0 per million second doses for the same categories, respectively. Importantly, VAERS relies on passive reporting, and the data cannot be used to determine whether a vaccine is causally related to an adverse event. Data from the largest integrated health care organization in Israel indicate that vaccination with BNT162b2 mRNA vaccine is associated with an excess risk of myocarditis (risk ratio 3.2 and risk difference 2.7 events per 100,000 persons when compared with age- and risk-matched controls). However, the risk of myocarditis following SARS-CoV-2 infection was much higher (risk ratio 18.3 and risk difference 11.0 events per 100,000 persons) (40).

Given the relatively short time frame with which COVID-19 vaccines have been administered, data regarding the prevalence and pattern of abnormalities at cardiac MRI following vaccination are still emerging. There are only a few published case series describing cardiac MRI findings after COVID-19 vaccination to date, summarized in Table 3. The largest MRI case series of vaccine-associated myocarditis includes 15 patients (range, four to 15 patients). Of note, almost all patients who underwent MRI in the context of myocarditis following COVID-19 vaccination included in case series to date have been hospitalized. It is possible that these patients reflect the more severe end of the spectrum of vaccine-associated myocardial changes due to reporting bias. Typical cardiac MRI findings reported to date in patients with myocarditis following COVID-19 vaccination are similar to findings in nonvaccine myocarditis, including subepicardial LGE with a predilection for the basal inferolateral wall along with corresponding myocardial edema (Fig 6) (41–51). Other findings include pericardial enhancement and axillary lymphadenopathy ipsilateral to the vaccine administration site (52). When reported, impaired LV ejection fraction (<50%–55%) was identified in 14%–25% of patients.

Differentiating vaccine-associated myocarditis from other causes of myocardial injury at cardiac MRI may be a challenge, as the pattern of findings is similar, and there are no longitudinal imaging studies to suggest how long abnormalities persist. However, accurate diagnosis is important, as this could impact patient treatment; current recommendations indicate that individuals who develop myocarditis or pericarditis after a dose of an mRNA vaccine defer receiving a subsequent dose until additional data are available (53). Clinical history, including the timing of symptom onset in relation to vaccine administration, is highly relevant. In patients with signs or symptoms suggestive of myocarditis following vaccination, cardiac MRI should ideally be performed as soon as possible after the onset of symptoms to maximize the likelihood of detecting myocardial edema, which would suggest an acute

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**Table 2 (continued): Suggested Cardiac MRI Protocol for Acute Myocarditis**

| Sequence         | Target                              | Acquisition | Strength                                           | Limitation                                                       | Key Analysis and Reporting Point                                      |
|------------------|-------------------------------------|-------------|----------------------------------------------------|-----------------------------------------------------------------|--------------------------------------------------------------------|
| First pass perfu-| Hyperemia and capillary leak         | Short-axis  | Assessment of hyperemia. Can be used to exclude   | Requires administration of contrast agent                       | Presence or absence and distribution of perfusion defects          |
| sion              |                                     | sections    | substantial coronary artery disease with          | Limited interobserver agreement                                 | Optional quantification of myocardial blood flow                    |
|                   |                                     | (base, mid,| pharmacologic stress.                             | Not included in the revised LLC                                |                                                                    |
|                   |                                     | and apex)  |                                                    |                                                                  |                                                                    |
| ECV               | Extracellular edema and fibrosis     | Short-axis  | Quantification of the extracellular space.        | Requires contemporary hematocrit levels for calculation         | Presence or absence of elevated ECV values (regional and global)   |
|                   |                                     | sections    | Included in the revised LLC/T1 criteria for      | Requires both pre- and post–contrast-enhanced T1 mapping       |                                                                    |
|                   |                                     | (base, mid,| myocardial damage.                                | Requires administration of contrast                              |                                                                    |
|                   |                                     | and apex)  |                                                    | Sequences may not be available at all centers                  |                                                                    |

Note.—ECV = extracellular volume, EGE = early gadolinium enhancement, LGE = late gadolinium enhancement, LLC = Lake Louise criteria, LVEF = left ventricular ejection fraction, SSFP = steady-state free precession.

*Native T1 and T2 mapping should be included in the core protocol when available, particularly if there is a contraindication to administration of contrast agent that would preclude LGE imaging (T1 mapping) or if T2-weighted imaging is unavailable or degraded by artifact (T2 mapping).
Cardiac MRI Assessment of Myocarditis Including after COVID-19 Vaccination

Figure 5: Myocardial injury and pericarditis following COVID-19. Case example in a 57-year-old woman with COVID-19 who presented with chest pain after having elevated troponin levels. Cardiac MRI performed at 1.5 T 4 weeks after polymerase chain reaction–confirmed diagnosis of SARS-CoV-2 infection demonstrates subepicardial late gadolinium enhancement at the (A) basal inferior lateral wall with adjacent pericardial enhancement (red arrows), with (B) corresponding high T2 signal (orange arrows), and (C) high regional native T1 (1236 msec) and (D) high regional native T2 (67 msec) on short-axis images, in keeping with myopericarditis.

process (36). If MRI is performed several weeks to months after symptom onset and no T2 abnormality is identified, it is difficult to attribute myocardial tissue changes to a specific cause. This may be a particular challenge in symptomatic patients who have received an mRNA vaccine and have a prior history of COVID-19. Importantly, there are no data to suggest a role for routine imaging or screening of asymptomatic individuals after COVID-19 vaccination in the absence of signs or symptoms suggestive of myocarditis.

In most reported cases of myocarditis following COVID-19 vaccination, the clinical course has been favorable, with rapid resolution of symptoms and corresponding decreases in troponin levels over short-term follow-up, suggesting that patients might have a good long-term prognosis. Given that the risk of myocardial injury and other severe outcomes after COVID-19 is higher, current data are supportive of continued COVID-19 immunization on the basis of the balance of risks and benefits (54). Larger studies with longer-term follow-up are required to evaluate long-term outcomes, to directly compare imaging findings after COVID-19 vaccination to other causes of myocarditis, to assess longitudinal MRI changes after clinical recovery, and to determine the risk associated with subsequent vaccine administration in patients with a prior history of myocarditis.

Conclusion
Cardiac MRI is an important imaging modality in patients with suspected myocardial inflammation and myocarditis, allowing for noninvasive assessment of myocardial edema and injury, and identification of potentially treatable underlying causes of inflammation to guide management and improve patient outcomes. Cardiac MRI may be particularly useful in patients presenting with signs and symptoms suggestive of myocarditis after COVID-19 vaccine administration, although further study is needed.
Table 3: Cardiac MRI Findings after COVID-19 Vaccination

| Reference | No. of Patients* | Age† (y) | Pfizer/ BioNTech | Moderna | J&J | Presentation after Second Dose | Days between Vaccination and Symptom Onset† | Timing of Cardiac MRI | Other Imaging and Clinical Finding | Cardiac MRI Finding |
|-----------|------------------|----------|------------------|---------|-----|-------------------------------|-------------------------------------------|----------------------|-----------------------------------|---------------------|
| Patel et al (41) | 5 (5) | 22 (19–37) | 4 (80) | 1 (20) | 0 (0) | 4 (80) | 2 (1–3) | 1–7 days after symptom onset | All admitted to hospital with elevated troponin levels and chest pain at presentation, all tested negative for COVID-19 at presentation, normal ECG findings in 80% (4/5) | LGE in 100% (5/5), subepicardial (5/5) and midwall (1/5); corresponding edema in three of five (60%) | Most common segment involved was the basal inferolateral wall (3/5) |
| Shaw et al (42) | 4 (2) | 21 (16–31) | 3 (75) | 1 (25) | 0 (0) | 2 (50) | 4 (2–5) | N/R | All presented with chest pain and elevated troponin levels; prior history of COVID-19 in 50% (2/4), and both presented with myocarditis after the first vaccine dose | LGE in 100% (4/4), subepicardial (4/4) and midwall (1/4) | Corresponding high T2, T1, and ECV in 100% (4/4) | Mildly impaired LVEF (54%) in 25% (1/4) | Pericardial enhancement in 0% | Most common segment involved was the basal inferolateral wall (4/4) |
| Rosner et al (43) | 7 (7) | 24 (19–30) | 5 (72) | 1 (14) | 1 (14) | 5 (71) | 3 (2–7) | 3–37 days after vaccination | All admitted to hospital with elevated troponin levels and chest pain at presentation; six of seven tested for COVID-19 at presentation and all negative; one (14%) prior history COVID-19; normal ECG findings in 29% (2/7); clinical symptoms resolved rapidly (with hospital discharge within 4 days) | LGE in 100% (7/7), subepicardial (7/7) and midwall (4/7); distribution not specified | Corresponding myocardial edema in 43% (3/7), two with no edema had MRI more than 7 days after presentation and one had artifact on T2 images | Impaired LVEF (<50%) in 14% (1/7) and regional wall motion abnormality with hypokinesis in 14% (1/7) | Pericardial enhancement in 29% (2/7) |
| Larson et al (44) | 8 (8) | 29 (21–56) | 5 (63) | 3 (37) | 0 (0) | 7 (88) | 3 (2–4) | N/R | All admitted to hospital with elevated troponin levels and chest pain at presentation; all tested negative for COVID-19 at presentation; one (13%) had prior history of COVID-19 (only patient who presented after the first vaccine dose); ECG not reported at baseline; clinical symptoms resolved in 100% | LGE in 100% (8/8); pattern and distribution not specified | Myocardial edema in 75% (6/8) | Pericardial effusion in 38% (3/8) | Impaired LVEF (<50%) in 25% (2/8) | Regional wall motion abnormality in 63% (5/8) and generalized hypokinesis in 38% (3/8) |

Table 3 (continues)
Table 3 (continued): Cardiac MRI Findings after COVID-19 Vaccination

| Reference   | No. of Patients* | Age† (y) | Pfizer/ BioNTech | Moderna | J&J | Presenta- tion after Second Dose | Days between Vaccination and Symptom Onset† | Timing of Cardiac MRI | Other Imaging and Clinical Finding | Cardiac MRI Finding |
|-------------|-----------------|----------|------------------|--------|-----|----------------------|------------------------------------------|---------------------|-----------------------------------|---------------------|
| Marshall et al (45) | 7 (7)         | 17 (14–19) | 7 (100) | 0 (0) | 0 (0) | 7 (100) | 2 (2–4) | 1–6 days after symptom onset | All admitted to hospital with elevated troponin levels and chest pain at presentation; 86% (6/7) tested for COVID-19 and all negative; normal ECG findings in 71% (5/7); clinical symptoms resolved rapidly (with hospital discharge within 6 days) | LGE in 100% (7/7), most frequently subepicardial; one patient had diffuse LGE at the left ventricular lateral wall. Myocardial edema in 86% (6/7). Most common segments involved were the basal and mid inferolateral wall, basal anterolateral wall, and apical lateral wall. Axillary lymphadenopathy in one patient. |
| Kim et al (46) | 4 (3)          | 30 (23–70) | 2 (50) | 0 (0) | 0 (0) | 4 (100) | 2.5 (1–5) | 3–5 days after vaccination | All admitted to hospital with elevated troponin levels and chest pain at presentation; all tested negative for COVID-19 at presentation; ECG not reported; clinical symptoms resolved (with hospital discharge between 2–4 days) | LGE in 100% (4/4), subepicardial (3/4) and pericardial or diffuse (1/4); distribution not specified in all. Corresponding high T1 in 100% (4/4), but remote T1 was normal in all (4/4). Corresponding myocardial edema in 75% (3/4). Pericardial effusion in 50% (2/4). Pericardial enhancement in 0% Impaired LVEF (<50%) in 25% (1/4). |
| Abu Mouch et al (47) | 6 (6)         | 23 (16–45) | 6 (100) | 0 (0) | 0 (0) | 5 (83) | 3 (1–16) | N/R | All admitted to hospital with elevated troponin levels and chest pain at presentation; all tested negative for COVID-19 at presentation; normal ECG findings in 67% (4/6); two of six mildly impaired LVEF (50%–55%); clinical symptoms resolved (with hospital discharge between 4–8 days) | LGE in 100% (6/6), subepicardial (4/6), midwall (2/6), and diffuse (1/6). Corresponding myocardial edema in 100% (6/6). Most common segments involved were basal to mid inferolateral wall, basal to mid anterolateral wall, basal anteroseptum, and apex. Pericarditis in 17% (1/6). |

Table 3 (continue)
Table 3 (continued): Cardiac MRI Findings after COVID-19 Vaccination

| Reference | No. of Patients* | Age† (y) | Vaccine | Presentation after Second Dose | Days between Vaccination and Symptom Onset† | Timing of Cardiac MRI | Other Imaging and Clinical Finding | Cardiac MRI Finding |
|-----------|-----------------|----------|---------|-------------------------------|---------------------------------------------|----------------------|------------------------------------|---------------------|
| Starukova et al (48) | 5 (4) | 21 (17–38) | Pfizer/BioNTech 3 (60) Moderna 2 (40) J&J 0 (0) | 5 (100) | 3 (2–3) | 3–5 days after vaccination | All admitted to hospital with elevated troponin levels and chest pain at presentation; all tested negative for COVID-19 at presentation, and none had a prior history of COVID-19; ECG findings not reported; no follow-up data reported | LGE in 100% (5/5), most commonly subepicardial; distribution not specified; Corresponding myocardial edema at T2-weighted imaging in 100% (5/5); Pericardial enhancement in 100% (5/5); Axillary lymphadenopathy ipsilateral to the site of vaccination in 80% (4/5) |
| Montgomery et al (49) | 23 (23) | 25 (20–51) | Pfizer/BioNTech 7 (30) Moderna 16 (70) J&J 0 (0) | 20 (87) | 2.5 (1–5) | N/R | Previously healthy military service members, all presented with chest pain and had elevated troponin levels; hospitalization status not reported; all three presenting after the first vaccine dose had a prior history of COVID-19; normal ECG findings in 83%; LVEF impaired in 17% (4/23); cardiac symptoms resolved within 1 week of onset in 16 of 23 | Only eight patients underwent cardiac MRI with limited data reported; Subepicardial LGE and/or edema in 100% (8/8) |
| Dionne et al (50) | 15 (14) | 15 (12–18) | Pfizer/BioNTech 0 (0) Moderna 15 (100) J&J 0 (0) | 14 (93) | 3 (1–6) | 1 to 7 days after symptom onset | All admitted to hospital with elevated troponin levels and chest pain at presentation; ECG demonstrated decreased LVEF in 20% (3/15) and abnormal global longitudinal or circumferential strain in 33% (5/15); All patients discharged from hospital within 5 days; at follow-up (1 to 13 days after discharge), troponin levels remained mildly elevated in 20% (3/15) | MRI consistent with myocarditis in 87% (13/15); LGE present in 80% (12/15); most common segments involved were the basal to mid anterolateral and inferolateral wall; Corresponding myocardial edema on T2-weighted imaging in 13% (2/15); High ECV (>30%) in 25% (3/15), and high global native T1 (>1100 msec at 1.5 T) in 13% (2/15); Low LVEF (<55%) in 25% (3/15) |
## Cardiac MRI Assessment of Myocarditis Including after COVID-19 Vaccination

| Vaccine | Days between Vaccination and Symptom Onset | Other Imaging and Clinical Finding | Cardiac MRI Finding |
|---------|--------------------------------------------|-----------------------------------|---------------------|
| Pfizer/BioNTech Moderna J&J | 5-8 days after vaccination | All admitted to hospital with elevated troponin levels and chest pain at presentation; normal ECG findings in 80% (6/7); LVEF impaired in 20% (2/10) during hospital admission, one patient discharged from hospital within 9 days. | LGE in 100% (5/5), subepicardial, Chelada et al (51) and mid to subepicardial in 80% (5/5); LVEF impaired in 20% (2/10); most common segments in 80% (5/5); during hospital admission, one patient discharged from hospital within 9 days. |
| Pfizer/BioNTech BNT162a, and BioNTech BNT162b2 | 5 (5) 17 (16–19) 4 (3–4) 5–8 days after vaccination | LVEF = left ventricular ejection fraction. | Myocardial edema at T2-weighted imaging in 100% (5/5). |

### Cardiac MRI Findings after COVID-19 Vaccination

- **Days between Vaccination and Symptom Onset**: 5 (5) 17 (16–19) 4 (3–4) 5–8 days after vaccination.
- **Other Imaging and Clinical Finding**: All admitted to hospital with elevated troponin levels and chest pain at presentation; normal ECG findings in 80% (6/7); LVEF impaired in 20% (2/10) during hospital admission, one patient discharged from hospital within 9 days.
- **Cardiac MRI Finding**: LGE in 100% (5/5), subepicardial, Chelada et al (51) and mid to subepicardial in 80% (5/5); LVEF impaired in 20% (2/10); most common segments in 80% (5/5); during hospital admission, one patient discharged from hospital within 9 days.

### Table 3 (continued): Cardiac MRI Findings after COVID-19 Vaccination

| Vaccine | Days between Vaccination and Symptom Onset | Other Imaging and Clinical Finding | Cardiac MRI Finding |
|---------|--------------------------------------------|-----------------------------------|---------------------|
| Pfizer/BioNTech Moderna J&J | 5-8 days after vaccination | All admitted to hospital with elevated troponin levels and chest pain at presentation; normal ECG findings in 80% (6/7); LVEF impaired in 20% (2/10) during hospital admission, one patient discharged from hospital within 9 days. | LGE in 100% (5/5), subepicardial, Chelada et al (51) and mid to subepicardial in 80% (5/5); LVEF impaired in 20% (2/10); most common segments in 80% (5/5); during hospital admission, one patient discharged from hospital within 9 days. |
| Pfizer/BioNTech BNT162a, and BioNTech BNT162b2 | 5 (5) 17 (16–19) 4 (3–4) 5–8 days after vaccination | LVEF = left ventricular ejection fraction. | Myocardial edema at T2-weighted imaging in 100% (5/5). |

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