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Review

MRI of cardiac involvement in COVID-19

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Abstract

The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has led to a diverse pattern of myocardial injuries, including myocarditis, which is linked to adverse outcomes in patients. Research indicates that myocardial injury is associated with higher mortality in hospitalized severe COVID-19 patients (75.8% vs 9.7%). Cardiovascular Magnetic Resonance (CMR) has emerged as a crucial tool in diagnosing both ischaemic and non-ischaemic myocardial injuries, providing detailed insights into the impact of COVID-19 on myocardial tissue and function. This review synthesizes existing studies on the histopathological findings and CMR imaging patterns of myocardial injuries in COVID-19 patients. CMR imaging has revealed a complex pattern of cardiac damage in these patients, including myocardial inflammation, oedema, fibrosis, and ischaemic injury, due to coronary microthrombi. This review also highlights the role of LLC criteria in diagnosis of COVID-related myocarditis and the importance of CMR in detecting cardiac complications of COVID-19 in specific groups, such as children, manifesting multisystem inflammatory syndrome in children (MIS-C) and athletes, as well as myocardial injuries post-COVID-19 infection or following COVID-19 vaccination. By summarizing existing studies on CMR in COVID-19 patients and highlighting ongoing research, this review contributes to a deeper understanding of the cardiac impacts of COVID-19. It emphasizes the effectiveness of CMR in assessing a broad spectrum of myocardial injuries, thereby enhancing the management and prognosis of patients with COVID-19 related cardiac complications.

Keywords: CMR; COVID-19; myocarditis; SARS-CoV-2.

Background

Since the onset of the COVID-19 pandemic in December 2019 in Wuhan, China, up to October 2023, there have been approximately 771 million confirmed cases and nearly 7 million associated deaths.[1] COVID-19 is caused by the SARS-CoV-2 virus, which belongs to the Coronaviridae family.[2] Notably, MERS-CoV, SARS-CoV, and SARS-CoV-2, members of this family, are probably zoonotic viruses responsible for severe respiratory illnesses.[3] COVID-19 is a multisystemic disease that predominantly affects the respiratory and cardiovascular systems. This is attributed to the fact that angiotensin-converting enzyme 2 (ACE2) receptors are abundant in these systems and serve as the entry receptors for SARS-CoV-2.[4,5] Studies have identified that the primary risk factors for mortality in COVID-19 patients include advanced age (over 60 years), male gender, and the presence of comorbidities such as hypertension, obesity, and myocardial injury.[6,7] Shaobo et al have revealed that 15.8% of all admitted patients have had myocardial injury based on high blood levels of troponin I (cTnI). Of note, patients who died had suffered more often from myocardial injury during hospitalization compared with survivors (75.8% vs 9.7%).[8] Cardiovascular magnetic resonance has emerged as a reference imaging tool for assessing myocardial function and tissue characterization. The American College of Cardiology, the European Society of Cardiology, and the Society for CMR all emphasize that CMR is a useful diagnostic tool for patients with COVID-19 who show evidence of myocardial injury and cardiac dysfunction.[9-12]

Myocardial injury in COVID-19

Myocardial injury can be broadly categorized into two types: ischaemic and non-ischaemic.

Ischaemic myocardial injury primarily results from acute plaque rupture or erosion,[13,14] but cases of myocardial infarction without obstructive coronary arteries (MINOCA) have been reported in the context of acute COVID-19 due to triggers like direct viral endothelial cell infection (endothelitis)[14,15] and prothrombotic effects from the massive cytokine release associated with COVID-19.[16]

Conversely, non-ischaemic myocardial injury may occasionally arise from direct viral infection of cardiomyocytes[17] or, indirectly, via immune responses and systemic hyperinflammation, as reported in multisystem inflammatory syndrome (MIS-C), particularly in children.[18-20] Infrequently, COVID-19 can also lead to stress-induced cardiomyopathy (Takotsubo) with reversible myocardial injury,[21,22] also potentially leading to acute heart failure.[23]

Histopathological findings of COVID-19-related myocardial injury

The mechanisms underlying myocardial injury in patients with COVID-19 are varied and not yet fully understood.

Most of our understanding of the histopathologic manifestations of COVID-related myocardial injury stems from post-mortem examination of deceased hospitalized patients. There is growing evidence highlighting that vascular leakage and

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tissue oedema are principal contributors to myocardial injury in severe COVID-19 cases.[24] Several mechanisms explain the development of myocardial oedema: First, direct invasion of endothelial cells by SARS-CoV-2, causing endothelitis characterized by endothelial dysfunction and subsequent cellular necrosis.[24,25] Second, the downregulation of the ACE2 receptor may lead to increased angiotensin 2 and activation of the kallikrein-bradykinin pathway, resulting in increased vascular permeability[4]; and third, a surge in inflammatory cytokines and vasoactive molecules, disrupting inter-endothelial junctions.[26]

Sang et al[27] reported having frequently observed myocardial fibrosis (80%), hypertrophy (72.0%), and coronary microthrombi (66.0%) in deceased COVID-19 patients post-mortem.

Halushka et al reviewed findings from 277 cardiac autopsies, revealing that non-myocarditis inflammatory infiltrate and single cell ischemia were the predominant cardiac findings, present in 12.6% and 13.7% of cases, respectively, while classical myocarditis based on Dallas criteria was identified in only 1.4% of the cases.[28]

A review analysing immunohistochemical data from 209 cardiac autopsies of severe SARS-CoV-2 patients revealed myocardial infiltration of CD3+, CD8+ cytotoxic lymphocytes, and CD68+ macrophages. The presence of CD3+ lymphocytes emphasizes that cellular immunity is an essential part of the host response during COVID-19 infection.[29] Another proposed mechanism underlying myocardial damage is ischaemic injury.[30] Upon examination of 40 hearts, Pellegrini et al identified microthrombus formation predominantly in small vessels as the main pathological cause of myocyte necrosis, accounting for 78.6% of cases.[31]

Imaging assessment of COVID-19 induced cardiac injury

Although endomyocardial biopsy is by many considered the gold standard for diagnosing acute or chronic inflammatory cardiac disorders, it is recommended only in patients with complicated presentations or significant clinical deterioration despite proper treatment, including high-grade heart block or ventricular arrhythmia, once obstructive coronary artery disease is excluded.[32,33] According to the ACC Expert Consensus Decision Pathway on Cardiovascular Sequelae of COVID-19 in Adults, transthoracic echocardiography (TTE) is the cardiac imaging modality of choice in patients presenting with cardiac symptoms (chest pain, dyspnoea, palpitations, syncope); other markers are elevated cardiac troponin (cTn); and abnormal electrocardiographic (diffuse T-wave inversion, ST-segment elevation without reciprocal ST-segment depression, prolongation of the QRS complex duration).[32]

Dweck et al analysed data from an international survey on the clinical use of TTE in 1216 patients hospitalized with COVID-19. Their findings revealed that LV abnormalities were observed in approximately half of the patients, and imaging findings led to change in management in one-third of cases (33%).[34] While TTE is a clinically extremely useful non-invasive tool with excellent accessibility and bedside applicability, especially for assessing ventricular function, it is not the optimal choice for ruling out myocardial injury in this group of patients. This limitation can be related to echocardiography's inability to provide significant information on the myocardial tissue characteristics associated with COVID-19.

There is limited data on the utility of molecular imaging for diagnosing acute myocardial injury related to COVID-19. However, a recent study by Hanneman et al showed evidence of myocardial inflammation on fluorodeoxyglucose—positron emission tomography (FDG-PET) in 17% of 47 patients recently recovered from COVID-19.[35]

Assessment of myocardial injury using CMR

Cardiovascular magnetic resonance has been established as the non-invasive gold standard tool for the *in vivo* diagnosis of ischaemic and non-ischaemic myocardial injury. Cine CMR imaging offers a high spatial and temporal resolution for an accurate analysis of both left and right ventricular regional wall motion and global function. Additionally, strain imaging can detect subtle and potentially subclinical myocardial functional abnormalities.[36] Furthermore, CMR has the unique capability to verify or rule out myocardial oedema by T2-weighted imaging or T2 mapping; it also provides markers from native and post contrast T1 maps for diffuse interstitial fibrosis via estimation of extracellular volume (ECV).[12,37] Late gadolinium enhancement (LGE) imaging is used for detecting of replacement fibrosis in the myocardium. The patterns of LGE help to differentiate between ischaemic and non-ischaemic causes of myocardial fibrosis.[38] Generally, ischaemic myocardial injury often leads to LGE in a subendocardial or transmural pattern in coronary arteries distribution, while non-ischaemic injury affects subepicardial or mid-myocardial layers.[39,40] The 2018 consensus criteria for CMR in myocardial inflammation, known as the Lake Louise Criteria (LLC), recommend utilizing at least one T2-based criterion for myocardial oedema (either a global or regional elevation in myocardial T2 relaxation time or heightened signal intensity on T2-weighted CMR images) in conjunction with at least one T1-based criterion for myocardial injury (elevated myocardial T1, high ECV fraction, or LGE[41] Figure 1). Furthermore, CMR can also be used to assess myocardial perfusion using stress perfusion imaging[42,43] or to study coronary vascular function by tracking changes of myocardial oxygenation during vasoactive interventions using Oxygenation-Sensitive CMR.[44]

Several non-invasive imaging modalities are available to assess microvascular dysfunction by measuring myocardial blood flow (MBF) and myocardial perfusion reserve (MPR), characterized by the ratio of maximum hyperaemic response to resting coronary blood flow.[45] While cardiac PET is acknowledged as the standard method for MBF quantification,[46] CMR has emerged as a reliable alternative. CMR employs stress-induced first-pass perfusion following the administration of a vasodilator agent such as adenosine or regadenoson to measure peak MBF, followed by an assessment at rest.[45,47] Various CMR techniques have been applied to myocardial perfusion measurement, including semi-quantitative and fully quantitative methods. Semiquantitative methods can be used to analyse variations in myocardial signal intensity during the passage of contrast media, whereas fully quantitative techniques require a measurable relationship between myocardial signal intensity variation and underlying coronary blood flow, offering the advantage of calculating a broader range of perfusion indexes.[45,48] The accuracy of the semiquantitative methods can be influenced by the pharmacodynamic and pharmacokinetic properties of the contrast agents used. Most quantitative analyses necessitate the mathematical deconvolution of measured blood (arterial input

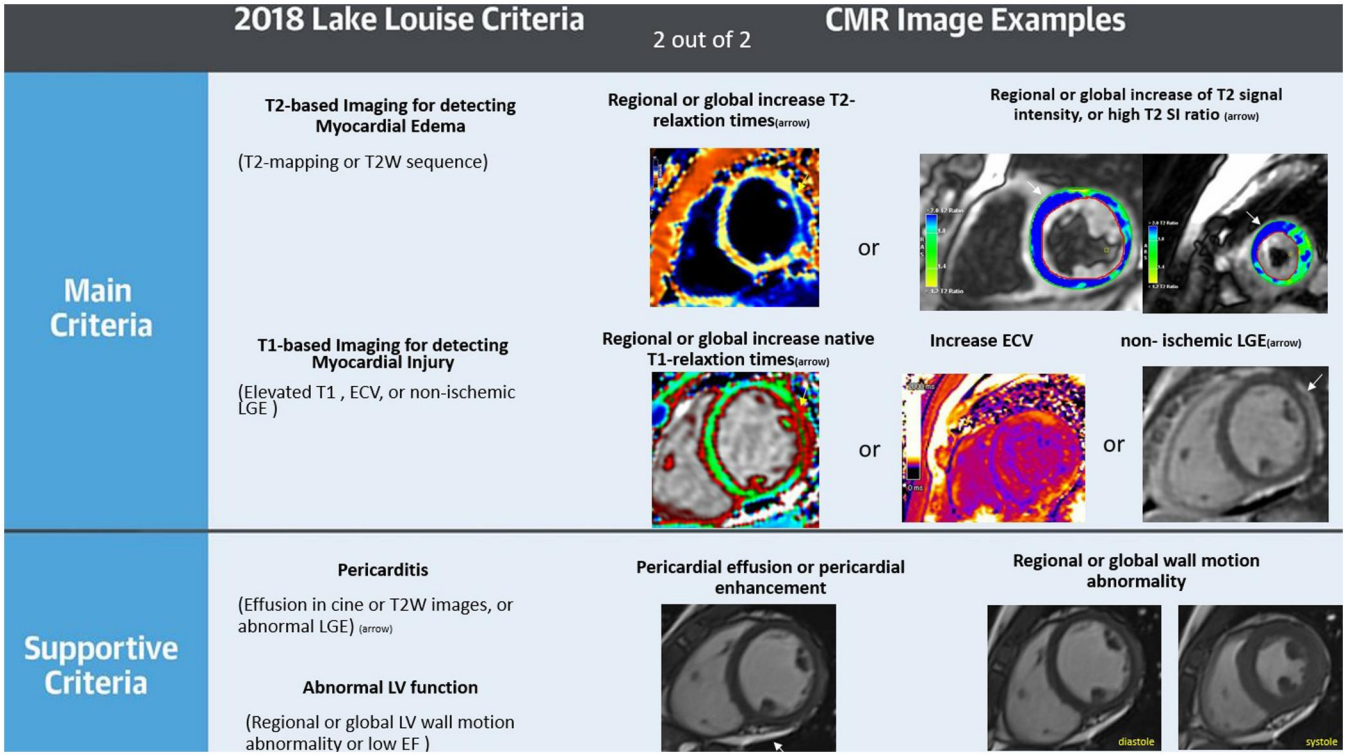

Figure 1. The LLC. The 2018 LLC diagnostic consensus guideline includes two primary criteria: T1- and T2-based. A positive T1-based criterion is indicated by prolonged native T1 relaxation times, elevated ECV, or non-ischaemic LGE marked by a white arrowhead. The T2-based criterion is considered positive with elevated T2 relaxation times, localized areas of high signal intensity on T2-weighted images identified by white arrows, or an increased overall T2 signal intensity. Of note, in case with very suggestive clinical presentation, even one criterion would support the diagnosis of myocarditis. Abbreviations: SI = signal intensity, T2-W = T2 weighted, LGE = late gadolinium enhancement, ECV = extracellular volume. Ferreira VM, et al. Journal of the American College of Cardiology. 2018 Dec 18;72(24):3158–76. with permission (41).

function) and tissue (tissue function) enhancement data, from which myocardial perfusion is computed.[45,46,49] Recent advancements have validated the accuracy of automated quantitative CMR MBF maps.[50]

Although the efficacy of CMR using the LLC for a non-invasive diagnosis of acute myocarditis is well-established, CMR remains underutilized in COVID-19. This underuse can be attributed in part to the limited accessibility of cardiac MRI in routine clinical settings,[51] the difficulty of patients with lung involvement to hold their breath, and concerns about procedure-related infections.[9]

Pattern of cardiac injury in CMR images

There are various patterns of cardiac injury secondary to SARS-CoV-2 infection, including myocarditis (Figure 2), pericarditis, myopericarditis, inducible ischaemia, infarction, and less commonly, stress-induced cardiomyopathy (Figure 3). The LLC are frequently used to diagnose COVID-19-related myocarditis[32,37] (Figure 2). Several studies found a more global myocardial injury as evident by an elevated myocardial T1, consistent with diffuse myocardial fibrosis (or oedema), and high T2 values, indicating myocardial oedema.[52-54] However, other studies have demonstrated more focal findings in a non-ischaemic pattern of myocardial injury, such as subepicardial, mid-wall, or patchy fibrosis, as evidenced in LGE images.[12,55,56] A controversial study reported myocardial LGE in 30% and pericardial enhancement in 22% of 100 patients who had recently recovered from COVID-19,[57] while in a large international multicentric study involving 56 963 hospitalized COVID-19 patients, Ammirati et al found a much lower prevalence of acute myocarditis, ie, between 2.4 and 4.1 per 1000 patients.[58]

The LLC have been validated with classic histopathologic evidence of myocarditis per the Dallas criteria.[37,41] Their efficacy in diagnosing myocardial injury after SARS-CoV-2 infection, mainly assessed in patients recovered from an acute episode[59] and those with long COVID-19 with persistent cardiac symptoms, however, is less well established.[12,60] Considering the moderate sensitivity of CMR, even one T1 or T2-based criterion may still support the diagnosis in cases with a very suggestive clinical presentation, especially when associated with supportive criteria like pericarditis (enhanced pericardium and/or evidence of pericardial effusion) (Figure 4) or left ventricular dysfunction.[12,37]

Reports on the clinical utility of CMR to evaluate myocardial injury in the early, hyperacute phase of severe COVID-19 are scarce, with initial findings derived from small cohorts or single-centre studies only. This limitation is largely due to the infrequent use of CMR in patients experiencing hemodynamic instability or requiring mechanical ventilation.

There are case reports that have highlighted abnormal myocardial T1 and T2-relaxation times, as well as non-ischaemic myocardial injury on LGE images[54,61] (Figure 2). Galea et al, in a small cohort of 27 patients with active COVID-19 and suspected cardiac involvement, found that prolonged T2-relaxation was the most prevalent abnormality correlated with hs-cTn values.[62] In a recent comprehensive, multicentric prospective study (COVID-HEART study), 519 hospitalized COVID-19 patients with elevated troponin levels underwent CMR to assess acute myocardial injury. A significant

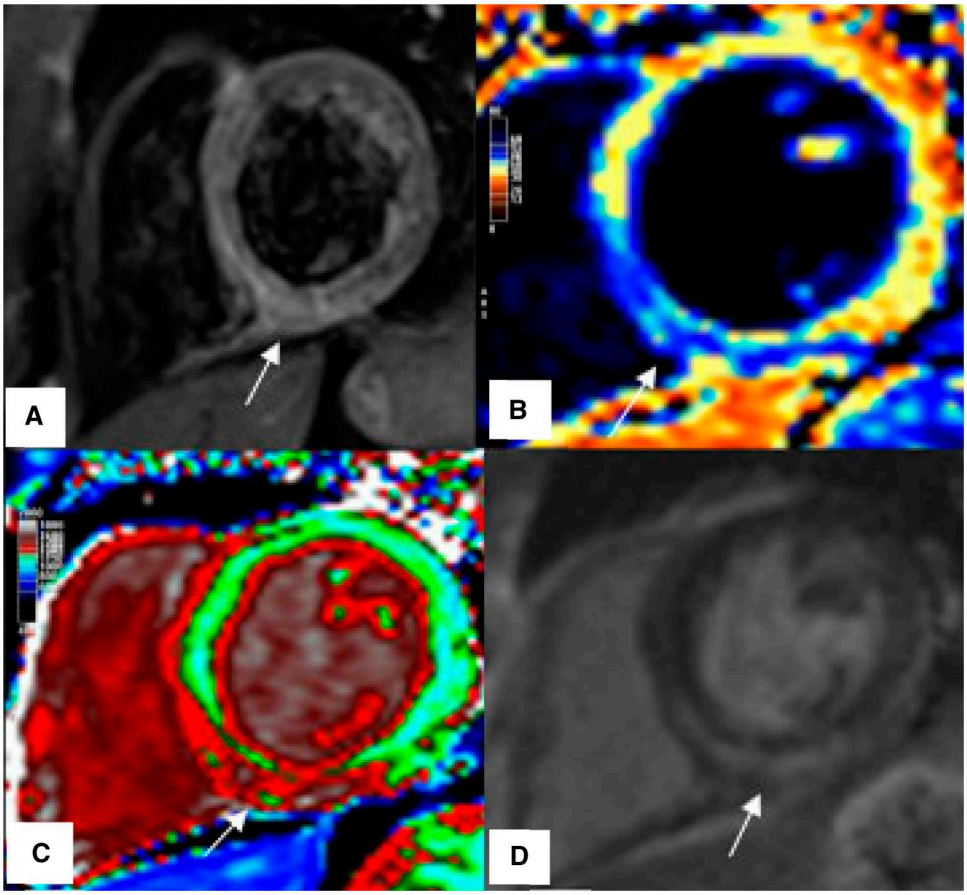

Figure 2. Example of classic myocarditis according to LLC during acute COVID-19, patient presented with chest pain and elevated troponin levels. (A) T2-W, Regional increase myocardial signal intensity in the mid inferoseptal, inferior wall consistent with myocardial edema (arrow), (B) T2 map, Regional high T2-times in the inferoseptal segment (blue), (C) T1 map, Regional increase native T1-times in the inferoseptal and inferior segments (red), (D) LGE, Linear subepicardial enhancement in the mid inferoseptal, inferior and inferolateral segments as well as mid-myocardial enhancement involving the septum (arrow).

proportion of 42% of patients demonstrated myocardial scarring on LGE images, six times more than COVID-19 patients with normal troponin levels. The LGE patterns were diverse, encompassing ischaemic, non-ischaemic, mixed, and nonspecific presentations. Moreover, the extent of the scarring was linked to adverse cardiac remodelling.[63] Interestingly, global myocardial T2 values were not elevated in these patients. However, this can be explained by the fact that CMR scans were performed on average 30 days after admission, when acute inflammation with associated oedema typically would have receded.

A study involving 148 patients hospitalized with severe acute respiratory syndrome from COVID-19 and elevated troponin levels revealed that about one-third showed a myocarditis-like pattern on LGE images. In addition, 22% showed evidence of infarction and/or ischaemia, and 6% had evidence of mixed ischaemic and non-ischaemic injury during the early post-infection recovery period.[64] Finally, in patients with a high pre-test probability for acute myocardial injury, CMR may improve diagnostic specificity, guide management decisions, and affect the prognosis[12,37] (Figure 5).

Another, albeit less frequent imaging manifestation in patients with acute COVID-19 can be focal or global inducible perfusion deficits during stress CMR.[43,65] This may be related to microvascular dysfunction, endothelial inflammation, or micro- and macrovascular fibrin thrombosis, observed in moderate to severe acute COVID-19.[65-68] Another suggested mechanism for myocardial ischaemia may be impaired flow-mediated epicardial coronary dilatation.[69]

Estimating the prevalence of myocardial infarction during acute COVID-19 is challenging. However, several studies have indicated an increased risk of myocardial infarction during the acute phase of COVID-19.[70,71] Saad et al demonstrated that myocardial infarctions associated with COVID-19 have higher mortality rates compared to cases without COVID-19 evidence.[72] In a retrospective multicentre study in 1047 patients with PCR-confirmed COVID-19 infection, Vidula et al demonstrated that 6.7% of participants had ischaemic myocardial injury, with 1.9% showing an acute ischaemic pattern on CMR.[73] CMR evidence of COVID-related ischaemic myocardial injury with characteristic subendocardial or transmural LGE was similar to other causes.[41,56,73]

Biventricular thrombus formation, as a rare cardiovascular complication of acute COVID-19, has been described after myocardial infarction or a prothrombotic state.[74,75] While cardiac thrombi are typically detected in echocardiography images, contrast-enhanced CMR has the highest sensitivity and specificity for LV thrombus detection, surpassing TTE, and transoesophageal echocardiography.[12,76]

Right ventricular dysfunction with cavity dilation, impaired RV strain, or depressed systolic function can be identified using CMR or echocardiography in about 40% of patients with COVID-19.[77] RV impairment is correlated

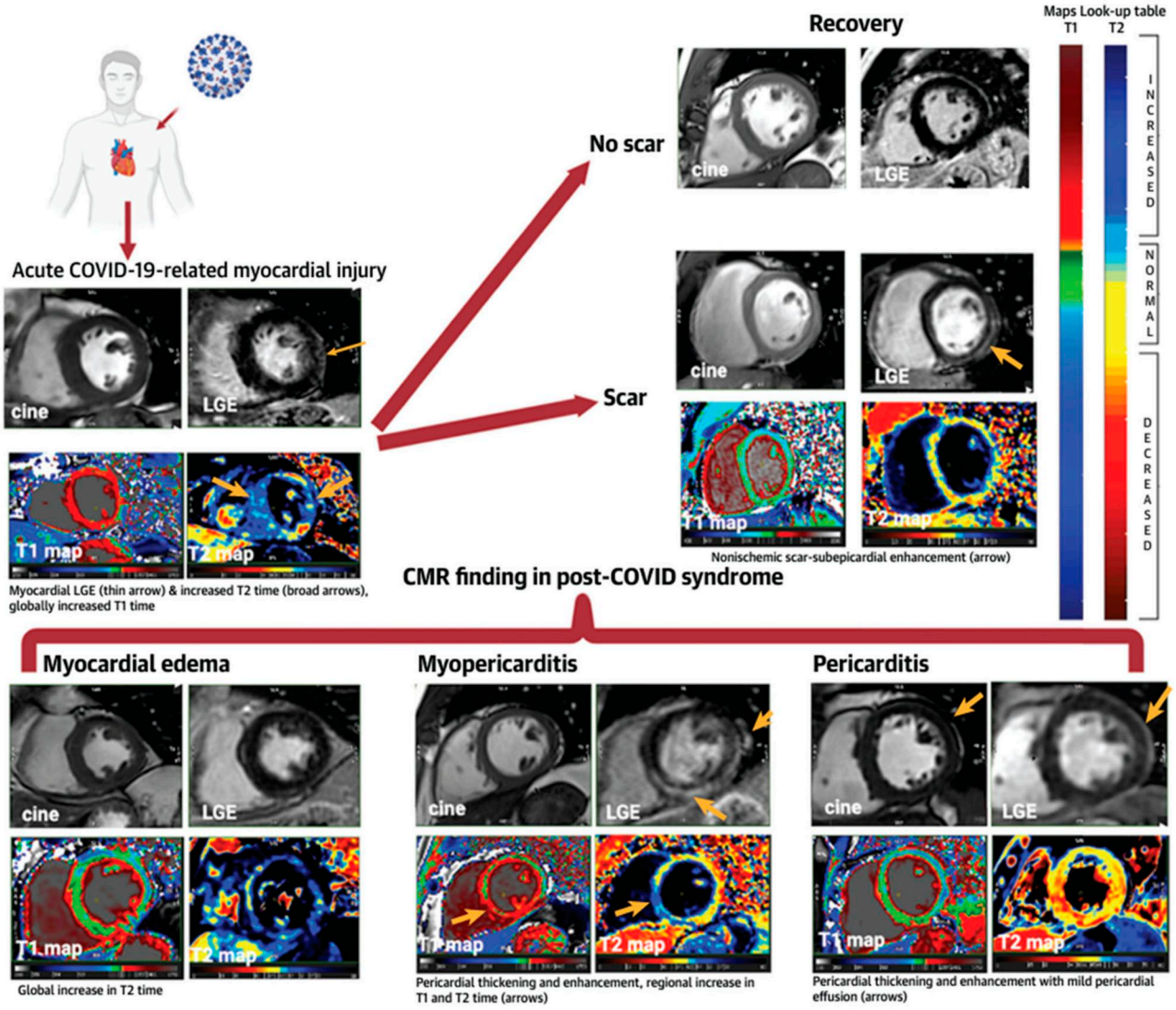

Figure 3. Summarized illustration of CMR findings of cardiac involvement in COVID-19.[37]

with an increased incidence of myocardial damage in COVID-19 and has been recognized as a predictor for adverse outcomes.[78-80] Although CMR is the gold standard method for assessing RV size and function (Figure 4), multiparametric CMR may be useful for identifying RV myocardial inflammation during acute myocarditis.[81] RV dysfunction has been shown to indicate an impaired prognosis in myocarditis.[82]

Several studies have indicated a significant increase in the prevalence of stress-induced cardiomyopathy or Takotsubo syndrome (TTS) during the COVID-19 pandemic.[83] Researchers found that during the pandemic, 7.75% of patients with acute coronary syndrome were diagnosed with TTS, in contrast with only 1.5%-1.8% before the pandemic.[84] TTS is characterized by a characteristic pattern of acute, transient regional left ventricular systolic dysfunction. The aetiology of TTS has been attributed to psychological or physical stress leading to a surge of catecholamines, causing an acute inflammatory response in tissues with a dense presence of adrenergic receptors, with myocardial oedema being a hallmark feature of Takotsubo cardiomyopathy.[21,85] Multiparametric CMR, including cine images, LGE, as well as T1- and T2-mapping, is more accurate than echocardiography (or other imaging modalities), in effectively distinguishing TTS from conditions like acute myocardial infarction and myocarditis.[86]

CMR in athletes after recovery from COVID-19

Given that acute myocarditis accounts for approximately 10% of sudden cardiac deaths in young and active adults (aged < 35 years),[87] there is a significant concern regarding myocardial injury and subsequent risk of adverse cardiovascular events in competitive athletes following COVID-19 infection. Available data shows lower rates of CMR findings of cardiac injury in the athletes.[37] In a study of 145 student-athletes with mild to moderate symptoms during acute infection, only two patients (1.4%) had CMR findings consistent with myocarditis according to the updated LLC[55] (Figure 6). A comprehensive multicentric study by Daniels et al,[88] encompassing 13 universities, involved 2461 athletes, with 1597 undergoing CMR. Clinically, myocarditis was diagnosed in 37 out of the 1597 athletes (2.3%). Of these 37, 31 exhibited CMR findings consistent with the LLC.

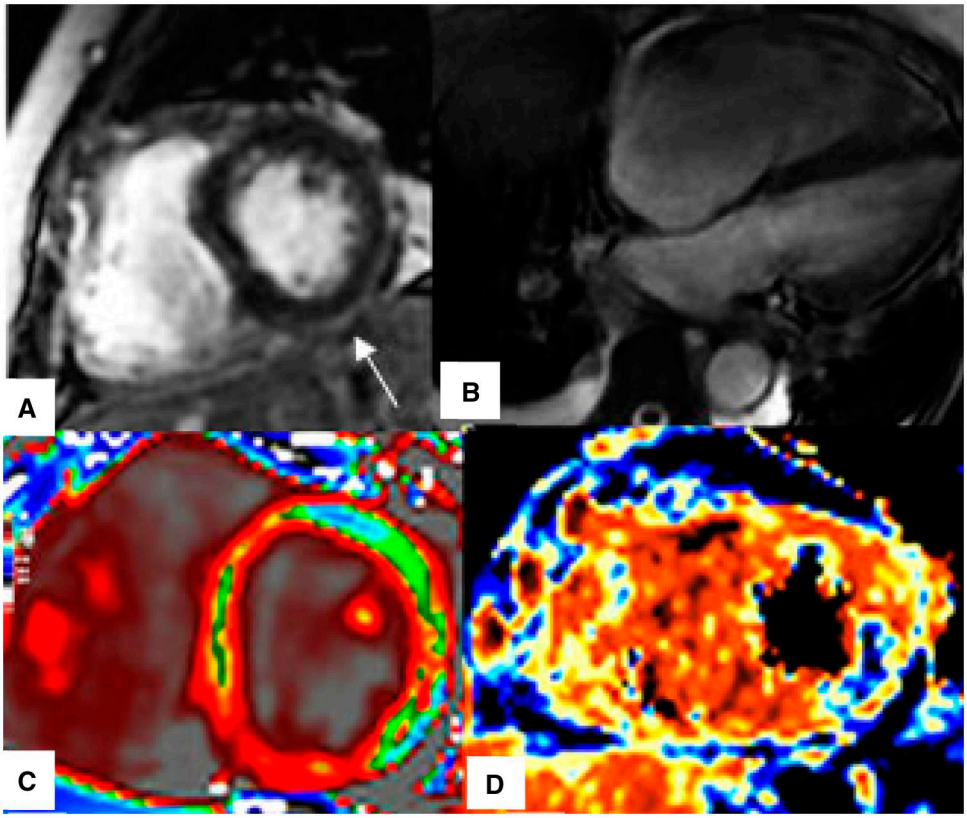

Figure 4. CMR findings of myocardial injury in patients with acute severe COVID-19. (A) LGE, Non-ischaemic subepicardial enhancement involving inferior and inferolateral segments (arrow), (B) Four-chamber SSFP, Dilated right ventricle, (C) T1 map, Diffusely increased T1-relaxation times, (D) T2 map, Focal high T2-values in the mid inferolateral wall (blue colour).

Interestingly, the rate of abnormal CMR results ranged from 0% to 7.6% across the participating institutions.[88] These results suggest that, while the prevalence of myocarditis-like manifestations on CMR in competitive athletes following COVID-19 is relatively low, there is uncertainty about the actual numbers, given the significant variation in reported rates across different studies, ranging from 0% to 15%.[37,89] A contributing factor to false-positive CMR findings in endurance athletes may be the interpretation of LGE at the RV insertion, a non-specific finding with a prevalence ranging from 0% to 26%,[37,90] as post-inflammatory injury.

CMR findings in children with multisystem inflammatory syndrome after COVID-19

Multisystem inflammatory syndrome in children has been recognized as a severe complication in a small proportion of children a few weeks after SARS-CoV-2 infection.[20,91] Similar to Kawasaki's Disease, it is characterized by a severe clinical presentation, often with circulatory shock, myocardial depression, and coronary involvement.[92] Several studies have shown that CMR may be an important diagnostic tool to identify a subset of patients at risk for cardiac sequelae and more prone to myocardial damage.[18,20,92] A large multicentre study found that out of 111 patients who met the WHO criteria for MIS-C and showed clinical signs of cardiac involvement, 20 (18%) fulfilled the LLC, which was associated with a worse prognosis.[18] Another important cardiac complication related to MIS-C is coronary artery dilatation or aneurysm, which has been reported in 6%-24% of cases.[20,93,94] CMR has been established to detect the presence of coronary artery aneurysms, wall motion abnormalities, reversible ischaemia, and myocardial infarction without the use of radiation or invasive procedures.[95]

COVID-19-vaccine-related cardiac injury

Following the initial case series of myocarditis in young men in Israel found after Pfizer-BioNTech mRNA SARS-CoV-2 vaccination, numerous international studies have shown an increased rate of myopericarditis following mRNA-based COVID-19 vaccination.[96-98] A systematic review reported an overall incidence of myopericarditis post mRNA COVID-19 vaccination of 18 cases per million doses, with significant variations depending on age, sex, and vaccine type and dose.[99] The highest risk of cardiac injury is observed among male adolescents and adults aged 18-25 years, specifically after their second COVID-19 mRNA vaccine dose within 1-7 days of vaccination.[100,101] The pathophysiology of vaccine-induced myopericarditis remains unclear. It has been suggested that vaccine-induced cardiac injury might be due to a hypersensitivity reaction, given its typical development post-second vaccine dose, or molecular mimicry between the SARS-CoV-2 spike protein and cardiac self-antigens.[102] CMR has a key role in the diagnosis of vaccination-related cardiac injury, and a systemic review showed that more than two-thirds of patients with clinically suspected post-COVID-19-vaccination myocarditis meet the LLC, consistent with acute myocardial inflammation.[103] Common CMR

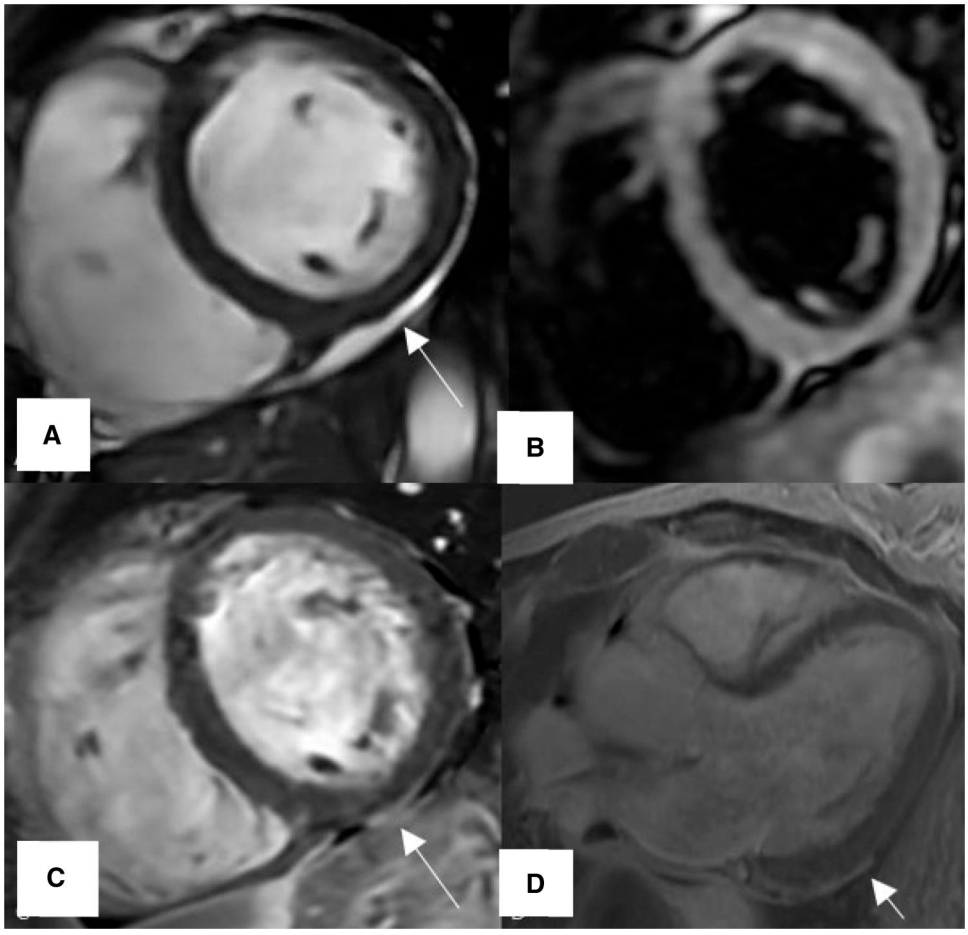

Figure 5. 41-year-old woman with COVID infection presented with dyspnoea, palpitation and fatigue and mild perimyocardial involvement. (A) short axis SSFP, small pocket of pericardial fluid as high signal intensity located adjacent to the basal inferior wall of the LV (arrow). (B) T2-weighted, Regional signal increase in the mid anterior, anterolateral segments to suggest myocardial oedema. (C, D) LGE images, Regional subepicardial and adjacent pericardial enhancement of the basal inferior, inferolateral segments, adjacent to a pocket of effusion (white arrows).

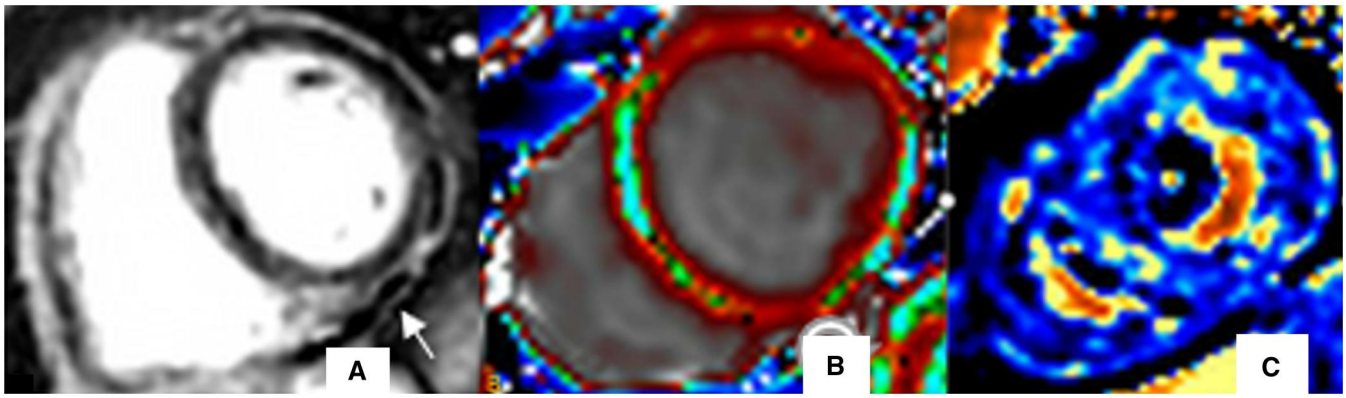

Figure 6. CMR findings in a young male athlete after recovery from COVID-19, clinically presented with persistent fatigue, malaise and shortness of breath. The CMR shows signs of persisting inflammation. (A) LGE-PSIR, Small pockets of pericardial effusion as low signal intensity region (arrow) along the mid-lateral and inferior walls associated with enhancement of the adjacent pericardium, mild subepicardial enhancement of the mid-inferior and inferolateral segments is visible. (B) T1 map, Increase in native T1-times in the anterior, anterolateral, and inferoseptal segments (red colour). (C) T2 map, Globally prolonged T2-relaxtaion times (blue colour). Abbreviation: PSIR = Phase Sensitive Inversion Recovery.

characteristics observed in patients with myocarditis post-COVID-19 vaccination were similar to those seen in myocarditis from other aetiologies. This includes subepicardial LGE, predominantly involving the basal inferolateral wall and associated myocardial oedema (Figure 7). In addition, small pericardial effusion may also be present, sometimes accompanied by enhancement of the adjacent pericardium.[56,104,105] While most patients with myocarditis following COVID-19

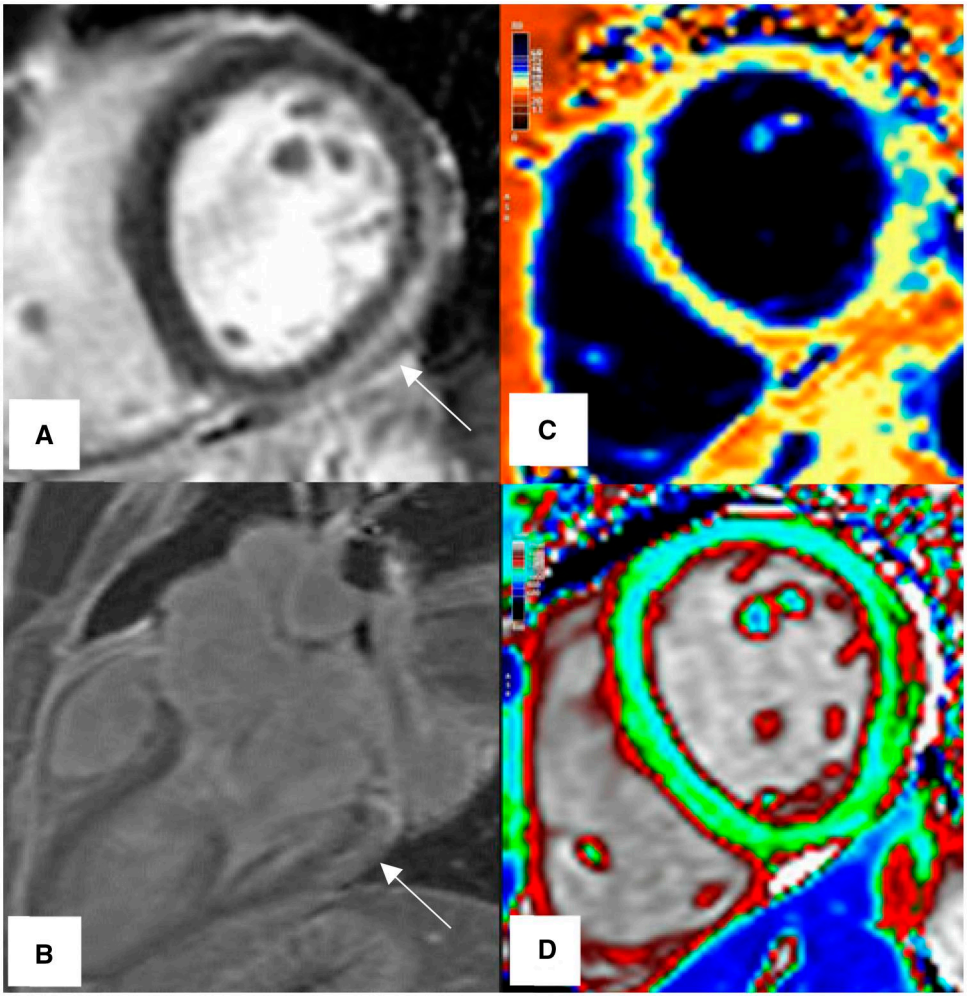

Figure 7. CMR imaging findings in a 27-year-old man with myocarditis after a second COVID-19 mRNA vaccine dose. (A, B) LGE images, Subepicardial enhancement involving basal to mid inferior, infero and anterolateral segments (white arrow). (C) T2 map, with focal myocardial inflammation manifesting as high T2-values in the mid-lateral wall (blue colour). (D) T1 map, with focal high T1-value in the subepicardial part of the mid lateral wall (red colour).

vaccination demonstrate favourable short to mid-term outcomes, data on long-term follow-up are scarce. A small case series by Patel et al indicates that acute myocarditis following mRNA-based COVID-19 vaccination showed CMR-based evidence of myocardial recovery within 3-6 months, while some mild abnormalities may persist.[106]

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Conflicts of interest

None declared.

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