REVIEW

# Multi-modality diagnosis of cardiac involvement in COVID-19 patients

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**Abstract:** The Coronavirus disease 2019 (COVID-19) pandemic, caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV2), exhibits a wide spectrum of manifestations, from asymptomatic presentations to acute respiratory failure, myocardial injury, arterial or venous thrombosis, multiorgan failure, and death. Although COVID-19 mainly disrupts the respiratory syndrome, it has been shown to have detrimental impact on cardiac morphology and function, resulting in a broad range of cardiovascular complications and poor outcome, increasing morbidity and mortality of these patients. This review will summarize the knowledge on characterization of myocardial injury in COVID 19, by using cardiac biomarkers, electrocardiographic, and cardiac multi-modality imaging findings for an early and accurate diagnosis, proper management, correct treatment, and follow-up of COVID-19 patients.
**Keywords:** COVID-19, myocardial injury, cardiac biomarkers, electrocardiogram, cardiac imaging.

**Rezumat:** Pandemia COVID-19, determinată de noul coronavirus al sindromului respirator acut sever (SARS-CoV-2) include o serie largă de manifestări, de la forme asimptomatice până la insuficiență respiratorie acută, injurie miocardică, tromboză arterială sau venoasă, insuficiență multiplă de organ și deces. Deși maladia COVID-19 afectează frecvent aparatul respirator, s-a dovedit că aceasta are un impact semnificativ și la nivel cardiac, cu apariția de complicații cardiovasculare variate asociate cu prognostic negativ și morbi-mortalitate crescută. Acest review sintetizează principalele date despre injuria miocardică secundară maladiei COVID-19, pe baza biomarkerilor cardiaci și a modificărilor electrocardiografice și imagistice multi-modale, cu scopul de a obține un diagnostic acurat cât mai precoce, un management optim, o schemă terapeutică corectă și o urmărire corespunzătoare a pacienților cu patologie de tip COVID-19.
**Cuvinte cheie:** COVID-19, injurie miocardică, biomarkeri cardiaci, electrocardiogramă, imagistica aparatului cardiovascular.

## INTRODUCTION

Since March 2020, the coronavirus disease (COVID-19) pandemic has become an important public health issue leading to increased morbidity and mortality[1]. Although initially it was thought that severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) causes only lung injury (Figure 1), several studies discovered that cardiac complications may occur in 10-20% of patients, making the management of the disease more difficult, with worse outcome[2]. Moreover, COVID-19 patients with pre-existing cardiovascular disease have a higher risk of in-hospital mortality[2]. Myocardial involvement could be the cause of cardiovascular complications of the viral disease or exacerbation of preexisting cardiovascular diseases, leading to acute coronary syndromes (ACS), myocarditis, pericarditis, myocardial injury, arrhythmias, and pulmonary embolism (PE)[3,4].

Electrocardiogram, various imaging techniques, and cardiac biomarkers are essential for the early diagnosis and timely management of cardiac involvement in COVID-19 patients in order to improve survival and long-term prognosis[4]. Thus, this review will focus on the electrocardiographic (ECG), imaging and biomarkers changes in SARS-CoV-2 disease with cardiac involvement.

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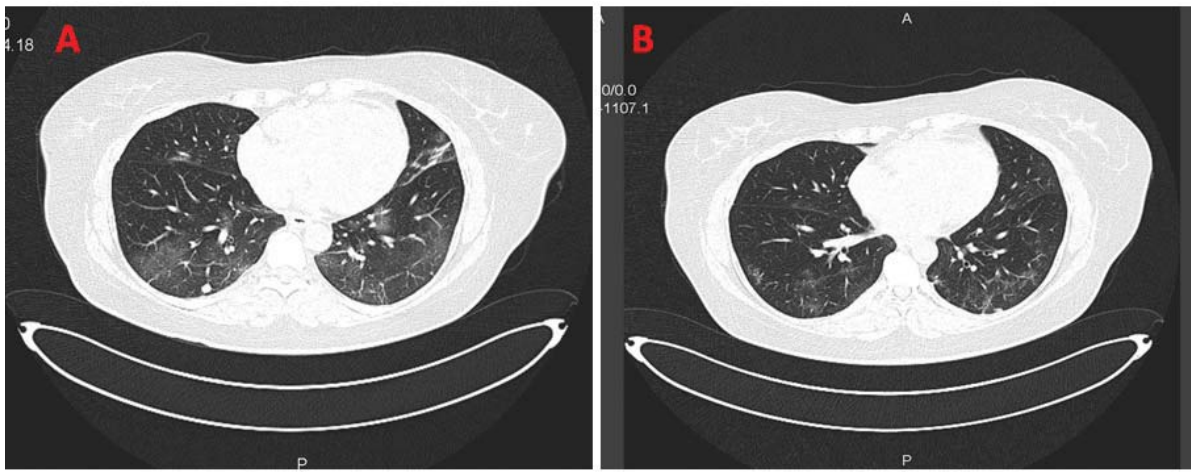


**Figure 1.** Thoracic computed tomography in a female patient with a moderate form of COVID-19. Bilateral infiltrates predominant at the level of the posterior and inferior pulmonary lobes, with a typical ground-glass aspect. Panel A. Acute phase of the disease; Panel B. Follow up at one month.

## Diagnosis of cardiac involvement in COVID-19 patients

1. ECG abnormalities in COVID-19 patients are nonspecific and dynamic, and reflect a wide spectrum of cardiovascular complications such as ACS, arrhythmias, acute myocarditis, pericarditis or PE (5). Therefore, the main ECG changes are:
   - ST-T abnormalities: ST-elevation in leads DII, DIII, aVF and DI, aVL, ST-depression in leads V1-V6, deep T wave inversion in precordial leads or diffuse concave ST-elevation[5-7];
   - QTc prolongation due to COVID-19 medication (hydroxychloroquine and azithromycin) that can lead to secondary torsades de pointes[8,9];
   - arrhythmias: atrial fibrillation in 8.5% of patients, ventricular tachycardia in 3.5% of patients and Brugada pattern - two cases reported in the United States[9-11];
   - S1Q3T3 pattern and sinus tachycardia: in PE patients[12].
2. Cardiac biomarkers. COVID-19 is a systemic infection characterized by abnormalities of inflammatory, hematologic, thrombotic, and cardiac biomarkers[13]. The mechanisms underlying acute myocardial injury in SARS-CoV2 disease are incompletely elucidated and include increased myocardial consumption in response to viral infection, inflammatory response due to cytokines, and thrombogenic environment due to platelet activation, endothelial dysfunction and direct cytotoxic myocardial damage[13]. Thus, increased N-terminal pro-brain natriuretic peptide (NT-pro-BNP) and high-sensitivity cardiac troponin (hs-cTn) may not necessarily diagnose heart failure or myocardial infarction, but they correlate with a worse prognosis of COVID-19 patients[13].
   a) Troponin. As a quantitative marker of cardiomyocyte injury, increased troponin levels reveal acute myocardial injury, associated or not with pre-existing cardiovascular disease, in 25% of COVID-19 patients hospitalized in the intensive care unit (ICU)[13]. Moreover, elevated hs-cTn correlates significantly with 28-day mortality, but with lower cut-offs than the cut-off used for cardiac disease in non-COVID-19 patients[14].

Isolated mild elevation of hs-cTn, below 3 times the upper limit of normal (ULN), may not require work-up or treatment for myocardial infarction unless strongly suggested by symptoms and ECG changes[14]. However, the troponin rise is explained by the combination of possible pre-existing cardiac diseases and/or associated acute myocardial injury in SARS-CoV2 disease[14]. Significant elevation of hs-cTn, more than 5 times the ULN, indicates a severe form of COVID-19, with shock, severe respiratory failure, tachycardia, systemic hypoxemia, myocarditis, Takotsubo syndrome or myocardial infarction[13]. Echocardiography should be considered for an initial diagnosis and to establish prognosis in COVID-19 patients[14].

   b) NT-pro-BNP, a quantitative biomarker of hemodynamic myocardial stress and heart failure, is frequently elevated among patients with severe inflammatory and respiratory diseases[14]. Increased level of NT-pro-BNP in COVID-19 patients represents a combination between presence or

extent of pre-existing cardiac disease and acute hemodynamic stress related to SARS-CoV2 disease[14]. Similar to troponin, NT-pro-BNP is an independent risk factor for adverse outcome in patients with severe forms of COVID-19[15]. A NT-pro-BNP cutoff value of 88.64 pg/ml, lower than the threshold used to diagnose heart failure, predicts in-hospital death of COVID-19 patients with a sensitivity of 100% and a specificity of 66.67%[15].

c) High D-dimer level suggests thrombin formation, disseminated intravascular coagulation associated with shock or an acute response in systemic infections or inflammations[16]. Thus, markers of activated coagulation or impaired fibrinolysis might contribute to acute myocardial injury, affecting coronary capillaries[16]. Although D-dimer has a low specificity for the diagnosis of thrombosis, patients with COVID-19 pneumonia and increased D-dimer levels have a greater probability of PE, regardless of the clinical suspicion[16,17]. Moreover, elevated D-dimer level is associated with poor outcome and increased mortality in SARS-CoV2 disease, regardless of the occurrence of thromboembolic disease[17]. A value of more than 2.01 μg/mL is associated with high in-hospital mortality, especially in elderly COVID-19 patients with diabetes[17].

## CARDIAC IMAGING

### a) Echocardiography

**I. Risks and guideline recommendations.** Cardiac involvement occurs in 20% to 30% of hospitalized COVID-19 patients and in at least half of the patients with preexisting cardiovascular diseases[18]. Echocardiography is a non-invasive, feasible, reproducible, and widely available imaging modality that provides information on myocardial dysfunction due to SARS-CoV-2 infection[18]. Thereby, it plays a pivotal role in the early diagnosis, management, and outcome of various COVID-19 cardiovascular complications[19]. However, in the current pandemic situation, cardiac ultrasound poses a high risk of exposure for healthcare workers. Hence, echocardiographic studies should be performed according to local standards and protocols, aiming at prevention of virus spread[19].

Current guidelines recommend a rigorous selection of COVID-19 patients requiring cardiac ultrasound, to which the management strategy could be modified by the results, providing clinical benefit[20]. In addition, the guideline highlights the utility of specific echocardiographic modalities, such as focused cardiac ultrasound, point-of-care cardiac ultrasound, and critical care echocardiography for the evaluation of cardiac involvement in SARS-CoV-2 disease[20]. These techniques evaluate in a very short time, important insight of chamber geometry, cardiac function and presence of pericardial effusion[20]. The use of hand-held device might be beneficial to decrease the risk of infection, as they are easier to disinfect, and the use of telemedicine to evaluate acquired echocardiographic images by an expert is also recommended[20].

**II. Main echocardiographic findings.** In SARS-CoV-2 disease, cardiac dysfunction diagnosed by biomarkers and echocardiography is present from admission in 49% of patients, while 70% of patients develop acute myocardial injury even after 14 days after intensive care unit (ICU) admission[21,22]. Although nonspecific, major echocardiographic findings in COVID-19 patients with cardiac involvement are left ventricular (LV) and right ventricular (RV) dysfunction, regional wall motion abnormalities (Figure 2) and pericardial effusion[21]. These cardiac alterations suggest a broad spectrum of cardiovascular diseases ranging from ACS, Takotsubo syndrome, acute myocarditis, cardiac tamponade to acute heart failure, PE, and acute cor pulmonale[22].

**III. Ventricular function assessment.** Recently, ECHOVID-19 study identified both LV and RV dysfunction in hospitalized COVID-19 patients compared with matched controls during a follow-up of 40 days. Moreover, in the same study, decreased systolic tricuspid annular velocity (TAPSE) and longitudinal strain (LS) were the most powerful echocardiographic parameters correlated with COVID-19 mortality[23]. Furthermore, when comparing COVID-19 patients without cardiac involvement to patients with acute myocardial injury, for the latter were identified, in addition to increased biomarkers, echocardiographic changes, such as RV dilatation (26% of patients), LV wall motion abnormalities (23% of patients), decreased LV ejection fraction (LVEF) (18% of patients), diastolic dysfunction grade II or III (13% of patients), and pericardial effusion (7% of patients)[24]. In addition, patients with myocardial dysfunction and ultrasound abnormalities have a higher in-hospital mortality (31%) versus patients with cardiac involvement and no echo changes (18%) or patients without myocardial injury (5%)[24]. Data on the occurrence of organic valvular heart disease or endocarditis induced by SARS-CoV2 infection are

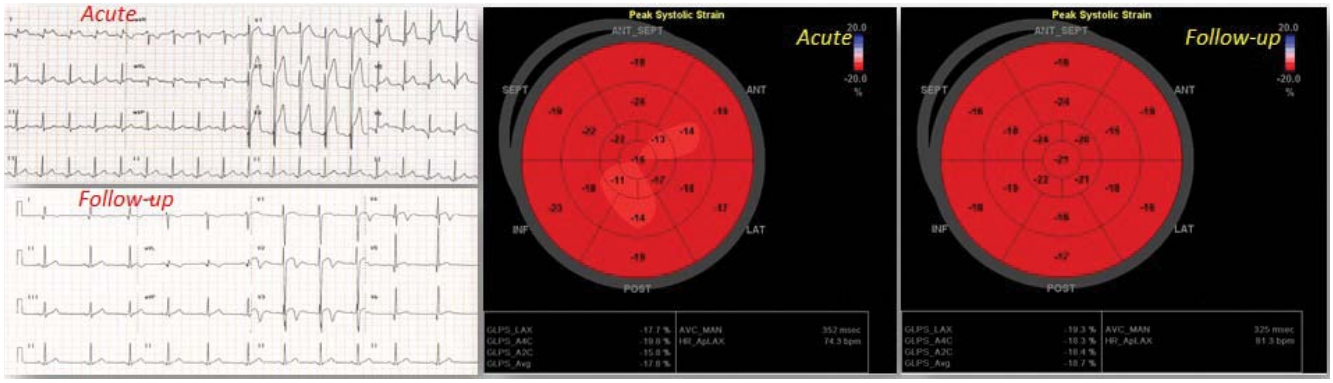


**Figure 2.** Transthoracic 12 leads electrocardiogram and bulls eye images for the left ventricular strain in a young patient presenting with a moderate form of COVID 19 and chest pain. In the acute phase, troponin levels were increased, coronary angiography showed normal coronary arteries, and strain revealed left ventricular wall motion abnormalities. Follow-up showed the regression of the "STEMI-like" aspect of the ECG, and the regression of the wall motion abnormalities by strain analysis.

scarce; aggravated preexistent valvular disease may occur during acute heart failure and myocardial injury in COVID-19 patients[22].

**IV. Myopericardial dysfunction.** In 12% of patients with SARS-CoV2 disease, echocardiographic abnormalities suggestive of myocarditis are found (Figure 3)[25]. They are defined by *American Heart Association* as LVEF <50%, segmental wall motion deficits, LV wall thickening >10mm or pericardial effusion ≥5mm after excluding preexisting cardiac disease or ACS[25]. However, diagnosis of myocarditis as the cause of acute myocardial injury in COVID-19 patients is assessed with a limited number of endomyocardial biopsies or autopsies[26]. In patients with newly onset of acute heart failure or suspicion of myocarditis, Escher et al. detected for the first time SARS-CoV-2 genome using endomyocardial biopsy in five cases of acute myocarditis[27].

Approximately 5-7% of COVID-19 patients develop pericardial effusion, without correlation with the degree of myocardial involvement; cases with cardiac tamponade are scarce[28]. Pericardial fluid is often exudative and without virus, secondary to an inflammatory response rather than infectious[28].

Withal, during COVID-19 pandemic, abnormal echocardiographic findings like apical ballooning and basal hyperkinesia from Takotsubo cardiomyopathy is constantly increasing, comparing to pre-pandemic period. Possible mechanisms are high psychological distress, elevated sympathetic nervous system activity, cytokine storm, and endothelial dysfunction[29].

**V. Impact of RV dysfunction on outcome.** However, right heart is more sensitive and early affected compared to left-sided chambers in COVID-19 patients, due to lung tropism of the virus, inflammatory response with increased sympathetic tone, and

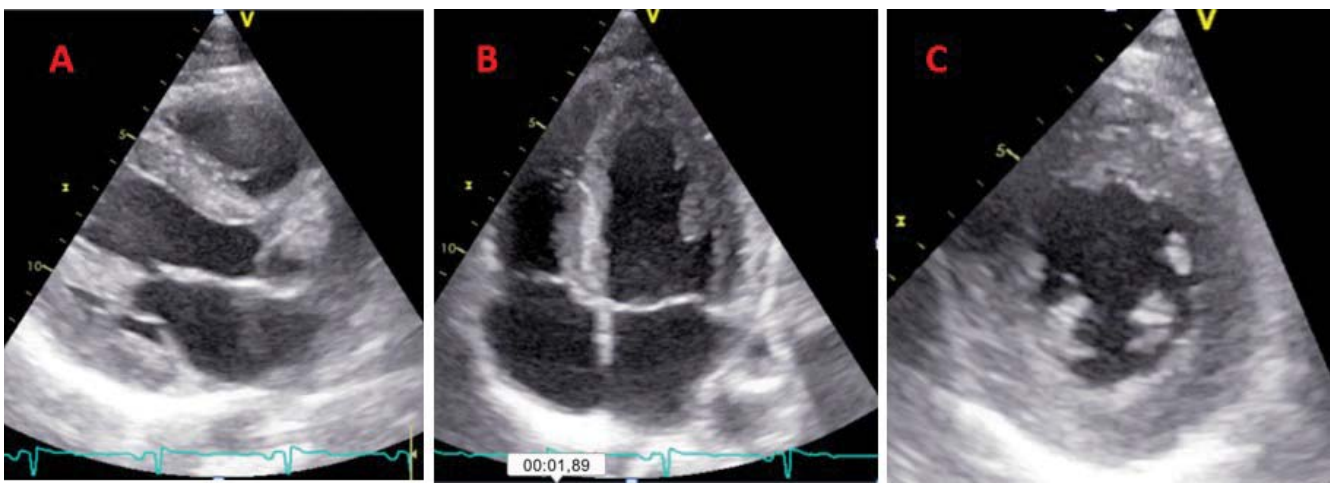


**Figure 3.** Two-dimensional echocardiographic images of the left ventricular walls edema in a patient with acute myocarditis and a severe form of COVID-19. Panel A. Parasternal long-axis view; Panel B. Apical 4 chamber view; Panel C. Short axis view.
LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

altered volume filling or use of ventilator therapies[21]. The major identified changes are RV dilatation with or without dysfunction in 39% of patients, while LV systolic and diastolic dysfunctions develop in only 10% of patients in response to the systemic disease or by ventricular interdependence[30,31]. Thereby, pulmonary velocity acceleration time, TAPSE and RV fractional area change, and inferior vena cava diameter are reduced with preserved Tei index[31]. Both RV dilatation and dysfunction are independent predictors for poor outcome and mortality in COVID-19 patients[32]. Often, serial echocardiographic assessments show further deterioration of RV parameters, explained by elevated pulmonary resistance pressures, secondary to microvascular lung thrombosis or acute respiratory distress syndrome (ARDS) in the course of SARS-CoV-2 infection[30]. Moreover, RV LS represents an important parameter for risk stratification and prognosis[24]. Thus, a RV LS below -23% represents an independent predictor for poor outcome and high mortality among COVID-19 patients[33].

PE occurs in 33% of COVID-19 patients and is associated with a higher mortality risk and cardiogenic shock comparing with non-PE patients[34]. When clinical suspicion is present, echocardiographic parameters, such as pulmonary ejection acceleration time <60ms, peak systolic tricuspid valve gradient <60mmHg, impaired contractility of the RV free wall compared to the RV apex, have a high predictive value in the diagnosis and management of PE[34,35].

ARDS is a complication of severe COVID-19, associated with high mortality; it requires mechanical ventilation with high positive end-expiratory pressure (PEEP) and prone positioning[36]. Patients with ARDS treated with PEEP are prone to developing RV failure, acute cor pulmonale with systolic and diastolic overload[37]. The main echocardiographic findings are right heart dilatation, end-systole paradoxical septal motion, and reduced RV global function[37].

**VI. Transesophageal echocardiography** has been extensively used for monitoring the evolution of mechanically ventilated patients and can be performed efficiently in the prone position in patients with ARDS[38]. RV assessment is mandatory, since an efficient method for improving RV function is prone positioning, which decreases pulmonary vascular resistance, RV pressure and volume overload, and improves RV preload[39]. RV free wall LS and Tei index are high-sensitive parameters of RV dysfunction and can be used for monitoring intubated COVID-19 patients under high-PEEP levels[40]. Furthermore, Repesse et al. suggested a RV-driven adjustment of PEEP level, thus reducing mortality due to RV failure in mechanically ventilated patients[37].

Currently, imaging data from follow-up of the patients recovered from COVID-19 and cardiac involvement is limited. However, cardiovascular assessment and standard echocardiography in the first six months after SARS-CoV2 disease is recommended for these patients in order to screen for post-residual myocardial damage, to establish the burden of long-term cardiac diseases, and to early initiate protective therapeutic measurements[24].

**CARDIAC MAGNETIC RESONANCE (CMR)** is the gold standard noninvasive imaging modality for quantification of volumes, systolic function and mass of heart chambers by a morphological and functional evaluation and an accurate tissue characterization[41].

I. Risks and guideline recommendations CMR is an essential tool for diagnosis and monitoring myocardial injury in COVID-19 patients[41]. Based on functional sequences, like cine white blood steady state free precession on the short and long axis of two-, three-, and four-chambers views, and tissue morphological characterization sequences such as T2 short tau inversion recovery (T2 STIR), T1 pre-contrast, and post-contrast mappings, T2 mapping and late gadolinium enhancement (LGE), CMR allows a differential diagnosis of ischemic and non-ischemic acute cardiovascular injury[41]. Moreover, in acute myocarditis, CMR is the method of choice that identifies with a high sensitivity focal or diffuse myocardial edema through T2 STIR, necrosis areas and fibrosis by LGE, diffuse expansion of extracellular volume fraction and hyperemia[41,42].

In order to reduce exposure risk during the COVID-19 pandemic, current guidelines recommend short CMR examinations, only when strongly indicated, and adapted to the patients' capacity of breath hold[43]. The main indications for CMR in active or convalescent phase of COVID-19 patients with cardiac involvement are heart failure, myocarditis, pericarditis, myocardial infarction, Takotsubo cardiomyopathy, and ventricular arrhythmias[43].

II. Main CMR findings in adults. CMR examination in patients recovered from SARS-CoV-2 disease with cardiovascular symptoms reveals myocardial edema in 54% of patients, and LGE in 31% of patients (Figure 4); more interesting, in all patients with CMR pathological findings, RV EF, cardiac index and stroke volume/body

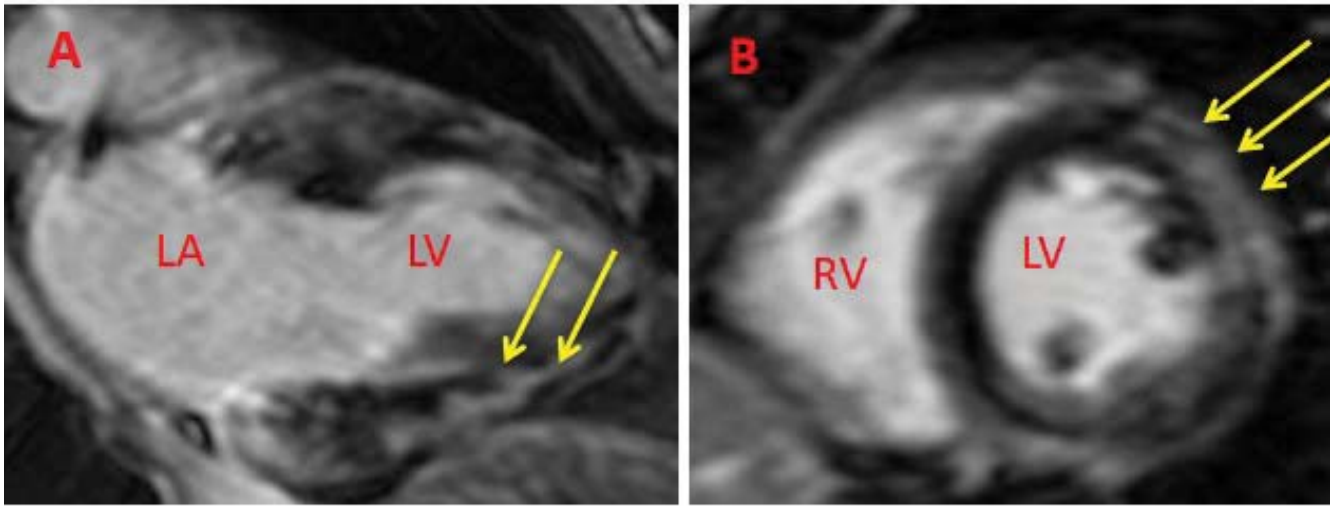


**Figure 4.** Cardiac magnetic resonance in a patient with COVID-19 and myocarditis. CMR shows late gadolinium enhancement (LGE) with endocardial spearing at the level of the lateral and infero-lateral walls in an apical 2 chamber view (panel A) and short axis view (panel B).
CMR, cardiac magnetic resonance; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

surface are impaired, suggestive of RV dysfunction[44]. On the contrary, even in patients recovered from COVID-19 and with no evidence of cardiac involvement during hospitalization, CMR shows increased T1, T2 and extracellular volume possibly explained by ongoing inflammation or fibrosis[44,45]. In addition, in the discharge day of twenty-nine COVID-19 patients with high troponin level at admission, using LGE and stress perfusion imaging, Knight et al. identifies non-ischemic heart disease in 38% of patients, ischemic heart disease in 17% of patients and both pathologies in 14% of patients, with preserved LV and RV EF[46]. The non-ischemic etiology is diagnosed based on the non-myocardial infarction LGE pattern, sparing the endocardium and without any correspondence with a coronary artery region[46]. In this group, LGE-myocarditis pattern is present in only 45% of the cases and a non-specific mid-wall LGE pattern is found in 18% of patients[46].

When compared to matched controls, competitive athletes that recovered from asymptomatic or mild forms of COVID-19 infection have increased myocardial T2 relaxation times in all segments, with inflammation or fibrosis in 9% of cases, without any changes on ECG or myocardial deformation[47]. Furthermore, they have increased mid-septal extracellular volume, similar with athletic controls[47]. Also, mild regional increase in T1 and T2 are found in 39% of COVID-19 athletes, 13% of healthy athletes and only 8% of normal subjects[47]. More than that, Rajpal et al. report a high rate of LGE (42%). This may correlate an increased risk of ventricular arrhythmias, poor outcome and suspended competitional activity[48].

III. Main CMR findings in children. In a series of four cases of children with multisystem inflammatory syndrome and Kawasaki disease-like due to COVID-19, CMR demonstrates diffuse myocardial edema and hyperemia, but not focal necrosis, fibrosis or coronary artery abnormalities[49]. On the contrary, Wacker et al. reports a mild reduction of LVEF and no signs of myocarditis in the acute phase of the disease, but one month after COVID diagnosis, coronary artery dilatation is found, suggestive of post-infectious vasculitis[50].

Currently, data about CMR findings in acute or recovered COVID-19 patients come from isolated or small cohorts of case reports. Future research is mandatory in order to define adapted diagnostic protocols, prognostic parameters, and management of COVID-19 patients with CMR abnormalities.

**COMPUTED TOMOGRAPHY ANGIOGRAPHY (CTA)** is the recommended initial non-invasive imagining modality for the diagnosis of coronary artery disease (CAD) in patients with chronic coronary syndromes, in whom the disease cannot be excluded only by clinical evaluation[51]. Moreover, CTA can be used in ACS as an alternative to invasive coronary angiography (ICA) when cardiac biomarkers and ECG are normal or inconclusive and there is a low-to-intermediate probability of CAD[52]. In COVID-19 patients presenting with acute chest pain and elevated cardiac enzymes, the diagnostic work- up is guided by clinical judgment and pretest probability of CAD, based on sex, age, previous history of CAD, and cardiovascular risk factors, and CTA is recommended in patients with intermediate risk of ACS[53]. For patients with

SARS-CoV-2 disease and chronic coronary syndromes, CTA may be postponed in the acute phase of the viral disease due to high patient contagiousness, medical workers exposure, and the non-urgent indication of the cardiac investigation[53].

When myocardial injury is detected, associated with myocardial thickening and wall motion abnormalities on echocardiography, myocarditis should be considered and investigated. Cardiac computed tomography (CT) might assess myocardial tissue characterization by completing the protocol with delayed- iodine enhanced scan or extracellular mapping and CTA for the exclusion of obstructive CAD[53]. However, CMR remains the gold standard imaging method for the diagnosis of myocarditis[53]. In 4.8% of COVID-19 patients, cardiac CT is a useful diagnostic tool for the diagnosis of pericarditis, associated or not with myocarditis[54,55]. Cardiac tamponade is a rare first manifestation of the SARS-CoV-2 disease, diagnosed by cardiac CT[55].

In addition, in COVID-19 patients, a high coronary calcium score, a marker of CAD, correlates with an unfavorable clinical outcome, represented by a severe form of the SARS-CoV-2 infection, transfer to ICU or death[56]. CTA can combine coronary artery, pulmonary artery, and thoracic aorta assessment by using a triple rule-out protocol for the rapid exclusion of severe acute pathologies with increased mortality, such as ACS, PE or aortic dissection[57]. In patients with different severity of respiratory symptoms, with high levels of cardiac biomarkers and D-dimer levels, a modified triple rule-out scan protocol with focus on lung parenchyma instead of the thoracic aorta as the third step of examination, may solve different clinical suspicions in just one stage[57]. Moreover, quadruple rule-out examination may be used, by adding delayed iodine enhanced scan in order to identify areas of myocardial fibrosis or necrosis[57].

**INVASIVE CORONARY ANGIOGRAPHY (ICA).** Since the beginning of COVID-19 pandemic, health care services went through a complex rearrangement, all efforts being dedicated to ensure the standard of care and early access to the catheterization laboratory for patients with SARS-CoV-2 infection and acute cardiovascular disease[58]. However, clinical status of patients, severity of the respiratory symptoms, and the balance between benefit and risk should be carefully evaluated when considering the indication of invasive coronary strategy[58].

The use of ICA in patients with SARS-CoV-2 is recommended to those with hemodynamic instability due to acute myocardial infarction, cardiogenic shock and cardiac arrest[58]. In addition, ICA and primary percutaneous coronary intervention remain the standard of care for COVID-19 patients with ST-elevation myocardial infarction (STEMI), high-risk non-STEMI or unstable angina[53,58]. Moreover, coronary intravascular imaging or left ventriculography add value to ICA for the differential diagnosis of myocardial infarction with non-obstructive arteries syndrome or Takotsubo cardiomyopathy[53,58]. Case et al. reports that only 20% of COVID-19 patients with acute myocardial infarction benefit from ICA and primary percutaneous intervention. Moreover, when compared with patients without SARS-CoV-2 disease, COVID-19 patients and ACS are older, with more comorbidities, and have a higher significant in-hospital mortality (27.9% versus 3.7%)[59].

Conclusions. SARS-CoV-2 infection has been spreading rapidly worldwide, with a crucial impact on health care services. Although respiratory syndrome prevails, COVID-19 affects also cardiovascular system by several mechanisms, increasing morbidity and mortality of these patients. Integration of cardiac biomarkers, electrocardiographic changes, and multi-modality imaging methods is essential for the early detection of cardiac damage, assessment of cardiovascular involvement extension, supporting the differential diagnosis of myocardial injury patterns and thorough management and follow-up of COVID-19 patients. Clinical and hemodynamic status, the severity of respiratory symptoms, comorbidities, as well as the availability and potential benefit on further management of patients with SARS-CoV-2 infection should be carefully taken into account, when considering the indication, benefit, and risk of different imaging modalities. Efforts should be targeted at collating multicenter experience into registries and elaborating specific assessment algorithms and protocols, in order to minimize exposure.

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**Abbreviations**

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| ACS | acute coronary syndrome; |
| ARDS | acute respiratory distress syndrome; |
| CAD | coronary artery disease; |
| CTA | computed tomographic angiography; |
| CMR | cardiac magnetic resonance; |
| COVID-19 | coronavirus disease 2019; |

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| CT | computed tomography; |
| ECG | electrocardiography; |
| LS | longitudinal strain; |
| hs-cTn | high-sensitivity cardiac troponin; |
| ICA | invasive coronary angiography; |
| ICU | intensive care unit; |
| LGE | late gadolinium enhancement; |
| LV | left ventricle; |
| LVEF | left ventricular ejection fraction; |
| LS | longitudinal strain; |
| NT-pro-BNP | N-terminal pro-brain natriuretic peptide; |
| PE | pulmonary embolism; |
| PEEP | positive end-expiratory pressure; |
| RV | right ventricle; |
| SARS-CoV2 | acute respiratory syndrome coronavirus 2; |
| STEMI | ST- elevation myocardial infarction; |
| TAPSE | systolic tricuspid annular velocity; |
| T2 STIR | T2 short tau inversion recovery; |
| ULN | upper limit of normal. |

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