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REVIEW ARTICLE

Cardiovascular Sequelae of Covid-19 Infection in Post-Acute Phase

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ABSTRACT

The wide array of clinical manifestations of the ongoing COVID-19 pandemic due to the SARS-CoV-2 infection continues to be a big puzzle to the healthcare professionals. The picture is becoming complex with appearance of new strains and widespread vaccination against the viral infection. The acute cardiovascular manifestations are diverse; but have been fairly well-documented. But the mechanism behind the persistent symptoms like dyspnea, fatigue, chest pain and other symptoms in some of the survivors, weeks and months after the onset of initial infection are poorly understood. The post-acute and long-term cardiovascular impact of COVID-19 affection on heart and other systems is not fully clear. As the survivors after an acute episode are fast growing in number, managing them is emerging as a new public health concern.

A recent study on cardiovascular outcomes of COVID-19 in post-acute phase by Xie et al in a cohort of 153,760 survivors of COVID showed that these individuals are at substantial risk of CV disorders like thromboembolic problems, arrhythmias, ischemic and non-ischemic myocardial injury, pericarditis, myocarditis and heart failure irrespective of the intensity of the initial infection. Currently, defining cardiovascular risk and predicting the long-term consequences in COVID survivors are issues of priority for the researchers and clinicians alike. The cardiac MRI is emerging as a useful tool to evaluate the myocardial damage in post-acute phase of COVID infection. This review is an attempt to analyze the existing knowledge and bring out the potential gaps in the understanding of these challenging issues after acute phase of COVID is over.

Keywords: SARS-CoV-2, long-COVID, myocardial injury, heart failure, Covid-heart

Introduction

CARDIOVASCULAR SEQUELAE OF COVID-19 INFECTION IN POST-ACUTE PHASE

Severe Acute respiratory Syndrome Coronavirus 2 (SARS-CoV-2) began in December 2019, spread across the globe as a pandemic in a short time and now appears to be settling down as an endemic. Nearly 30 months passed since WHO declared it a pandemic; but the spectrum of clinical manifestations of the SARS-CoV-2 infection continues to puzzle the healthcare professionals and the medical researchers. The picture is becoming complex with appearance of new strains and wide spread vaccination against this viral infection. The acute cardiovascular manifestations had been diverse but have been well-documented[1]. But the long-term impact of COVID-19 affection on heart and other systems and the means to identify the most vulnerable individuals remains unclear. The survivors after an acute episode are fast growing in number. The cardiovascular impact in the post-acute-phase of an episode of severe Covid infection deserves further research and analysis. This review is an attempt to analyze the existing knowledge in this regard and bring out the potential gaps in the understanding of new challenging issues long after COVID.

ACUTE CARDIAC EFFECTS:

Depending on the population studied, as high as 50% of hospitalized patients have elevated cardiac biomarkers, which may be reflection of myocyte injury and vascular inflammation[2]. Early studies reported 'myocarditis' in a good number of patients, based on the rise of troponins or imaging features in the acute phase[2-4]. There are good number of case-reports of fulminant myocarditis, new onset heart failure, stress cardiomyopathy, acute myocardial infarction (plaque-instability or demand-supply mismatch), arrhythmias and arterial and venous thrombotic disorders in hospitalized patients of Covid-19[5]. However, subsequent observations confirmed that it incidence of myocarditis was not very alarming as reported in earlier days. It was realized that any rise in Hs-troponins should not be labeled as myocarditis and the reports have to be interpreted in view of the clinical context[6]. Large epidemiological studies estimated that myocarditis occurs in only 11 of 100000 infections[7]. The pathogenesis of cardiac effects is multifactorial like dysregulation of renin-angiotensin-aldosterone system, endothelitis, cytokine associated vascular inflammation, direct viral cytopathic affection, plaque stability, catecholamine surge and formation of micro-thrombi. Multi-systemic inflammatory syndrome plays a role as well. Hypoxemia secondary to lung affection and toxicity of the medications can also add to the myocardial injury[8]. Acute heart failure can develop due to new myocardial injury in patients with no prior heart disease or the pre-existing stable heart failure can be decompensated during an acute episode of Covid infection[9]. Stress cardiomyopathy has also been reported in hospitalized patients of Covid -19 infection[10]. Pneumonia and/ or pulmonary embolism causing right ventricular

strain with biomarker elevation were reported during severe disease. In about 20% of hospitalized patients, transient atrial fibrillation, flutter or SVTs were reported. Ventricular arrhythmias were uncommon[11]. In presence of DM, atherosclerosis, hypertension, and/ or obesity, the immune system is down-regulated and significant cardiac manifestations appear early[12]. The new variants-Delta and Omicron strains are observed to cause less severe manifestations without occurrence of significant acute cardiac effects compared to the initial Delta variants[13].

CARDIAC EFFECTS IN POST-ACUTE PHASE

Many patients affected with COVID-19, reported lingering symptoms like chronic fatigue, head ache, joint pains or muscle weakness, dyspnea, chest pain, palpitations and autonomic disturbances in post-acute-phase [By convention taken as 4 weeks from onset of symptoms] which are clubbed and stamped as "LONG-COVID'. Terms like long-haul COVID, chronic COVID, post-COVID syndrome, post-acute COVID-19 syndrome, post-acute sequalae of SARS-CoV2 infection (PASC) and post-COVID conditions are all used by various authors to describe this ill-understood phenomenon[14]. This constellation of diverse range of symptoms is somewhat akin to other post -viral syndromes (Epstein-Barr, Human herpes, influenza SARS etc). The concept of Long-COVID was standardized for the first time by NICE guidelines (UK) in 2020 as the syndrome of persistence of symptoms following SARS-CoV2 infection beyond 4 weeks[15,16]. The symptoms in COVID survivors, weeks after the acute phase may be ongoing, recurring or new-onset.

WHO proposed a clinical case definition of post-COVID-19 condition [Delphi consensus,2021]. They defined it as a syndrome consisting of fatigue, shortness of breath, and cognitive dysfunction that is seen about 3 months from onset, lasts at least 2 months and cannot be explained by alternate diagnosis[17]. Royal College of general practitioners (RGCCP) has suggested that the COVID -19 manifestations are best studied in 3 time-frames[18].

1. Acute phase: up to 4 weeks
2. Ongoing symptomatic: 4th week to 12 weeks
3. Post-acute Covid-19 phase: from 12 weeks onwards

Satterfield[19] et al categorized the cardiac consequences in post- acute COVID patients as

1. Long haulers: persistent symptoms in those with initial asymptomatic mild-moderate infection
2. Evidence of cardiac injury in initial mild infection
3. Cardiovascular complications in patients with moderate to severe initial infection who had cardiac injury [elevated cardiac troponins and or reduced left ventricular ejection fraction]
4. Collateral damage due to delayed or no treatment of acute coronary syndrome, stroke or other cardiovascular disorder

At least 60-80% of hospitalized and about 30% of non-hospitalized COVID patients have one or other residual symptom even by end of 2 months[20,21]. In post-acute COVID-19 US study (n=1250), 32.6% reported persistent symptoms like dyspnea (22.9%), cough (15.4%) and persistent loss of taste and smell (13.1%). In this 60-day outcomes study, 488 patients completed the telephonic survey. Of them 6.7% died and 15.1% got re-hospitalized; 38.5% could not resume normal activities and 11% developed new issues in doing daily activities[22].

COVID-HEART

During the months after acute phase (4 weeks), approximately 20% of patients have significant cardiac involvement, more so, if they had prior cardiac illness[23]. The diverse cardiovascular manifestations beyond the acute-phase are summarized in table-1.

Table-1: Long COVID syndrome: Common cardiovascular manifestations

Serial Number	Manifestations	Remarks
1	Asymptomatic raised troponins, D-dimer/MRI abnormalities	As high as 60%
2	Myocarditis	True incidence very low
3	Ischemic syndromes	Chest pain ~20% at 60-days -5% at 6 months
4	LV Dysfunction; Heart failure; Stress cardiomyopathy	Diastolic dysfunction is more common. LV systolic dysfunction may be persistent in only about 3%; RV dysfunction more common than LV dysfunction on follow-up
5	Arrhythmias/Conduction disorders i. SVTs ii. Prolonged QT/VT/V. Fib iii. AV blocks	Palpitations ~10% at 60 days; 9% at 6 months
6	Autonomic dysfunction	Postural Orthostatic tachycardias, Inappropriate sinus tachycardia
7	Arterial and Venous thromboembolism	<5%

An elegant study was conducted by Xie et al, on cardiovascular outcomes of COVID-19 in the post-acute phase, at the Veterans Affairs (VA) St Louis Health care system in a cohort of 153,760 survivors of COVID-19. The control group had 5637647 patients with no evidence

of SARS-CoV-2 infection during the study period and the historical control group was 5859411 patients that used the VHA services in 2017. The study group had 89% males (largely white male patients) with mean age-62 years. These individuals were at substantial risk of CV disorders like thromboembolic problems, arrhythmias, myocarditis, ischemic and non-ischemic myocardial injury, pericarditis, and heart failure. Those who had COVID had 23 excess events per 1000 patients; adjusted hazard ratio 1.55. Development of new CV disease especially in those with severe initial infection who were hospitalized[24]. What was surprising is that the enhanced risk was no less significant even in those with mild or moderate initial affection and in those not hospitalized[25]. The authors suggested that 'history of Covid-19' should be considered as a cardiovascular disease risk-factor, as it did not spare any sub-group or those without any prior disease or any known risk factors. Another suggestion put forth was that such presentation has to be considered as one facet (COVID heart) of the multifaceted disorder that may be named –Long COVID syndrome[24]. The definition of this new emerging syndrome is not yet clear; but it forms an important topic for further research and refinement.

Xie's eye-opening study stimulated many others to have a focused look at the newly emerging cardio-vascular challenge. There are a few smaller observational studies reporting similar cardio-metabolic problems in post-acute phase of COVID infection. In an analysis of a cohort of 428,650 COVID-19 patients from UK family practice database, from 2020 to 2021, the incidence of new onset DM and CVD were found to be unexpectedly high. On 12 month-follow-up, the diagnosis of new-onset DM increased by 81% in acute phase and the higher incidence persisted by 27% at 4 to 12 weeks after infection. During the acute phase, the diagnosis of pulmonary embolism, atrial arrhythmias and venous thrombosis increased by 11, 6 and 5 folds respectively; however, the rise in the incidence reached baseline levels by 12 weeks to 1 year after infection. This study seems to reassure that individuals without prior CVD or DM may not have a higher risk for long term incidence of CVD in contradiction to the observations of Xie et al[26]. Tereshchenko LG et al tried to determine absolute and relative risk of CV events (primary outcome) and all-cause mortality (secondary outcome) both in symptomatic and asymptomatic patients with SARS-CoV-2 infection in a retrospective double cohort study. At a median of 6 months, the COVID-19 positive group had 12% primary events which were double of the primary events in the COVID-19 negative group. The average time to all-cause mortality was 65.5 days lesser in those who had COVID-19 infection in the recent months[27]. The probable enhanced cardiovascular risk and the long-term consequences of COVID infection are becoming significant issues for the researchers and clinicians alike. Surprisingly a good number of COVID survivors are asymptomatic but show cardiac abnormalities like raised troponins or MRI detectable lesions suggestive of myocardial

damage. Koteza reported that of the 148 troponin positive patients he found myocarditis in 26%, inducible ischemia in 22% and in 6% both can occur[28]. The possible mechanism underlying the enduring cardiac damage in post-acute stage following COVID-19 infection is not well-understood. Several possible mechanisms have been proposed as enlisted in table-2 below[29].

Table-2: Proposed mechanisms for COVID-HEART syndrome

Mechanism	Remarks
Chronic vascular inflammation	? Due to persistent viral reservoirs
Endothelial dysfunction	Cytokine/adipokine related
Enhanced myocardial fibrosis	Increased myocardial stiffness Increased arrhythmogenicity
Impaired perfusion	Reduced contractility
Microthrombi	Leading to pulmonary hypertension
Auto-immune response to cardiac antigens	

We are still not clear about the appropriate work-up and management of such individuals. True prevalence of this cohort is also unknown as there is no defined protocol to test COVID survivors. Several centers at different countries have registered prospective clinical trials on various aspects of this new clinical challenges which are ongoing and are expected to throw more light[29].

Myocardial Injury/Myocarditis/Pericarditis

In early reports a significant raise (more than 99th percentile URL) in cardiac biomarkers was taken to indicate myocarditis which was seen in as many as 60% of the hospitalized cases. CMR imaging showed abnormalities in most of such cases, which were thought to indicate persistent myocardial inflammation[30]. Subsequent studies were more reassuring by showing that both seropositive and seronegative subjects had very negligible incidence of myocarditis. The irreversible myocardial damage as detected on MRI is rare and histopathological correlation is uncommon. These lesions have little functional consequences[28,31,32].

Athletes who recovered from Covid infection and are asymptomatic formed a special group for research as they need a scientific answer as to when to resume their sports activities following COVID infection. Cardiac MRI was chosen as a convenient and useful tool for studying this question by several authors.

Rajpal et al reported that 15% of college level-athletes showed LGE pattern on MRI, suggestive of myocarditis following recovery from COVID infection[32]. But a larger study by Daniels found that only 2.5% of athletes had imaging based-myocarditis[33]. Udelson clarified that focal LGE in inferolateral RV insertion is common with physiological remodeling that occurs in athletes in training and it should not be confused with myocarditis[34] . In view of these recent studies, AHA/ACC recommended a 3 to 6-month holiday from sport if a CMR confirmed myocarditis is present in an athlete[35-37].

Altered Cardio-metabolic profile

Al Aly retrospectively studied 73435 non-hospitalized COVID patients and found high risk of deaths and incident CV and metabolic diseases beyond 4 weeks of the initial infection[38]. Risk of developing type 2 diabetes mellitus was higher in Covid survivors compared to others recovering from pneumonia[39].

Ischemic syndromes

During the initial months of the pandemic, the admissions for acute coronary syndromes declined by about 50% which also reflected on the number of primary PCIs performed. This was largely explained by patients not reporting to the hospitals for fear of getting infected and due to redistribution of health care priorities to tackle the pandemic[40,41]. In view of stress-induced cardiomyopathy, plaque instability, uncontrolled glycemic state and hypertension and poor drug compliance patients in post-acute phase of are expected to present with coronary events for months after acute phase[42]. In a prospective cohort of 2316 confirmed COVID patients PET myocardial perfusion imaging was done at Houston Methodist heart and Vascular Centre and CFR was calculated. Coronary microvascular disease defined as CFR less than 2 was present in 58% (vs. 46% of the control). Such significantly higher microvascular dysfunction was equally in all sub-groups and also in those with normal stress tests[43].

With experience of managing the COVID patients presenting with chest pain or breathlessness and elevated troponins, it is now realized that patients have to be classified as acute MI, acute non-ischemic myocardial injury and chronic myocardial injury to plan out appropriate therapy based on objective testing and clinical correlation.

Stress Cardiomyopathy

Like the acute phase the post-acute period of a Covid-19 survivor can be very stressful. Some of the patients presenting with chest pain are due to stress-induced cardiomyopathy. Data on this occurrence is lacking.

Heart failure

Cytokine -mediated systemic inflammation, microvascular dysfunction, hypoxic damage, hemodynamic disturbances and direct effects of virus on the myocardium can lead to myocardial injury following Covid infection. Both precipitation of acute decompensated heart failure in previously healthy individuals

and worsening of heart failure in relatively stable chronic HF patients were reported in acute phase of COVID-19 infection but there is no concrete data on the long-term implications of such events[9]. In Xie's study veterans who had COVID-19 showed 72% higher risk of having heart failure at 12 months compared to the control cohort. Ayoubkhani et al noticed increased incidence of exacerbations of heart failure beyond 30 days after SARS-CoV2 infection[44].

Many of the mildly affected patients of COVID showed cardiac MRI abnormalities suggestive of new myocardial injury and hence there was an initial apprehension that there could be a steep-rise in symptomatic and deteriorating HF patients among the survivors in the months that followed the first wave of COVId-19. However, in patients with evidence of cardiac injury (elevated troponins and or MRI abnormalities) there was no correlation with tissue abnormalities, symptoms and the LV ejection fraction[45]. A recent echocardiographic study on long-COVID from Norway is very reassuring. In the 182 COVID survivors with more than 50% complaining of dyspnea at 3 months did not show progressive changes in echocardiographic parameters. Low LV global longitudinal strain was seen in 19% at 3 months and 15% at 12 months. The corresponding figures for low LVEF were 16% and 14% and for diastolic dysfunction 13% and 17% respectively[46]. Persistent LV systolic function is reported in about 9-11% cases on echo and CMR follow-up. However diastolic abnormalities can be seen in as many as 60% of patients. Right ventricular dilation and dysfunction are the most common echocardiographic abnormalities found in acute stage but they recover in most case during follow up[47]. However in hospitalized patients with about 50% needing mechanical ventilation de Graaf et al reported LV dysfunction in 18% and RV dysfunction in 10% of the survivors at 6 weeks after discharge[48]. Even the NT-pro BNP levels were abnormal in only 7% at 5 month follow-up after hospitalization for SARS-CoV2 infection in a UK study[49].

Arrhythmias, POTS and heart blocks:

Palpitations are a common symptom in the months that follow an acute COVID infection. Both bradyarrhythmias and tachyarrhythmias are possible during acute phase but their persistence into chronic phase is not well-documented. Wellborn et al studied the association of Covid and atrial fibrillation/flutter in 3050 Covid survivors and compared with matched 11004 Covid negative patients. The Covid positive patients had 1.57 the odds (95% CI 1.00, 1.41). Compared to the matched pre-pandemic population, Covid survivors were developed atrial fibrillation at odds of 1.57 times[50].

Heart-rate recovery, a surrogate measure for autonomic health recovery was impaired in some patients. Like any other viral illness, the COVID affected persons develop postural orthostatic tachycardia syndrome (POTS) which has to be treated in conventional methods like salt and volume manipulations, midodrine, betablockers and ivabradine[51].

Inappropriate sinus tachycardia was also seen in COVID survivors, for months after the acute episode, which may be explained by the adrenergic modulation[52]. In a recent small study Jamal et al performed head-up tilt test (HUTT) in 24 post-covid symptomatic patients at a mean interval of 5 months and found that 23 of them tested abnormal[53].

Long et al dynamic ECG changes like depolarization and repolarization abnormalities which are frequent in acute phase disappear in the majority in later 6 months[54]. New ECG abnormalities are reported in about one-thirds of survivors even after not so severe initial infection[55]. QT prolongation was observed in some patients during acute Covid-infection that was attributed to acute inflammation and the drug effect of hydroxyl chloroquine and azithromycin. But its occurrence in post-acute phase unrelated to drugs appears to be very rare. There is one case report of a 75 old female developing remarkable QT prolongation that could not be explained by medications or electrolyte disturbances many weeks after acute Covid infection[56]. Hosseini Z et al. reported a 48-year-old without any co-morbidities being admitted for COVID-19 pneumonia that was treated with azithromycin and hydroxychloroquine. He developed complete heart block on day-1, prior to administration of the drugs. It persisted even after 7 weeks and pacemaker implantation was done. His troponins and other parameters ruled out myocardial ischemia or infarction. The inflammatory markers which were elevated on admission normalized in 2 weeks[57].

Thromboembolic phenomena

In a small study at 3 months follow-up of Covid survivors by dual energy CTA, 5.5 % showed proximal arterial thrombosis. In acute –COVID, elevated D-dimer and venous thrombotic phenomenon are frequently reported. In several survivors repeat testing during follow-up shows continued elevation of D-dimer for an indefinite period. But the long-term risk for VTE is less well-defined. There are contradictory reports of increased VTE in COVID-19 survivors in view of raised D-dimer[58]. A recent large UK study showed that there is an increased incidence of both arterial and venous thrombotic disease during the acute phase and such tendency declines to some extent after the first 3 weeks, with an elevated risk remaining upto 49 weeks after COVID-19[59]. Implication for such persistent elevation is unknown though it is likely to develop pulmonary hypertension during follow-up.

Multi-system Inflammatory Syndrome in adults (MIS-A)

Initially it was thought to be a phenomenon specific to Covid in children, but similar cases were also observed in adults. It can present with features of myocarditis, shock, acute

coronary syndromes or arrhythmias in acute phase but the manifestations persist even for weeks beyond 4 weeks in some individuals. It is reported to be very rare or probably underdiagnosed. There are a few late presenting cases reported with initial mild or missed diagnosis of COVID-19[60,61].

The Vaccinated Individuals:

Population which had been vaccinated (to various degrees) irrespective of their actual exposure and infection-status to the virus, is growing in great numbers. In sub-analysis of unvaccinated patients in the study by Xie et al showed higher incidence of myocarditis compared to the vaccinated individuals[24]. Vaccinated individuals are noted to be protected from severe forms of COVID infection and the hospitalizations are lesser in them. On the other hand, vaccination itself was reported to cause myocarditis in stray cases causing considerable fear among public in the initial days of its introduction. Fortunately, the cardiac side-effects due to vaccines were proven to be rare and mild in nature compared to cardiac effects of Covid-19 infection[62]. In a recent study the reported incidence of myocarditis following mRNA vaccination was 2.7 per 100000 cases[63]. A BMJ update has stated that the male adolescents and young adults had the highest risk for myocarditis after mRNA vaccine, more so with the Moderna vaccines. A meta-analysis of 4 cohort studies comprising 231,22,522 persons aged 12 or above, revealed that there is a small but definite risk of development of myocarditis (0.004%) and pericarditis (0.005%) following vaccination with mRNA-1273/ BNT162b2/AZD1222 or combinations. Such occurrence is more likely if 2 doses of the same vaccine are taken. The incidence was highest in young males. The management of such cases is non-specific and supportive. If myocarditis occurs, subsequent usage of mRNA vaccine is not recommended[64].

Predicting the risk population

Obesity, female sex, hypertension and immune deficit states are purported risk factors. Need based basic blood tests like hemoglobin, total and differential count, CRP, BNP, Troponins, serum ferritin, and D-dimer are to be done during follow-up visits in primary care[65,66]. The degree of elevation of CRP, BNP and troponins can have potential for prediction the risk for cardiovascular damage on long term basis. Serial measurements of D-dimer in post-acute phase of COVID-19 is a simple strategy to detect and control venous thromboembolism and decide on long-term use of low molecular heparins/ NOACs[66,67]. Recently a test based on inflammatory markers-SARS-CoV-2 protein in CD14+, CD 16+ monocytes was approved by CE [IncellDx] that was validated for detection of post-acute sequelae after COVID-19(PASC). It is expected to pick up long COVID earlier and help in differentiate confusing clinical presentations[68]. Similarly, Swank et al detected SARS-CoV-2 spike elevated even upto 12 months in patients with post-acute sequelae of Covid-19[69]. Cardiac MRI is gaining acceptance as the choice imaging tool to know cardiac affection following COVID infection due to its ability to

diagnose myocardial inflammation, remodeling changes and differentiate from other non-COVID pathologies[70]. It appears that the new research is going on to bring out dependable biomarkers to identify the cohort vulnerable to develop long-COVID sequelae.

Non-cardiac manifestations

Pulmonary, renal and neurological effects following Covid infection can indirectly impact the cardiovascular manifestations. Huang and colleagues followed up 1733 adults who were hospitalized and discharged from a hospital in Wuhan, China for 6 months. More than 50% had residual radiological shadows on chest X-rays and lung diffusion abnormalities. Xenon MRI study suggested that in long-term COVID-19 in patients with unexplained dyspnea, the ventilation and gas -exchange may be impaired due to capillary level abnormalities. It was also observed that of those discharged with normal renal function, 13% developed decline in eGFR at follow-up[71]. In another MRI study of 401 patients (mostly mildly affected and non-hospitalized) there was overall reduction in brain size, grey matter thickness and tissue contrast in several sites of brain. These patients were found to have neurocognitive defects on clinical assessment. A recent small study by Andrew Callen and colleagues suggested that abnormal cerebrovascular reactivity and endothelial dysfunction persist in long-COVID patients[72].

Long COVID in children

Long term impact of COVID infection in neonates, infants and children is not well understood. MIS-C is uncommon after viral infections and it can have long-term consequences in children. Its incidence sharply increased after the recent COVID pandemic. As no data is available on longitudinal follow-up of COVID in children, NIH and CDC have designed certain research studies addressing this issue[73]. NIAID is conducting a natural history study and it supports RECOVER initiative [Researching COVID to Enhance Recovery]. NICHD, NHLBI and NIAID also initiated 'Caring for children with COVID' program and the PRISM study. Many hospitals started special clinics like JH-PACT (John Hopkins' Post-acute Covid-19 team to focus on this vulnerable cohort[74].

Practical implications and Conclusions

The long-term consequences of cardiac symptoms and asymptomatic signs of cardiac injury, weeks or months after the initial infection are still not clear. Understanding the disparate natural history and long-term impact of the acute cardiac damage during COVID-19 infection is a research priority. Patients with post-acute sequelae of SARS-CoV-2 infection are advised for appropriate evaluation especially if symptomatic . The cardiac MRI is emerging as the most useful tool to evaluate the myocardial damage and to advice on return to work or play in post-acute phase of COVID infection. The role of CRP and cardiac troponins as risk predictors need further validation. As the survivors following an episode of COVID are fast growing in number, care of these individuals can emerge as a new public health issue.

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