Assessment of Cardiac Involvement in Adult Filipino Patients Recovered From Corona Virus Disease-19 (COVID-19) Infection Using Magnetic Resonance Imaging

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ABSTRACT

BACKGROUND
Cardiac sequelae after recovery from Coronavirus Disease 2019 (COVID-19) infection has not been well-established. Recent studies have used cardiac magnetic resonance (CMR) imaging to assess myocardial involvement or ongoing myocarditis in patients with prior COVID-19 infection.

OBJECTIVES
The primary objective of this study was to identify the CMR imaging findings in adult Filipino COVID-19 recovered patients.

METHODS
This was a single-center retrospective observational cohort study of adult Filipino COVID-19 recovered patients who underwent cardiac magnetic resonance imaging. Patient demographics, CMR findings, blood marker results, and treatment received were obtained.

RESULTS
Of the 77 included patients, 42 (54.55%) were male, and the median age was 45 years. Only 6 patients had completely normal scans, making the prevalence of an abnormal CMR 92.21% (95% CI 83.5%–96.5%). All those with an abnormality demonstrated late gadolinium enhancement (LGE), and of these, 64 (90%) had preserved left ventricular ejection fraction (LVEF). Thirty-four patients (44%) had evidence of myocardial edema. Among patients with myocardial edema and LGE, the median numbers of involved segments were 4 (range 1-16) and 7 (range 1-15), respectively. Myocardial edema was most frequently found in the mid inferolateral segment (53%), followed by the basal inferior septum, basal anterior septum, and basal inferolateral segments (each with 41%). Meanwhile, late gadolinium enhancements were most commonly located in the basal inferior septum (75%), mid inferior septum (80%), and basal anterior septum (73%).

CONCLUSION
Cardiac involvement, particularly edema and late gadolinium enhancement, affecting multiple myocardial segments were observed in a considerable number of patients recovered from COVID-19.

INTRODUCTION

In December of 2019, the Coronavirus illness (COVID-19) was first diagnosed in Wuhan, China. The World Health Organization classified COVID-19 as a pandemic on March 11, 2020, owing to the rapidity and scope of its spread, which affected more than 200 nations and caused more than a million fatalities globally.[1] COVID-19 is primarily a respiratory illness, but other organs can be affected. SARS-CoV-2 binds to transmembrane ACE2 to enter host cells, such as type-2 pneumocytes, macrophages, endothelial cells, pericytes, and cardiac myocytes, causing inflammation and multi-organ failure.[2]

Heart failure and myocardial damage, as shown by elevated troponin plasma levels, occur in at least 10% of hospitalized COVID-19 patients, with greater percentages (i.e. 25% to 35% or more) when critically ill patients or those with associated cardiac illness are involved.[3] It is possible for activated T lymphocytes and macrophages to penetrate infected myocardium, leading to the development of fulminant myocarditis and severe cardiac damage, which may end in heart failure and arrhythmias.[4] Acute lung damage may potentially contribute to cardiovascular illness, as it increases cardiac workload, particularly in individuals with preexisting cardiovascular disease.[4]

In a local study by Ong et al, 90 confirmed COVID-19 patients with cardiac biomarker determination showed acute myocardial injury, as evidenced by increased levels of High-sensitivity Troponin-I.[5] Ong et al also found that High-sensitivity troponin-I was significantly higher among non-survivors, with significant positive association between elevated high-sensitivity troponin-I and in-hospital mortality.[5]

Late sequelae of COVID-19 have yet to be identified and its overall impact remain undetermined. At present, there is no data regarding the extent of cardiac involvement among Filipino adults who have recovered from COVID-19 infection. This study aims to determine the prevalence and extent of cardiac involvement among adults who have recovered from COVID-19 infection based on magnetic resonance imaging. Information regarding the long-term cardiovascular effects of COVID-19 will provide data on the burden of disease and clinical profile of patients, which may perhaps influence treatment practices.

The main objective of this investigation was to determine the presence and extent of cardiac involvement among Filipino adults who have recovered from COVID-19 infection based on magnetic resonance imaging. Specifically, the study aimed the following:

1. To identify the following cardiac magnetic resonance (CMR) imaging findings in COVID-19 recovered patients
2. To determine the patients' profiles in patients with CMR findings

3. To describe COVID-19 disease severity and CMR findings
4. To describe time from diagnosis of COVID-19 and CMR findings
5. To describe laboratory tests and CMR findings
6. To describe treatment received and CMR findings:

METHODS

Study design and participants

This was a single-center retrospective observational cohort of seventy-seven (77) adult Filipino patients who have recovered from COVID-19 infection and were referred for cardiac magnetic resonance imaging from December 2020 to March 2022. The definitions used for confirmed COVID-19[6] and recovered COVID-19[7] cases were according to the Philippine recommendations at the time of study (see Appendix). Ethical approval was obtained from the institutional review board. Clinical data regarding patient demographics, past medical history, smoking history, date of COVID-19 diagnosis and disease severity, inpatient blood test results, and treatment received were obtained through chart review.

All patients underwent cardiac magnetic resonance imaging (Siemens Magnetom Aera 1.5T MRI scanner) with contrast for morphology and function. The following pulse sequences were used: (1) turbo spin echo and gradient echo imaging for anatomic definition; (2) dynamic cine imaging (steady-state free precession) for cardiac chamber and wall motion analysis; (3) delayed gadolinium enhancement analysis. The CMR diagnosis of myocarditis-like injury was made based on published expert guidelines and the evidence of non-ischemic myocardial injury with a characteristic distribution and myocardial edema.

Analysis of Data

Descriptive statistics was used to summarize the general and clinical characteristics of the participants. Frequency and proportion were used for categorical variables (nominal/ordinal), mean and standard deviation for age, and median and range for age and laboratory variables. All valid data was included in the analysis. Missing data was neither replaced nor estimated. STATA 15.0 was used for data analysis.

RESULTS

Seventy-seven patients with CMR data were analyzed (Table 1). Their ages ranged from 18 to 82 (median 45) years, with males (55%) non-significantly outnumbering females (p = .425). About half of patients had hypertension (50%) and dyslipidemia (49%). Four in 10 (n = 31) had no data on the severity of their COVID-19, but for the remaining 46 patients, over half (59%) were noted to have had mild disease. The most common estimate for interval from COVID-19 diagnosis to CMR imaging was about 1 to 3 months (43%).

At most, 18 patients had known levels for any given biomarker at a time (Table 2). Most of the findings on white blood cell count (14/17) and procalcitonin (16/16) were normal, while the majority of results for high-sensitivity C-reactive protein [Hs-CRP] (15/17), lactate dehydrogenase [LDH] (12/17), and ferritin (12/17) were elevated. Only 2 of the 17 (12%) patients with available data showed elevated high-sensitivity Troponin-I (HS-TnI, but 3 of the 4 (75%) patients with known NT pro-Brain Natriuretic Peptide (NT-proBNP) demonstrated high levels of the cardiac marker (Table 2).

Table 1. Clinico-demographic profiles of patients (n = 77)

	Mean ± SD; Frequency (%)
Age, years	46.88 ± 15.47
Sex	
Male	42 (54.55)
Female	35 (45.45)
COVID-19 severity	
Mild	27 (35.06)
Moderate	7 (9.09)
Severe	10 (12.99)
Critical	2 (2.6)
Unknown	31 (40.26)
Time from COVID-19 diagnosis to CMR	
≤30 days	4 (5.19)
>30 to ≤90 days (1-3 months)	33 (42.86)
>90 to ≤180 days (3-6 months)	10 (12.99)
>180 days	20 (25.97)
Unknown	10 (12.99)
With smoking history [n = 65]	8 (12.31)
Comorbidities	
Hypertension [n = 66]	33 (50)
Diabetes mellitus type 2 [n = 65]	17 (26.15)
Asthma [n = 65]	10 (15.38)
COPD [n = 65]	1 (1.54)
Obesity [n = 65]	12 (18.46)
CAD [n = 65]	4 (6.15)
Dyslipidemia [n=65]	32 (49.23)
CKD [n=66]	7 (10.61)
Others	48 (62.34)

Table 2. Cardiac and Inflammatory markers at the time of COVID-19 Infection

	Median (Range); Frequency (%)
Hs-CRP, mg/L [n = 17]	9.1 (0.67–80.22)
<5	2 (2.6)
≥5	15 (19.48)
D-dimer, ug/mL [n = 18]	0.425 (0.1–1.78)
≤0.5	10 (12.99)
>0.5	8 (10.39)
Hs-TnI, ng/mL [n = 17]	0.007 (0.01–0.047)
≤0.034	15 (19.48)
>0.034	2 (2.6)
LDH, U/L [n = 17]	263.23 (109.94–1071.77)
<125	1 (1.3)
125-220	4 (5.19)
>220	12 (15.58)
Ferritin, ng/mL [n = 17]	444.86 (13.12–3035.14)
<21.81	1 (1.3)
21.81-274.66	4 (5.19)
> 274.66	12 (15.58)
NT-proBNP, pg/mL [n = 4]	431.57 (39.16–1571)
<125	1 (1.3)
≥125	3 (3.9)
Procalcitonin, ug/L [n = 16]	0.055 (0.01–0.38)
<0.5	16 (20.78)
≥0.5	0
WBC count, x109/L [n = 17]	6.6 (2.75–9)
<4.4	3 (3.9)
4.4-11	14 (18.18)
>11	0

Up to 42 patients had data on their reception of any given type of treatment for COVID-19 (Table 3). Antiviral therapy, corticosteroid, and oxygen support were the most common types of therapies used. Two patients were noted to have been given tocilizumab.

Only 6 patients had completely normal scans, making the prevalence of an abnormal CMR 92.21% (95% CI 83.5%–96.5%) in the sample (Table 4). All those with an abnormality demonstrated late gadolinium enhancement, and of these, 64 (90%) had preserved left ventricular ejection fraction. Thirty-four patients (44%) had evidence of myocardial edema and were diagnosed with myocarditis (4 acute, 6 acute to subacute, and 24 subacute). The other most common types of abnormalities were dilated LA (19%), dilated RA (17%), and elevated LVEDVI (19%).

Among patients with myocardial edema and LGE, the median numbers of involved segments were 4 (range 1-16) and 7 (range 1-15), respectively (Table 5). Myocardial edema was most frequently found in the mid inferolateral segment (53%), followed by the basal inferoseptal, basal anteroseptal, and basal inferolateral segments (each with 41%). Meanwhile, late gadolinium enhancements were most commonly located in the mid inferoseptal (80%), basal inferoseptal (75%), and basal anteroseptal (73%) segments.

Out of 77 patients who recovered from COVID-19, 71 had abnormal CMR findings and 6 had normal CMR findings (Table 6). The mean age in years of patients in abnormal CMR group was higher than those with normal CMR group (47.06 + 15.58 vs 44.83 + 15.22). There is an equal distribution (1:1) of gender in both groups. Almost a third of the patients (33.8%) in the abnormal CMR group and half of the patients (50%) in the normal CMR group had mild COVID-19 (33.8%). In the abnormal CMR group, approaching half (45.07%) had estimated interval of more than 30 days but within 90 days (1-3 months) between COVID-19 diagnosis and CMR. Among the 65 patients with known smoking history, about 8 (13.56%) belonged to the abnormal CMR group. The top comorbidities were dyslipidemia (51%) and hypertension (50%) in the abnormal CMR group. Only one patient with elevated High sensitivity C-reactive protein, LDH, and ferritin had a normal CMR. The most common treatments given to the abnormal CMR group were use of corticosteroid (39.47%) and oxygen support (35.9%).

Table 3. Treatments for COVID-19 episode

	Frequency (%)
Antiviral therapy [n=41]	19 (46.34)
Antibiotic therapy [n=41]	10 (24.39)
Corticosteroid [n=41]	16 (39.02)
Oxygen support [n=42]	14 (33.33)
Tocilizumab [n=41]	2 (4.88)

Table 4. Cardiac MRI findings

	Median (Range); Frequency (%)
LV EF, %	64 (28–73)
<50	7 (9.09)
≥50	70 (90.91)
LV mass index, g/m^2	56 (38–121)
>115	1 (1.3)
LV EDVI, mL/m^2	73 (49–244)
>90	15 (19.48)
RV EF, %	65 (47–75)
>70	6 (7.8)
Dilated LA	15 (19.48)
Dilated LV	6 (7.79)
Dilated RA	13 (16.88)
Dilated RV	3 (3.9)
Pericardial effusion	4 (5.19)
Pericarditis	2 (2.6)
Myocardial edema	34 (44.16)
LGE	71 (92.21)
Myocarditis	
None	9 (11.69)
Acute	4 (5.19)
Acute to subacute	6 (7.79)
Subacute	24 (31.17)
Prior	34 (44.16)

EDV, end-diastolic volume index; EF, ejection fraction; LGE, late gadolinium enhancement; LV, left ventricle; RA, right atrium; RV, right ventricle.

Table 5. Sites of myocardial edema and LGE

	Myocardial Edema (n = 34)	Late Gadolinium Enhancement (n = 71)
	Frequency (%)	
Diffuse	1 (2.94)	-
Basal anterior	5 (14.71)	5 (7.04)
Basal anteroseptal	14 (41.18)	52 (73.24)
Basal inferoseptal	14 (41.18)	53 (74.65)
Basal inferior	9 (26.47)	36 (50.7)
Basal inferolateral	14 (41.18)	42 (59.15)
Basal anterolateral	10 (29.41)	23 (32.39)
Mid anterior	5 (14.71)	4 (5.63)
Mid anteroseptal	10 (29.41)	43 (60.56)
Mid inferoseptal	13 (38.24)	57 (80.28)
Mid inferior	10 (29.41)	31 (43.66)
Mid inferolateral	18 (52.94)	41 (57.75)
Mid anterolateral	11 (32.35)	28 (39.44)
Apical anterior	2 (5.88)	2 (2.82)
Apical septal	4 (11.76)	13 (18.31)
Apical inferior	4 (11.76)	11 (15.49)
Apical lateral	4 (11.76)	24 (33.8)
Apex	-	3 (4.23)
RV insertion point	-	5 (7.04)

DISCUSSION

There are reports of long-term multiorgan effects following an acute COVID-19 infection. More importantly, many patients continue to experience symptoms during recovery.[8] This study shows cardiac involvement, including fibrosis and myocarditis involving multiple myocardial segments in patients recovered from COVID-19. In a study by Puntmann et al[9] which included COVID-19 in patients who recently recovered from the disease, 78 patients (78%) had abnormal CMR findings, including raised myocardial native T1 and T2, myocardial late gadolinium enhancement or pericardial enhancement. The mean age was 49 years, most patients (67%) recovered at home (ranging from asymptomatic to mild to moderate symptoms), and median interval between COVID-19 diagnosis and CMR was 71 days. This is similar to our study population comprising of patients with a median age of 45 years, mostly with mild to moderate

Table 6. Patient profile and abnormality on CMR findings

	Abnormal (n = 71)	Normal (n = 6)
	Mean ± SD; Median (Range); Frequency (%)	
Age, years	47.06 ± 15.58 45 (18–82)	44.83 ± 15.22 46 (23–63)
Sex		
Male	39 (54.93)	3 (50)
Female	32 (45.07)	3 (50)
COVID-19 severity		
Mild	24 (33.8)	3 (50)
Moderate	6 (8.45)	1 (16.67)
Severe	10 (14.08)	0
Critical	2 (2.82)	0
Unknown	29 (40.85)	2 (33.33)
Time from COVID-19 diagnosis to CMR		
≤30 days	3 (4.23)	1 (16.67)
>30 to ≤90 days (1-3 months)	32 (45.07)	1 (16.67)
>90 to ≤180 days (3-6 months)	9 (12.68)	1 (16.67)
>180 days	18 (25.35)	2 (33.33)
Unknown	9 (12.68)	1 (16.67)
With smoking history [n = 65]	8 (13.56)	0
Comorbidities		
Hypertension [n = 66]	30 (50)	3 (50)
Diabetes mellitus type 2 [n = 65]	17 (28.81)	0 (0)
Asthma [n = 65]	9 (15.25)	1 (16.67)
COPD [n = 65]	1 (1.69)	0 (0)
Obesity [n = 65]	11 (18.64)	1 (16.67)
CAD [n = 65]	4 (6.78)	0 (0)
Dyslipidemia [n=65]	30 (50.85)	2 (33.33)
CKD [n = 66]	7 (11.67)	0 (0)
Elevated biomarker		
Hs-CRP	14 (19.72)	1 (16.67)
Hs-TnI	7 (9.86)	1 (16.67)
D-dimer	2 (2.82)	0
LDH	11 (15.49)	1 (16.67)
Ferritin	11 (15.49)	1 (16.67)
NT-proBNP	3 (4.23)	0
Procalcitonin	0	0
WBC count	0	0
COVID-19 treatment		
Antiviral therapy	18 (47.37)	1 (33.33)
Antibiotic therapy	10 (26.32)	0
Corticosteroid	15 (39.47)	1 (33.33)
Oxygen support	14 (35.9)	0
Tocilizumab	2 (5.26)	0

COVID-19 infection (n = 30/42, 71%), and CMR scans performed most frequently more than 30 up to 90 days from COVID-19 diagnosis. In their study, the most frequent preexisting conditions were obesity, hypertension and hypercholesterolemia, which is again similar to our findings wherein hypertension and dyslipidemia are the most common comorbidities. However, our study had a higher prevalence of abnormal CMR findings at 92.21%.

Kotecha et al[10] identified 148 hospitalized COVID-19 patients with acute cardiac damage, as indicated by increased troponin concentrations. Over-all N-terminal pro-brain natriuretic peptide (NT-proBNP) levels were elevated (231 ng/L, 72–878) in the 92 patients with available result. C-reactive protein levels were likewise raised (>5 mg/L) in all patients in at least one measurement. The time between discharge or confirmed diagnosis (defined as a positive COVID-19 swab result or diagnostic chest radiography, or CT imaging in the event of swab-negative patients) and CMR investigation was 56 days (IQR 30–88 days) and 68 days (IQR 39–103 days), respectively. Fifty-four percent of patients (80/148) had cardiac abnormalities. Scar/injury patterns were inflammatory in 32% (48/148 patients) and ischemic in 28% (41/148), with 9 patients showing both. 12 patients (8%) had evidence of (possibly ongoing) myocardial inflammation. This study by Kotecha et al adds to the data that cardiac damage is prevalent 2 months following COVID-19 infection.

In a systematic review by Ojha et al[11] with a total of 34 studies comprising 199 patients, ventricular function was normal in most of the patients. Our study showed that 90% of patients with abnormal CMR had preserved left ventricular ejection fraction, which is consistent with current data.

Raman and colleagues[12] found lower rates, with only 26% having abnormal CMR results and 11.5% having an inflammatory LGE pattern. Even with lower rates, it's important to remember that given the vast number of COVID-19 patients, an 11.5 percent incidence may have societal impact.

It is important to determine the whether the cardiac damage observed during the acute phase of COVID-19 will result in subsequent cardiac dysfunction and morbidity. In our study, all those with an abnormality demonstrated late gadolinium enhancement, thirty-four patients (44%) had evidence of myocardial edema and were diagnosed with myocarditis (4 acute, 6 acute to subacute, and 24 subacute) (Figure 1 and 2). Myocardial edema was most frequently found in the mid inferolateral segment (53%), while LGE was most commonly located in the mid inferoseptal (80%) segment. Viral myocarditis is a leading cause of unexpected and sudden death, and it can evolve into dilated cardiomyopathy.[13, 4,15] A total of 183 patients with biopsy-proven viral myocarditis and cardiovascular magnetic resonance were followed up at a median of 10.1 years in a study by Greulich et al.[16] They found that patients with biopsy-proven myocarditis had a high percentage of long-term mortality (39.3% percent all-cause, 27.3% cardiac, and 10.9% SCD); 101 patients (55.2%) had LGE.[16] The presence of LGE was linked with an increased risk of mortality (hazard ratio [HR], 2.40; 95 percent confidence interval [CI], 1.30–4.43), with an HR of 3.00 (95 percent CI, 1.41–6.42) for cardiac death and an HR of 14.79 (95 percent CI, 1.95–112.00) for SCD; all P0.009.[16] In particular, midwall LGE, (antero-) septal LGE, and LGE extent were strongly linked with mortality, all P0.001.[16] The greatest independent predictor of SCD was septal LGE (HR, 4.59; 95% CI, 1.38–15.01; $P = 0.01$).[16] We have yet to discover whether these results will apply to patients recovered from COVID-19.

The HEAL-COVID clinical trial (https://clinicaltrials.gov/ct2/show/NCT04801940), which began in April 2021, intends to enroll individuals who have healed from COVID-19 but continue to endure disease-related symptoms. The trial, with an estimated completion date on January 31, 2024, seeks to assess the impact of therapies on COVID-19-related long-term morbidity, mortality, re-hospitalization, symptom burden, and quality of life.

We found in this study that following recovery from COVID-19, CMR can reveal cardiac abnormalities in a substantial percentage of individuals. It will be essential to track these individuals over time to determine the prognostic significance of these CMR imaging results.

Figure 1: 34-year-old male with mild COVID-19 about 5 months prior, had acute chest pain, elevated troponin-I, and no significant coronary artery disease on coronary angiography. Cardiac MRI, at this short axis mid ventricle level, showed mid wall (green arrow) and subepicardial (yellow arrowhead) patterns of late gadolinium enhancement.

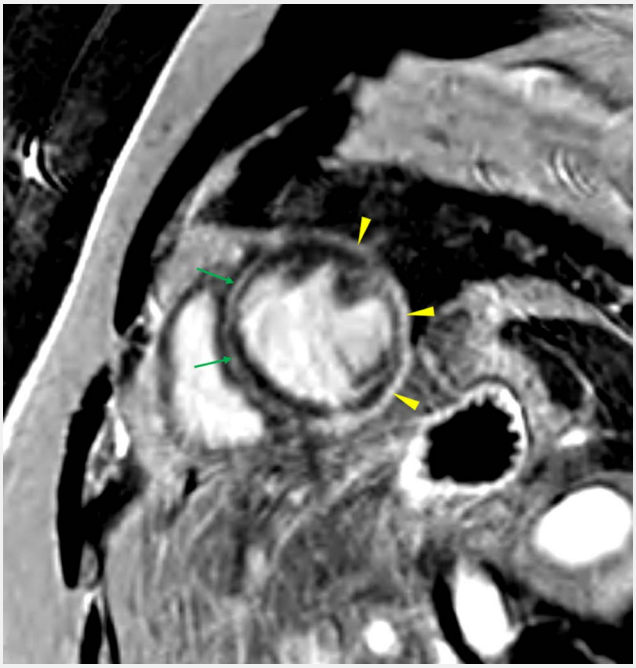
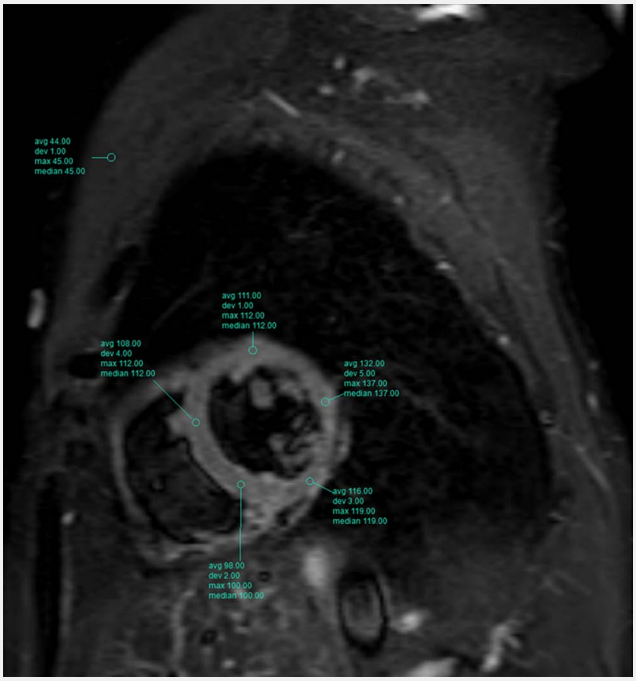


Figure 2: 54-year-old male with mild COVID-19 about 2 months prior had persistent shortness of breath and easy fatigability. Cardiac MRI (short axis mid ventricle level) showed diffuse myocardial edema.



LIMITATIONS

This was a single-center, retrospective observational cohort with an inherent selection bias: only the patients who were at risk of cardiac lesions post-COVID were likely be subjected to CMR. As such, we can describe the patients, but we cannot measure/establish associations. Despite this, we think that this study is important because it has a substantial number of patients for a relatively rare test (CMR) for COVID-19 recovered patients, locally.

CONCLUSION

Cardiac involvement, particularly edema and late gadolinium enhancement, affecting multiple myocardial segments were observed in a considerable number of patients recovered from COVID-19. Further study is required to determine whether these abnormalities are likely to affect the long-term clinical outcomes of these patients.

APPENDIX

Operational Definitions

1. Confirmed COVID-19 case - any individual, irrespective of the presence or absence of clinical signs and symptoms, who is laboratory-confirmed for COVID-19 in a test conducted at the national reference laboratory, a subnational reference laboratory, and/or officially accredited laboratory testing facility.[6] Rapid antigen tests are recommended as an alternative to RT-PCR if the following conditions are met: individuals are in the early phase of illness (less than or equal to 7 days from onset of symptoms); self-administered or laboratory-based tests are acceptable if with sensitivity of more than or equal to 80% AND specificity of more than or equal to 97%.[7]

2. COVID-19 Disease Severity as defined by Philippine Department of Health:[6]

    a. Mild: Symptomatic patients presenting with fever, cough, fatigue, anorexia, myalgias; other non-specific symptoms such as sore throat, nasal congestion, headache, diarrhea, nausea and vomiting; loss of smell (anosmia) or loss of taste (ageusia) preceding the onset of respiratory symptoms with NO signs of pneumonia or hypoxia

    b. Moderate: Adolescent or adult with clinical signs of non-severe pneumonia (e.g. fever, cough, dyspnea, respiratory rate (RR) = 21-30 breaths/minute, peripheral capillary oxygen saturation (SpO_2) >92% on room air)

    c. Severe: Adolescent or adult with clinical signs of severe pneumonia or severe acute respiratory infection as follows: fever, cough, dyspnea, RR>30 breaths/minute, severe respiratory distress or SpO_2 < 92% on room air

d. Critical: Acute respiratory distress syndrome, Sepsis, Septic Shock

3. Recovered COVID-19 patients according to disease severity:

   a. Recovered Mild COVID-19[7]– an individual with improvement of clinical status (afebrile for at least 24 hours without antipyretics and respiratory symptoms reduced significantly) and has completed 7 days of isolation from day 0 (onset of symptoms) for fully vaccinated individuals or completed 10 days of isolation from day 0 (onset of symptoms) for not fully vaccinated individuals

   b. Recovered Moderate COVID-19[7] - improvement of clinical status (afebrile for at least 24 hours without antipyretics, respiratory symptoms reduced significantly, chest X-ray shows significant improvement if available) and has completed 10 days of isolation from day 0 (onset of symptoms)

   c. Recovered Severe COVID-19[7] - improvement of clinical status (afebrile for at least 24 hours without antipyretics, respiratory symptoms reduced significantly, chest X-ray shows significant improvement if available) and has completed 21 days of isolation from day 0 (onset of symptoms)

   d. Recovered Critical COVID-19[7] - improvement of clinical status (afebrile for at least 24 hours without antipyretics, respiratory symptoms reduced significantly, chest X-ray shows significant improvement if available) and has completed 21 days of isolation from day 0 (onset of symptoms)

KEY WORDS
COVID-19, Cardiac Magnetic Resonance Imaging, Recovered COVID-19 Infection

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