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Coronavirus Pandemic

Patients with SARS-CoV-2 Omicron variant infection complicated with myocardial injury: a retrospective cohort study

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Abstract

Introduction: The SARS-CoV-2 virus and its variants affect not only the respiratory system but also the heart. The impact of SARS-CoV-2 on the heart during the Omicron pandemic is not yet fully elucidated.

Methodology: We conducted a retrospective cohort study of 1,026 hospitalized patients with the Omicron variant at Southwest Hospital (December 2022 to March 2023). Adverse events were defined as a composite of all-cause mortality, intensive care unit admission, mechanical ventilation use, and discharge against medical advice. The risk factors were analyzed by Logistic regression and the Cox regression model.

Results: Median age was 69 years (IQR 56–79), myocardial injury occurred in 54.7% (n = 561); adverse events occurred in 25.5% (n = 262). The risk factors of myocardial injury included older age (OR 1.04 [1.03, 1.05], $p < 0.001$), body temperature at admission (OR 1.64 [1.19, 2.29], $p = 0.003$), critical COVID-19 (OR 4.96 [2.34, 11.54], $p < 0.001$), severe valvular heart disease (OR 2.46 [1.06, 6.12], $p = 0.042$), renal insufficiency (OR 4.04 [2.75, 6.01], $p < 0.001$), anemia (OR 3.10 [2.12, 4.58], $p < 0.001$), type II respiratory failure (OR 5.54 [1.79, 24.39], $p = 0.008$) and higher white blood cell (OR 1.09 [1.05, 1.14], $p < 0.001$). After adjustment, myocardial injury (HR 1.79 [1.28, 2.48], $p = 0.001$) was significantly associated with adverse events in patients with Omicron variant infection.

Conclusions: Myocardial injury, the most common extrapulmonary complication of COVID-19 caused by the SARS-CoV-2 Omicron variant, requires timely attention to prevent adverse events in hospitalized patients.

Key words: Omicron variant; myocardial injury; adverse events; risk factors; SARS-CoV-2.

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Introduction

The COVID-19 pandemic, which emerged in 2019, has caused millions of deaths and widespread economic disruption globally. The Omicron era began in November 2021with the emergence of the B.1.1.529 variant. Marked a new phase of the pandemic. By late 2022, the Omicron variant had become the dominant SARS-CoV-2 variant due to its enhanced transmissibility, immune evasion and replicative advantage, which were primarily responsible for Omicron's vast geographical spread and high rates of transmission, because the Omicron lineages emerged with a much larger number of mutations in the spike gene (the spike protein of Omicron carries almost 30 mutations that facilitate immune evasion) than the previous variants of concern, and the Omicron variant rapidly replaced the Delta variant [1,2]. Consequently, vaccine effectiveness against infection declined, increasing population vulnerability [3]. It's necessary to keep evaluating risk factors for improving prognosis against the latest Omicron subvariants. However, most of the previous studies were about traditional SARS-CoV-2 [4]; the evidence of clinical characteristics related to the Omicron variant is limited.

Growing clinical data indicate that acute cardiac injury is a frequent extrapulmonary manifestation of COVID-19, associated with disease severity and poor outcomes [5,6]. A reasonable explanation could be that human angiotensin-converting enzyme 2 (hACE2), which is a cell receptor in the lungs with a strong binding affinity to the spike protein of SARS-CoV-2, is also highly expressed in the heart [7]. Notably, the Omicron sub-lineage BA.2 exhibits even stronger hACE2 binding affinity than other variants [2].

Early consideration of relevant risk factors enables timely interventions that may mitigate disease progression and improve clinical outcomes. Cardiac troponin (cTn) is a specific marker identifying ischemic or non-ischemic myocardial processes in COVID-19 [8]. A large number of studies showed that cTn elevation was associated with increased risk of adverse cardiovascular events, such as acute myocarditis,

arrhythmia, systolic heart failure, and pericardial effusion [9,10]. Mounting clinical studies have found elevated cTn concentrations among hospitalized patients with COVID-19 infection, particularly those with cardiovascular complications [11,12]. Integrating cTn levels with clinical parameters may improve risk stratification, especially in patients with uncertain severity [13]. However, data on cardiovascular manifestations and outcomes in Omicron-infected patients in China since 2023 remain scarce. This study describes the clinical characteristics and outcomes of more than 1000 hospitalized patients in southwestern China during the Omicron wave, identifies the risk factors of myocardial injury, examines its prognosis impact, and determines the optimal cTnT cut-off values for risk stratification.

Methodology

Study population

A retrospective cohort study was designed to include hospitalized patients with Omicron variant infection in Southwest Hospital (Chongqing, China) between December 1, 2022, to March 3, 2023. We included all hospitalized patients with SARS-CoV-2 infection confirmed by reverse-transcription polymerase chain reaction (RT-PCR), and excluded the following patients: (1) those under 18 years old; (2) pregnant and lactating women; (3) those without cTnT measurement; (4) those discharged within one day and hospitalized for more than 25 days. cTnT measurement: The first test was selected for those who were still within the normal range across multiple measurements, and the first elevated value was selected for those who tested positive. The patients with COVID-19 enrolled in this study were diagnosed accordingly to the World Health Organization interim guidance [14].

According to the "Clinical Protocol for the Treatment of Novel Coronavirus Infection (Trial Version 10)", the classification criteria are followed: Mild: Upper respiratory tract infection as the main manifestation, such as dry throat, sore throat, cough, fever, etc. Moderate: Persistent high fever > 3 days or/and cough, shortness of breath, etc, but respiratory rate (RR) < 30 times/min, oxygen saturation > 93% when breathing at rest. Imaging shows the characteristic manifestations of COVID-19 pneumonia. Severe: In adults, any of the following criteria are met and cannot be explained by anything other than COVID-19 infection: 1. Shortness of breath with RR > 30 times/min; 2. At rest, the oxygen saturation of the finger is less than 93% when sucking air; 3. Arterial oxygen partial pressure (PaO_2)/ fraction of inspired oxygen (FiO2) < 300mmHg. Critical: meet one of the following conditions: 1. Respiratory failure and the need for mechanical ventilation; 2. Shock; 3. With other organ failure and requiring ICU care. The study has been approved by the Ethics Committee of the First Affiliated Hospital of Army Medical University, PLA (approval number (B) KY2023023).

Patient information was collected from electronic health records, including demographic information, medical history, vital signs, and symptoms at admission, electrocardiographic (ECG) and echocardiographic results, laboratory examination, and hospitalization outcomes. Oxygenation index is defined as the PaO_2/FiO_2 ratio, abbreviated as P/F ratio. Patients were assigned according to whether they had myocardial injury during hospitalization, which was defined as the concentration of serum cTnT higher than 0.014μg/L. The clinical COVID-19 classification criteria were defined according to the "Clinical Protocol for the Treatment of Novel Coronavirus Infection (Trial Version 10)" and NIH COVID-19 Treatment Guidelines [15]. Adverse events of this study were all-cause mortality, transfer to the intensive care unit, the need for mechanical ventilation, and discharge against medical advice due to critical illness during hospitalization.

Statistical analysis

Descriptive analyses were used for demographics. Continuous variables are reported as median (interquartile range) or as mean ± SD. The Wilcoxon rank sum test was conducted to compare the differences in clinical characteristics between patients with myocardial injury and those without. Categorical variables are reported as total count percentages of patients, and the chi-square test was applied to analyze the difference between groups. The univariate and multivariate logistic regression models were used to explore the independent risk factors of myocardial injury, and the results were reported with odds ratio (OR) and 95% confidence intervals (CI). The multivariate Cox regression model was performed to evaluate the association between clinical variables and the adverse events, and the results were reported with hazard ratios (HR) and 95% CI. Baseline variables that were considered clinically relevant or that showed a univariate relationship with outcome (candidate variables with $p < 0.05$) were included in multivariate logistic and Cox proportional-hazards regression models. Variables for inclusion were carefully chosen, given the number of events available, to ensure parsimony of the final model.

Table 1. Baseline characteristics: clinical and laboratory characteristics of Omicron variant infected patients with or without myocardial injury.

Characteristics	Overall (n = 1026)	No injury (n = 465)	Injury (n = 561)	*p*
Male n (%)	660 (64.3)	276 (59.4)	384 (68.5)	0.003
Age (median [IQR]) year	69 [56, 79]	63 [51, 72]	74 [60, 82]	< 0.001
Age group n (%)				< 0.001
≤ 65	421 (41.0)	246 (52.9)	175 (31.2)	
66-80	272 (26.5)	142 (30.5)	130 (23.2)	
> 80	333 (32.5)	77 (16.6)	256 (45.6)	
Admission temperature (median [IQR]) ℃	36.5 [36.3, 36.8]	36.5 [36.3, 36.7]	36.5 [36.3, 36.8]	< 0.001
Maximum temperature (median [IQR]) ℃	37.0 [36.8, 37.9]	36.9 [36.7, 37.4]	37.3 [36.9, 38.3]	< 0.001
BMI (median [IQR]) kg/m2	23.4 [21.1, 25.7]	23.8 [21.5, 25.8]	22.9 [20.3, 25.4]	0.001
Heart rate (median [IQR]) beats/min	88 [78, 100]	88 [79, 98]	89 [78, 102]	0.183
SBP (median [IQR]) mmHg	125 [112, 139]	122 [111, 135]	129 [112, 144]	< 0.001
Presenting symptoms				
Cough n (%)	697 (67.9)	289 (62.2)	408 (72.7)	< 0.001
Chest pain n (%)	246 (24.0)	114 (24.5)	132 (23.5)	0.768
Breath shortness n (%)	440 (42.9)	176 (37.8)	264 (47.1)	0.004
Nausea n (%)	143 (13.9)	45 (9.7)	98 (17.5)	< 0.001
Diarrhea n (%)	58 (5.7)	20 (4.3)	38 (6.8)	0.116
Fever n (%)	105 (10.2)	216 (46.5)	277 (49.4)	0.384
Palpitate n (%)	37 (3.6)	14 (3.0)	23 (4.1)	0.445
Dizziness n (%)	161 (15.7)	84 (18.1)	77 (13.7)	0.069
Fatigue n (%)	433 (42.2)	170 (36.6)	263 (46.9)	0.001
Muscle aches n (%)	107 (10.4)	47 (10.1)	60 (10.7)	0.838
COVID-19 classification n (%)				
mild	306 (29.8)	193 (41.5)	113 (20.1)	< 0.001
moderate	408 (39.8)	194 (41.7)	214 (38.1)	
serious	200 (19.5)	69 (14.8)	131 (23.4)	
critically	112 (10.9)	9 (1.9)	103 (18.4)	
Past medical history				
Smoking history n (%)	324 (32.2)	141 (31.1)	183 (33.2)	0.509
Drinking history n (%)	289 (28.6)	134 (29.3)	155 (28.1)	0.716
Vaccinate n (%)	481 (67.7)	240 (70.8)	241 (65.0)	0.114
Hypertension n (%)	522 (50.9)	187 (40.2)	335 (59.7)	< 0.001
Diabetes mellitus n (%)	315 (30.7)	114 (24.5)	201 (35.8)	< 0.001
Diabetic complications n (%)	62 (6.0)	25 (5.4)	37 (6.6)	0.494
Coronary heart disease n (%)	237 (23.1)	74 (15.9)	163 (29.1)	< 0.001
COPD n (%)	66 (6.4)	20 (4.3)	46 (8.2)	0.016
Chronic bronchitis n (%)	28 (2.7)	8 (1.7)	20 (3.6)	0.107
Renal transplantation n (%)	57 (5.6)	29 (6.2)	28 (5.0)	0.465
Dialysis history n (%)	78 (7.6)	14 (3.0)	64 (11.4)	< 0.001
Severe valvular disease n (%)	36 (3.5)	9 (1.9)	27 (4.8)	0.02
Malignancy n (%)	81 (7.9)	57 (12.3)	24 (4.3)	< 0.001
Complications				
Renal insufficiency n (%)	282 (27.5)	56 (12.0)	226 (40.3)	< 0.001
Liver insufficiency n (%)	165 (16.1)	59 (12.7)	106 (18.9)	0.009
Anamia n (%)	253 (24.7)	61 (13.1)	192 (34.2)	< 0.001
Bacterial pneumonia n (%)	179 (17.4)	44 (9.5)	135 (24.1)	< 0.001
Fungal pneumonia n (%)	104 (10.1)	22 (4.7)	82 (14.6)	< 0.001
Expiratory failure n (%)	261 (25.4)	54 (11.6)	207 (36.9)	< 0.001
ARDS n (%)	12 (1.2)	1 (0.2)	11 (2.0)	0.022
Coagulation dysfunction n (%)	834(81.3)	340(73.1)	494(88.1)	< 0.001
Hypokalemia n (%)	158 (15.4)	59 (12.7)	99 (17.6)	0.035
Hyponatremia n (%)	92 (9.0)	24 (5.2)	68 (12.1)	< 0.001
Hypoproteinemia n (%)	332 (32.4)	87 (18.7)	245 (43.7)	< 0.001
Laboratory testing				
Oxygenation index (median [IQR]) mmHg	343 [252, 412]	371 [297, 433]	319 [213, 399]	< 0.001
Lactic acid (median [IQR]) mmol/L	3.7 [2.0, 8.0]	2.7 [1.7, 5.7]	4.7 [2.5, 9.1]	< 0.001
Oxygen saturation (median [IQR]) %	97.3 [94.3, 98.6]	98.0 [96.5, 98.8]	96.7 [91.9, 98.5]	< 0.001
Peak NT-proBNP (median [IQR]) pg/mL	693.1 [165.5, 3618.3]	177.4 [67.5, 489.4]	2237.0 [542.9, 7896.0]	< 0.001
WBC (median [IQR]) 10^9/L	6.5 [4.7, 9.2]	5.7 [4.4, 8.0]	7.4 [5.0, 10.4]	< 0.001
RBC (median [IQR])10^{12}/L	3.9 [3.4, 4.4]	4.0 [3.6, 4.4]	3.8 [3.3, 4.3]	< 0.001
Hemoglobin (median [IQR]) g/L	119 [104, 133]	122 [109, 134]	115 [100, 130]	< 0.001
Platelet (median [IQR]) 10^9/L	185 [135, 252]	196 [144, 260]	175 [128, 234]	< 0.001
Neutrophil (median [IQR])10^9/L	4.76 [3.21, 7.51]	3.97 [2.83, 6.10]	5.56 [3.54, 8.86]	< 0.001
Lymphocyte (median [IQR]) 10^9/L	0.96 [0.59, 1.46]	1.06 [0.74, 1.48]	0.85 [0.51, 1.41]	< 0.001
Hs-CRP (median [IQR]) mg/L	37.30 [9.17, 92.22]	20.95 [3.92, 68.75]	51.90 [14.65, 108.75]	< 0.001
Procalcitonin (median [IQR]) ng/L	0.12 [0.06, 0.46]	0.08 [0.05, 0.16]	0.20 [0.08, 0.76]	< 0.001
Interleukin-6 (median [IQR]) pg/ml	24.67 [8.08, 68.47]	13.89 [5.08, 39.13]	37.91 [13.47, 100.82]	< 0.001
Albumin (median [IQR]) g/L	33.10 [29.50, 37.10]	35.20 [31.90, 38.80]	31.40 [28.40, 35.60]	< 0.001
LDL-C (median [IQR]) mmol/L	2.45 [1.95, 3.16]	2.69 [2.09, 3.29]	2.27 [1.75, 2.96]	< 0.001
Adverse events n (%)	262(25.5)	55 (11.8)	207 (36.9)	< 0.001
Invasive ventilator n (%)	100(9.8)	7(1.5)	93(16.6)	< 0.001
Transfer to ICU n (%)	154(15.0)	37(8.0)	117(20.9)	< 0.001
Automatic discharge n (%)	143(13.9)	17(3.7)	126(22.5)	< 0.001
Death in-hospital n (%)	33(3.2)	1(0.2)	32(5.7)	< 0.001
Length of stay (median [IQR]) day	10 [7, 14]	10 [7, 14]	10 [7, 15]	0.093

BMI: body mass index; SBP: systolic blood pressure; COPD: chronic obstructive pulmonary disease; ARDS: acute respiratory distress syndrome; WBC: white blood cell; RBC: red blood cell; Hs-CRP: hypersensitive C-reactive protein; LDL-C: low-density lipoprotein cholesterol; ICU: Intensive care unit.

X-tile software was utilized to perform risk stratification. Missing values were filled in using multiple imputations. A 2-tailed *p* less than 0.05 was considered statistically significant. Kaplan-Meier methods were used to compute the survival analyses. All analyses were performed using SPSS software (version 25.0; IBM Corporation, Armonk, NY, USA), X-tile software (Version 3.6.1), and R software (version 4.3.1).

Results

Patients' characteristics

Table 1 shows the information, including demographics, clinical characteristics, and laboratory characteristics. The median age was 69 (56, 79) years, with elderly and men contributing more than half of the total. Most patients presented with cough (697, 67.9%), shortness of breath (440, 42.9%), fatigue (433, 42.2%), chest pain (246, 24.0%), and dizziness (161, 15.7%). COVID-19 was mostly classified as mild (306, 29.8%) and moderate (408, 39.8%). Hypertension (522, 50.9%), diabetes mellitus (315, 30.7%), and coronary heart disease (237, 23.1%) were the most common comorbidities. Both the admission and maximum body temperature were below 37.0℃.

Of the 1026 patients, 561 patients (54.7%) had myocardial injury, and 465 patients (45.3%) did not. Compared with patients without myocardial injury, those with myocardial injury were older (74, 60-82) years vs. 63 (51-72) years, ($p < 0.001$) and had a higher body temperature at admission and during hospitalization 36.5 (36.3-36.8) ℃ vs. 36.5 (36.3-36.7) ℃ or 37.3 (36.9-38.3) ℃ vs. 36.9 (36.7-37.4) ℃; $p < 0.001$). Cough (408, 72.7% vs. 289, 62.2%; $p < 0.001$), nausea (98, 17.5% vs. 45, 9.7%; $p < 0.001$), shortness of breath (264, 47.1% vs. 176, 37.8%; $p = 0.004$) and fatigue (263, 46.9% vs. 170, 36.6%; $p = 0.001$) were more common symptoms in patients with myocardial injury. Compared with patients without myocardial injury, those with myocardial injury were more likely to have a higher severity in the scale of COVID-19 classification ($p < 0.001$).

In addition, chronic cardiovascular diseases (including hypertension, (335, 59.7% vs 187, 40.2%), diabetes mellitus (201, 35.8% vs. 114, 24.5%), and coronary artery disease (163, 29.1% vs. 74, 15.9%); all $p < 0.001$), COPD (46, 8.2% vs. 20, 4.3%; $p < 0.05$), severe valvular heart disease (27, 4.8% vs. 9, 1.9%; $p < 0.05$), and dialysis history (64, 11.4% vs. 14, 3.0%; $p < 0.001$) were present more often in patients with myocardial injury. The difference was not statistically significant between patients with and without myocardial injury when considering smoking (183, 33.2% vs. 141, 31.1%; $p = 0.509$), drinking history (155, 28.1% vs. 134, 29.3%; $p = 0.714$), and dichotomous vaccination history (241, 65.0% vs. 240, 70.8%; $p = 0.144$).

Laboratory testing

In all the 1026 patients, the median values of P/F ratio (343 [252, 412] mm hg) and serum albumin (33.10 [29.50, 37.10] g/L) were reduced, and serum lactic acid

Table 2. Electrocardiographic and echocardiographic characteristics of Omicron variant infected patients with or without myocardial injury.

Characteristics	Overall (n = 635)	No injury (n = 242)	Injury (n = 393)	*p*
Electrocardiogram at presentation				
ECG abnormality n (%)	359 (63.1)	111 (51.2)	248 (70.5)	< 0.001
Premature beat n (%)	70 (12.3)	19 (8.7)	51 (14.5)	0.056
Conduction disturbances n (%)	94 (16.5)	26 (12.0)	68 (19.3)	0.030
Atrioventricular block n (%)				**0.407**
I	54 (9.5)	17 (7.8)	37 (10.5)	
II	1 (0.2)	0 (0.0)	1 (0.3)	
Bundle branch block n (%)	44 (7.7)	11 (5.0)	33 (9.4)	0.085
ST-T changed* n (%)	224 (39.2)	59 (27.1)	165 (46.7)	< 0.001
Arrhythmogenesis n (%)	214 (36.9)	52 (23.9)	162 (44.8)	< 0.001
Sinus tachycardia n (%)	102 (17.9)	20 (9.2)	82 (23.2)	< 0.001
Sinus bradycardia n (%)	21 (3.7)	7 (3.2)	14 (4.0)	0.813
Atrial fibrillation (%)	63 (11.0)	12 (5.5)	51 (14.4)	0.001
Echocardiographic characteristics				
RV (median [IQR]) mm	20 [19, 20]	20 [19, 20]	20 [19, 20]	0.932
PA (median [IQR]) mm	20 [20, 22]	20 [20, 22]	21 [20, 23]	0.071
IVS (median [IQR]) mm	11.0 [9.9, 12.1]	10.7 [9.6, 11.6]	11.0 [10.0, 12.3]	0.012
RA (median [IQR]) mm	35 [32, 37]	34 [31, 37]	35 [32, 38]	0.002
LA (median [IQR]) mm	36 [32, 38]	35 [32, 38]	36 [33, 39]	0.023
LV (median [IQR]) mm	45 [42, 48]	46 [42, 48.75]	45 [42, 48]	0.197
LV posterior wall (median [IQR]) mm	9.8 [8.9, 10.5]	9.6 [8.9, 10.4]	9.9 [9.0, 10.6]	0.105
LVEF (median [IQR]) %	63 [57, 67]	64 [59, 68]	61 [56, 66]	< 0.001
Pericardial effusion (%)	40 (6.4)	7 (3.0)	33 (8.5)	0.010

RV: right ventricular; PA: pulmonary artery; IVS interventricular septum; RA: right atrium; LA: left atrium; LV: left ventricular; LVEF: left ventricular ejection fraction. *Defined as the composite of ST-segment elevations, depressions, or T-wave inversions.

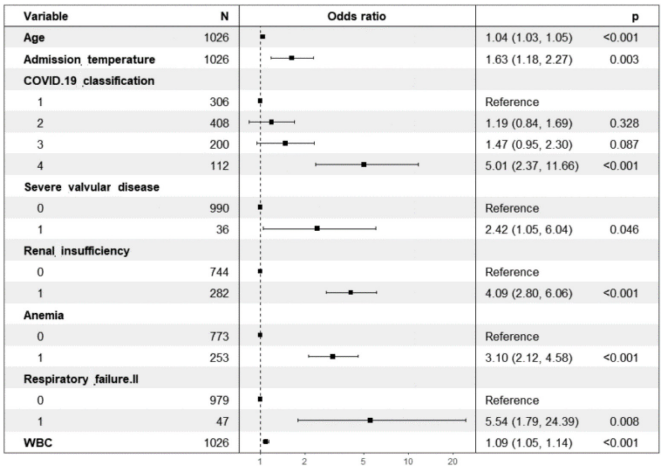
(3.7 [2.0, 8.0] mmol/L, highly sensitive c-reactive protein (HS-CRP) (37.30 [9.17, 92.22] mg/L), interleukin-6 (IL-6) (24.67 [8.08, 68.47] pg/mL) were increased. The median values of the other laboratory indicators were within the normal range, such as oxygen saturation, red blood cell, hemoglobin, platelet, WBC, neutrophil, lymphocyte, procalcitonin, serum albumin, and low-density lipoprotein cholesterol (LDL-C). Patients with myocardial injury usually had a lower P/F ratio and oxygen saturation, RBC, hemoglobin, platelet, lymphocyte, serum albumin, LDL-C, while higher serum lactic acid, peak NT-proBNP, WBC, neutrophil, HS-CRP, procalcitonin, and IL-6 (all $p < 0.001$) (Table 1).

Electrocardiogram and echocardiography findings

Overall, a total of 635 patients underwent echocardiography and electrocardiogram testing. Of these, 359 patients (63.1%) had abnormal ECG findings. ST-segment alterations, arrhythmia (sinus tachycardia, sinus bradycardia and atrial fibrillation, and ventricular tachycardia), and conduction disturbances (bundle branch block and atrioventricular block) occurred in 224 patients (39.2%), 214 patients (36.9%), and 94 patients (16.5%), respectively. In terms of echocardiographic findings, the median left ventricular ejection fraction (LVEF) was 63 [57, 67] %, and 40 patients (6.4%) had pericardial effusion. The volume of the left atrium was larger (36 [32, 38] mm), and other values were within the normal ranges.

Patients with myocardial injury were more likely to have abnormal ECG findings than those without myocardial injury ($p < 0.001$). The echocardiogram showed that patients with myocardial injury also had a thicker interventricular septum, larger atrial size (left and right atrium), lower LVEF, and higher frequency of pericardial effusion (all $p < 0.05$) (Table 2).

Figure 1. Multivariable adjusted logistic regression model for risk of myocardial injury in patients with Omicron variant infection.



Complications and clinical outcomes

Overall, 12 patients (1.2%) had acute respiratory distress syndrome (ARDS). Other common complications included coagulation disorders (834, 81.3%), serum hypoproteinemia (332, 32.4%), renal insufficiency (282, 27.5%), respiratory failure (261, 25.4%), liver insufficiency (165, 16.1%), anemia (253, 24.7%), bacterial pneumonia (179, 17.4%), fungal pneumonia (104, 10.1%), hypokalemia (158, 15.4%), and hyponatremia (92, 9.0%). A total of 262 patients (25.5%) had adverse events during hospitalization, of which 154 patients (15.0%) were admitted to the intensive care unit, 100 patients (9.8%) were put on invasive ventilation, 143 patients (13.9%) were discharged against medical advice, and 33 patients (3.2%) died in the hospital.

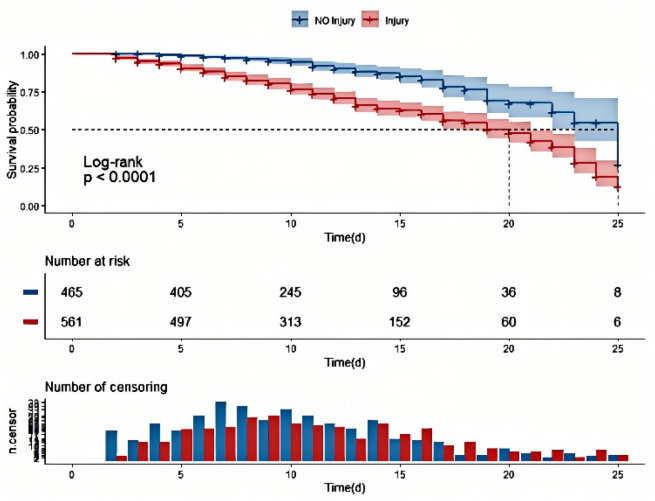
Complications occurred more frequently among patients with myocardial injury than those without, including ARDS (11, 2.0% vs. 1, 0.2%; $p < 0.05$), hypoproteinemia (245, 43.7% vs. 87, 18.7%; $p < 0.001$), renal insufficiency (226, 40.3% vs. 56, 12.0%; $p < 0.001$), respiratory failure (207, 36.9% vs. 54, 11.5%; $p < 0.001$), liver insufficiency (106, 18.9% vs. 59, 12.7%; $p < 0.001$), anemia (192, 34.2% vs. 61, 13.1%; $p < 0.001$), bacterial pneumonia (135, 24.1% vs. 44, 9.5%; $p < 0.001$), fungal pneumonia (82, 14.6% vs. 22, 4.7%; $p < 0.001$), hypokalemia (99, 17.6% vs. 59, 12.7%; $p < 0.05$) and coagulation disorders (494, 88.1% vs. 340, 73.1%; all $p < 0.05$) (Table 1).

Myocardial injury and adverse events

The univariate logistic regression analysis for risk of myocardial injury is shown in Supplementary Table 1. The multivariable adjusted logistic regression model showed older age (OR 1.04 [1.03, 1.05], $p < 0.001$), body temperature at admission (OR 1.64 [1.19, 2.29], $p = 0.003$), critical COVID-19 (OR 4.96 [2.34, 11.54], $p < 0.001$), severe valvular heart disease (OR 2.46 [1.06, 6.12], $p = 0.042$), renal insufficiency (OR 4.04 [2.75, 6.01], $p < 0.001$), anemia (OR 3.10 [2.12, 4.58], $p < 0.001$), type II respiratory failure (OR 5.54 [1.79, 24.39], $p = 0.008$) and higher WBC count (OR 1.09 [1.05, 1.14], $p < 0.001$) were significantly association with myocardial injury in patients with Omicron variant infection (Figure 1).

A total of 262 patients (25.5%) presented adverse events during hospitalization. As shown in Table 1, the rate of in-hospital adverse events was higher in patients with myocardial injury were 36.9% (207 patients) and

Figure 2. Kaplan-Meier survival curves for adverse events during hospitalization. Patients with myocardial injury had a higher rate of mortality in the log-rank test.



11.8% (55 patients), respectively, than in those without it ($p < 0.001$). The median length of stay was 10 (7-14) days, and it was similar between the two groups, 10 (7-15) days vs. 10 (7-14) days; $p = 0.093$). According to the Kaplan-Meier survival curves for myocardial injury and adverse events (Figure 2, $p < 0.001$) and multivariable adjusted Cox regression analysis, myocardial injury may be an independent risk factor for predicting the adverse events in patients with Omicron variant infection (HR 1.79 [1.28, 2.48], $p = 0.001$). X-tile software was utilized to classify all patients into high-risk (n = 108) and low-risk (n = 918) for myocardial injury according to the value of cTnT, and the optimal cut-off value to define elevated troponin was cTnT ≥ 0.09 μg/L in patients with Omicron variant

Figure 4. Multivariable Cox-regression analysis for the risk of adverse events in patients with Omicron variant infection.

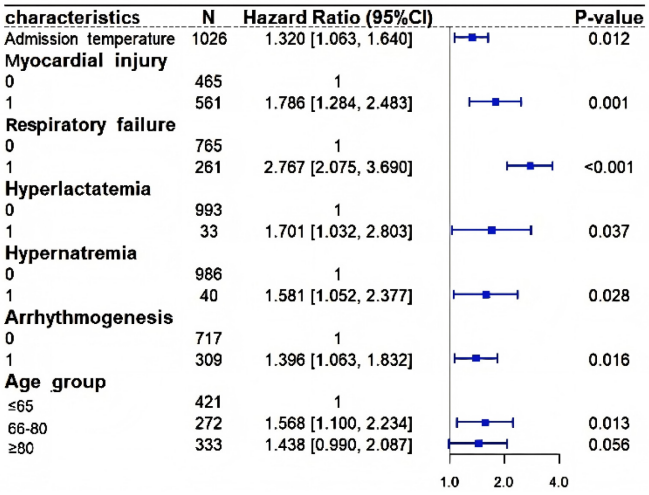
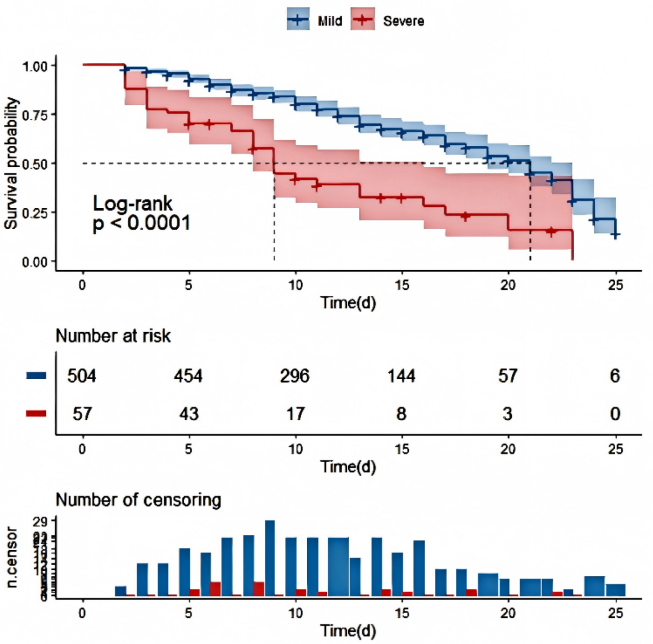


Figure 3. Adverse events between admission cTnT < 0.18 μg/L vs. > 0.18 μg/L in patients with Omicron variant infection during hospitalization.



infection (Supplementary Figure 1). And then patients with myocardial injury were divided into two groups, mild (n = 504) and severe (n = 57), according to the optimal cut-off value determined (for cTnT, mild: < 0.18 μg/L, and severe: > 0.18 μg/L) (Supplementary Figure 2). Kaplan-Meier curves for admission cTnT and adverse events were plotted (Figure 3), indicating that the concentrations of admission cTnT above 0.18 μg/L were significantly associated with a greater risk of adverse events ($p < 0.001$). After adjustment for clinical variables (including age, gender, vital signs, vaccination history, patient history and complications) the multivariate Cox regression model demonstrated that myocardial injury (HR 1.79 [1.28, 2.48], $p = 0.001$), body temperature at admission (HR 1.32 [1.06, 1.64], $p = 0.012$), age among 65 years to 80 years (HR 1.57 [1.10, 2.23], $p = 0.013$), respiratory failure (HR 2.77 [2.08, 3.69], $p < 0.001$), hyperlactatemia (HR 1.70 [1.03, 2.80], $p = 0.037$), hypernatremia (HR 1.58 [1.05, 2.38], $p = 0.028$) and arrhythmia (HR 1.40 [1.06, 1.83], $p = 0.016$) were significantly associated with the higher risk of adverse events in hospital patients with Omicron variant infection (Figure 4).

Discussion

Previous studies have shown that SARS-CoV-2 could cause multi-organ dysfunction, particularly in the cardiovascular system, often linked to poor clinical outcomes [16,17] and ongoing myocardial

inflammation [18]. However, few studies have characterized myocardial injury specifically tied to the Omicron variant infection. This retrospective cohort study of over 1,000 patients shows myocardial injury is frequently prevalent among those infected with the Omicron variant. Patients with comorbidities (chronic cardiovascular disease, COPD, or renal insufficiency) are more susceptible. Myocardial injury correlates with a higher risk of adverse events: mechanical ventilation, ICU admission, critical presentation, and in-hospital death.

Earlier reports note that the incidence of myocardial injury in COVID-19 patients with SARS-CoV-2 ranges from 7% to 63% [19,20]. This rate was 54.7% in our study, aligning with prior cardiac MRI studies [21]. The present postulated mechanisms of COVID-19 leading to myocardial injury include inflammatory cascade, disseminated intravascular coagulation and thrombosis, direct viral entry via ACE2 receptors, hypoxemia with increased metabolic demands, and acute coronary syndrome from acute inflammation-triggered atheromas destabilization [22].

Consistent with prior findings, our data showed that aging increases susceptibility, especially beyond age 65. According to several studies, COVID-19 vaccination could prevent morbidity due to acute cardiovascular diseases associated with SARS-CoV-2 infection and reduce mortality [23,24]. In our cohort, 67.7% were vaccinated ≥ 1 time, yet vaccination did not prevent myocardial injury. A previous study found booster doses against the ancestral strains would not further significantly enhance individual humoral responses against the newer Omicron subvariants [3]. A meta-analysis confirmed emerging SARS-CoV-2 variants had increased transmissibility, and full vaccination was less associated with reductions in susceptibility and infectiousness for the Omicron variant, versus Alpha[25].

Average LVEF was similar between patients with and without myocardial injury, indicating preserved cardiac systolic function-consistent with a meta-analysis [26]. COVID-19 was reported as an independent indicator of atrial fibrillation [27,28]; one meta-analysis (30 studies) reported about 10.1% arrhythmia incidence in COVID-19 patients [28]. We found arrhythmias were associated with increased risk of adverse events in the setting of the Omicron variant.

After adjusting for relevant clinical factors, the Cox regression model showed that myocardial injury significantly predicts worse outcomes in the Omicron-infected patients. Respiratory failure and hypernatremia also correlated with poor prognosis, aligning with prior studies [29,30].

Furthermore, we identified an optimal cut-off value of cTnT (≥ 0.09 μg/L) that may serve as a new value for recognizing high risk of myocardial injury in patients with the Omicron variant infection. The conventional cut-off value (< 0.014 μg/L) may over-classify risk, leading to stringent monitoring, prolonged stays, or unnecessary interventions. cTnT ≥ 0.18 μg/L significantly increased the odds of adverse events, supporting targeted medical interventions. A similar study also evaluated the optimal cut-off value of cardiac biomarkers to predict increased risk of 30-day all-cause mortality [31], underscoring early risk stratification, though more data are needed.

This study has several limitations. First, the data collection was retrospective and from a single medical center, which may cause ascertainment bias. Second, cardiac magnetic resonance imaging analysis to identify myocardial damage was largely lacking. Third, due to the limitations of time, economic conditions, and clinical facilities, we can only speculate that all patients were infected with this virus variant during the Omicron pandemic and did not test for the virus variant; perhaps some of them were infected with other types of COVID-19 variants. Additionally, not all COVID-19 patients received troponin testing in medical practice; therefore, these results may overestimate the actual incidence of myocardial injury. In addition, uncorrected multiple comparisons may result in an elevated Class I error rate, but given the exploratory model development nature of this study, the recommendations of the TRIPOD statement were followed, and no corrections for multiple comparisons were made during predictor screening. Therefore, the current model's parameters need to be validated in an independent cohort before being put into clinical use, particularly in light of the new mutant variant. Further prospective cohort studies with more detailed information are also required. This work provides reliable and scientific information to increase understanding of clinical characteristics of COVID-19 and highlight the important role of myocardial injury in patients' management.

Conclusions

In the current multicenter international study, a high proportion of patients with confirmed cases of SARS-CoV-2 infection presented with myocardial injury, which was a risk factor for poor prognosis. In this retrospective cohort study of hospitalized patients with Omicron variant infection who underwent cTnT testing due to specific clinical suspicion, myocardial injury is not only a highly prevalent complication but also

associated with a higher risk of adverse events. Identifying myocardial injury, controlling risk factors for myocardial injury, and stratifying patients according to troponin levels may be effective in preventing adverse events in patients with Omicron variant infection. While the cardiac effects of this wave of Omicron infection were mostly mild to moderate, the myocardial injury experienced by patients with COVID-19 appears to be long-lasting, and the long-term effects remain unknown. Future studies may need to focus on the long-term quality of survival and the risk of complications in surviving patients.

Data availability

The data and code supporting the findings of the present study are available via the corresponding author upon reasonable request.

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Conflict of interest

No conflict of interest is declared.

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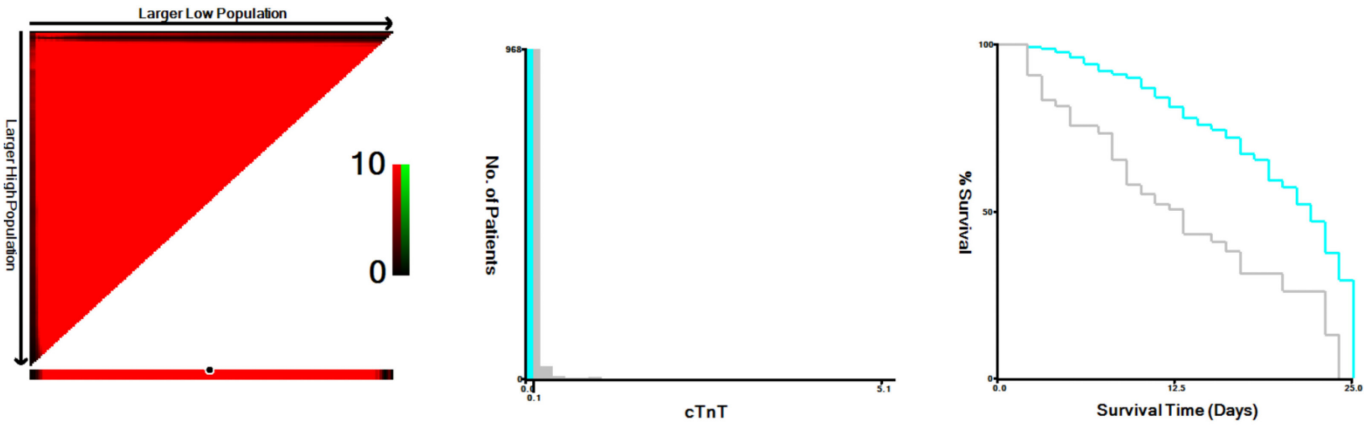
Annex – Supplementary Items

Supplementary Table 1. Univariate logistic regression analysis for risk of myocardial injury in patients with Omicron variant infection.

Variable	OR	95% CI	*p*
Male	1.49	1.15 - 1.92	0.002
Age	1.04	1.03 - 1.04	< 0.001
Body temperature at admission	1.81	1.37 - 2.41	< 0.001
Respiratory rate	1.18	1.10 - 1.26	< 0.001
Pulse	1.01	1.00 - 1.01	0.049
Systolic blood pressure	1.01	1.00 - 1.02	0.001
COVID-19 classification			
mild	reference		
moderate	1.88	1.39 - 2.55	< 0.001
severe	3.24	2.23 - 4.71	< 0.001
critical	19.55	9.52 - 40.15	<.001
Vaccination	0.72	0.55 - 0.95	0.02
Hypertension	2.2	1.72 - 2.83	< 0.001
Diabetes	1.72	1.31 - 2.26	< 0.001
Coronary disease	2.16	1.59 - 2.94	< 0.001
COPD	1.99	1.16 - 3.41	0.013
Severe valvular disease	2.56	1.19 - 5.50	0.016
ARDS	9.28	1.19 - 72.09	0.033
Renal insufficiency	4.93	3.56 - 6.83	< 0.001
Hepatic dysfunction	1.6	1.13 - 2.26	0.007
Anemia			
0	reference		
1	3.56	2.39 - 5.30	< 0.001
2	3.02	1.80 - 5.08	< 0.001
3	4.65	1.55 - 13.95	0.006
Lung cancer	0.45	0.25 - 0.81	0.008
Respiratory failure			
Type I	3.32	2.36 - 4.69	< 0.001
Type II	13.11	4.04 - 42.49	< 0.001
Hyperlactaemia	6.28	2.19 - 18.00	<.001
Hypocythemia	1.47	1.04 - 2.09	0.029
Hypernatremia	1.76	0.90 - 3.45	0.101
Hyponatremia	2.53	1.56 - 4.11	< 0.001
Hypoproteinemia	3.37	2.53 - 4.49	< 0.001
WBC	1.13	1.09 - 1.17	< 0.001
Lymphocytes	0.95	0.94 - 0.96	< 0.001
Hemoglobin	0.99	0.98 - 0.99	< 0.001
Neutrophils	1.15	1.11 - 1.20	< 0.001
RBC	0.8	0.70 - 0.92	0.002

COPD: chronic obstructive pulmonary disease; ARDS: acute respiratory distress syndrome; WBC: white blood cell; RBC: red blood cell.

Supplementary Figure 1. Patients with Omicron variant infection were divided into high-risk (n = 108) and low-risk (n = 918) groups according to optimal cut-off values cTnT < 0.09μg/L. Kaplan-Meier curves for adverse events were plotted.



Supplementary Figure 2. Myocardial injury patients were divided into two groups according to optimal cut-off values (mild: cTnT < 0.18μg/L; severe: cTnT > 0.18 μg/L). Kaplan-Meier curves for adverse events were plotted.