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ORIGINAL ARTICLE

Cardiac Injury and Outcomes of Patients With COVID-19 in New York City

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Background Prior studies demonstrated that elevated troponin in patients with COVID-19 was associated with increased in-hospital mortality. However, the association of cardiac injury and electrocardiogram (ECG) changes remains unclear. The aim of this study was to investigate the association of cardiac injury with ECG abnormality and with in-hospital mortality.

Methods We conducted a retrospective cohort study of patients who were hospitalised with COVID-19 between 13 March and 31 March 2020. Those patients with troponin I measurement were included in the study and divided into those who had elevated troponin I (cardiac injury group) and those who did not (no cardiac injury group). Statistical analyses were performed to compare differences between the groups, and a multivariate logistic regression model was constructed to assess the effect of cardiac injury on in-hospital mortality.

Results One hundred and eight-one (181) patients were included, 54 of whom were in the cardiac injury group and 127 in the no cardiac injury group. The mean age was 64.0±16.6 years and 55.8% were male. The cardiac injury group was more likely to be older, have a history of coronary artery disease, atrial fibrillation and congestive heart failure compared to the no cardiac injury group (all $p<0.05$); there was no difference in presence of chest pain (cardiac injury group versus no cardiac injury group: 17.0% versus 22.5%, $p=0.92$); the cardiac injury group had a significantly higher value of brain natriuretic peptide, procalcitonin, interleukin-6 and D-dimer (all $p<0.05$); they had numerically more frequent ECG abnormalities such as T wave inversion (13.2% versus 7.5%, $p=0.23$) and ST depression (1.9% versus 0.0%, $p=0.13$) although statistically not significant; they had significantly higher in-hospital mortality (42.3% versus 12.6%, $p<0.001$). With a multivariate logistic regression model, age (odds ratio [95% confidence interval]: 1.033 [1.002–1.065], $p=0.034$) and cardiac injury (3.25 [1.40–7.54], $p=0.006$) were independent predictors of in-hospital mortality.

Conclusions Patients with COVID-19 with elevated troponin I had a relatively low proportion of chest pain and ECG abnormality. Cardiac injury was independently associated with in-hospital mortality.

Keywords COVID-19 • Coronavirus • New York • Troponin

Introduction

Coronavirus disease 2019 (COVID-19), caused by a novel coronavirus severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has emerged as a global public health emergency since December 2019 [1]. Prior studies demonstrated that 12–28% of patients with COVID-19 presented with acute cardiac injury defined as elevated troponin I, and that cardiac injury was associated with increased in-hospital mortality [2–5]. Guo et al. noted that 64% of patients had

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Table 1 Baseline characteristics at admission.

	No Cardiac Injury Group N=127	Cardiac Injury Group N=54	P-value
Age	60 [49, 71]	73.5 [67, 83]	<0.001
Male	54.3% (69/127)	59.3% (32/54)	0.54
Body mass index	28.7 [25.1, 33.2]	28.1 [25.6, 31.9]	0.79
Race	White 38.6% (49/127)	White 31.5% (17/54)	0.78
	Black 19.7% (25/127)	Black 24.1% (13/54)	
	Asian 10.2% (13/127)	Asian 11.1% (6/54)	
	Others 29.1% (37/127)	Others 33.3% (18/54)	
	Hispanic 46.3% (57/123)	Hispanic 46.3% (25/54)	0.996
Symptoms			
Fever	70.9% (90/127)	42.3% (22/52)	<0.001
Myalgia	19.7% (25/127)	3.8% (2/53)	0.38
Chest pain	22.8% (29/127)	17.0% (9/53)	0.92
Malaise	27.6% (35/127)	32.1% (17/53)	0.54
Sore throat	8.7% (11/127)	0.0% (0/53)	0.027
Runny nose	7.9% (10/126)	5.7% (3/53)	0.59
Dyspnoea	66.9% (85/127)	56.6% (30/53)	0.19
Cough	74.0% (94/127)	60.4% (32/53)	0.069
Sputum	25.4% (32/126)	8.7% (4/46)	0.049
Abdominal pain	11.8% (15/127)	7.5% (4/53)	0.47
Nausea/Vomiting	29.1% (37/127)	22.6% (12/53)	0.37
Diarrhoea	28.3% (36/127)	13.2% (7/53)	0.030
Initial Vital Signs			
Temperature (°C)	36.8 [36.3, 37.7]	36.9 [36.2, 37.5]	0.71
Heart rate (beat/min.)	97 [86, 110]	86.5 [75.5, 109]	0.074
SBP (mmHg)	133 [117, 148]	137 [109, 155]	0.90
DBP (mmHg)	77 [68, 86]	75 [64, 88]	0.57
MAP (mmHg)	94.7 [86.7, 106]	93.5 [80.8, 111]	0.97
RR (/min.)	20 [18, 24]	20 [18, 24]	0.59
SpO_2 (%) at room air	95 [93, 98]	95 [92, 97.3]	0.71
Altered mental status	5.6% (7/126)	26.4% (14/53)	<0.001
Aspirin	18.9% (24/127)	41.5% (22/53)	0.002
Anticoagulation	6.3% (8/127)	28.3% (15/53)	<0.001
ACEI	15.0% (19/127)	24.5% (13/53)	0.13
ARB	15.1% (19/126)	26.4% (14/53)	0.074
Steroid use at home	0.8% (1/126)	5.6% (3/54)	0.047
Comorbidities			
Hypertension	53.5% (68/127)	90.7% (49/54)	<0.001
Hyperlipidaemia	36.2% (46/127)	63.0% (34/54)	0.001
DM	29.1% (37/127)	46.3% (25/54)	0.026
COPD	5.5% (7/127)	14.8% (8/54)	0.038
Asthma	11.0% (14/127)	12.5% (6/48)	0.99
CVA	3.9% (5/127)	14.8% (8/54)	0.010
PVD	4.7% (6/127)	13.0% (7/54)	<0.001
Dialysis	0.8% (1/127)	9.3% (5/54)	0.004
Cirrhosis	1.6% (2/127)	3.7% (2/54)	0.37
CAD	10.0% (9/127)	44.4% (27/54)	<0.001
Previous PCI	3.1% (4/127)	33.3% (18/54)	<0.001
Previous CABG	0.8% (1/127)	14.8% (8/54)	<0.001
Previous PE/DVT	7.9% (10/127)	3.7% (2/54)	0.30
History of AF	3.9% (5/127)	25.9% (14/54)	<0.001
Previous CHF	4.0% (5/126)	35.2% (19/54)	<0.001

Table 1 (continued).

	No Cardiac Injury Group N=127	Cardiac Injury Group N=54	P-value
History of cancer	9.4% (12/127)	5.6% (3/54)	0.39
HIV	4.7% (6/127)	5.6% (3/54)	0.81
Smoker			0.065
Never smoker	73.8% (93/126)	58.8% (30/51)	
Former smoker	22.2% (28/126)	39.2% (20/51)	
Current smoker	4.0% (5/126)	2.0% (1/51)	

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; RR, respiratory rate; SIRS, systematic inflammatory response syndrome; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; DM, diabetes mellitus; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; PVD, peripheral vascular disease; CKD, chronic kidney disease; CAD, coronary artery disease; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; PE/DVT, pulmonary embolism/deep vein thrombosis; AF, atrial fibrillation; CHF, congestive heart failure; HIV, human immunodeficiency virus.

evidence of myocardial ischaemia on electrocardiogram (ECG), although only 27% of patients with cardiac injury were assessed with ECG [4]. It is hypothesised that elevation of troponin in patients with COVID-19 stems from direct damage of cardiomyocytes from SARS-CoV-2 or inflammatory pathogenesis [6–10] rather than typical large coronary artery occlusion; however, the exact mechanism remains unclear. The aim of this study was to investigate the association of cardiac injury with ECG abnormality as well as in-hospital mortality in our diverse multi-ethnic population in New York City, the global epicentre of this pandemic to date.

Materials and Methods

This study was a retrospective cohort study using the data of patients hospitalised from 13 March to 31 March 2020 with diagnosis of COVID-19 at Mount Sinai Beth Israel, an urban academic medical centre in downtown Manhattan [11]. Of note, the first case of hospitalisation occurred on 13 March 2020. The study protocol was approved by the institutional review board and conducted in accordance with the principles of the Declaration of Helsinki. Diagnosis of COVID-19 required detection of SARS-CoV-2 in a nasopharyngeal swab specimen using reverse transcriptase polymerase chain reaction. The decision whether to admit the patients diagnosed with COVID-19 was provider dependent and not based on any specific predetermined criteria. Since our study was a retrospective observational study, it did not require specific tests including troponin I, ECG, or any other treatments.

A retrospective review of patient electronic medical records was conducted mainly by two authors (R.O. and T.M.) for demographics, comorbidities, and clinical outcomes including troponin I and ECG findings. Regarding ECG findings, T wave inversions were subdivided into inferior (II, III, aVF), anterior (V_1-V_4), lateral (I, aVL), and apex (V_5, V_6); we also collected the data of upright T wave in aVR [12].

Among the patients who were admitted with COVID-19, those who had troponin I measured upon admission were included in our study. They were divided into two groups: those with cardiac injury (cardiac injury group) and those without cardiac injury (no cardiac injury group). Cardiac injury was defined as troponin I greater than 0.031 ng/ml, which was the institutional cut-off and the 99th percentile upper reference limit [2,13]. Laboratory data, radiographic findings reviewed by attending radiologists, and characteristics of hospital stay such as total length of stay and ICU stay were collected. Our institution initially used hydroxychloroquine for moderate cases which was defined as radiologic evidence of pneumonia or oxygen saturation <94%.

Continuous variables were presented as mean±standard deviation or median (interquartile range), as appropriate for the data distribution; categorical variables were expressed as percentages. The changes from baseline in continuous variables were evaluated using Student's *t*-test or Mann-Whitney U test; the χ^2 or Fisher's exact *t*-test was used for analysing categorical variables. A multivariate logistic regression model was constructed in order to assess the association of cardiac injury with in-hospital mortality. We included age and troponin as variables because they have been reported as independent predictors of mortality in patients with COVID-19 [3,14]. Then, we performed a stepwise multivariate analysis including age and cardiac injury, and other baseline characteristics such as hypertension, diabetes mellitus, history of coronary artery disease, congestive heart failure, peripheral vascular disease, cerebrovascular disease, chronic obstructive pulmonary disease, and end-stage renal disease on dialysis. All statistical calculations and analyses were performed using SPSS (version 24, SPSS, Armonk, NY, USA), with p-values <0.05 considered statistically significant.

Results

Two hundred and twenty-four (224) patients were hospitalised with a diagnosis of COVID-19, 181 (80.8%) of whom had

Table 2 Baseline laboratory findings.

	No Cardiac Injury Group N=127	Cardiac Injury Group N=54	P-value
Complete blood count			
White blood cell, K/μL	7.0 [4.9, 9.2]	7.5 [5.5, 10.2]	0.28
Neutrophil, K/μL	5.2 [3.3, 7.1]	5.5 [3.9, 7.2]	0.31
Lymphocyte, K/μL	1.0 [0.8, 1.4]	0.80 [0.63, 1.38]	0.076
Haemoglobin, g/dL	14.1 [13.1, 15.2]	12.6 [11.0, 13.6]	<0.001
Platelet, K/μL	177 [133, 233]	148 [123, 171]	0.005
Biochemistry panel			
Glucose, mg/dL	116 [99, 149]	122 [103.8, 154]	0.50
BUN, mg/dL	15 [11, 22]	26 [17.8, 43]	<0.001
Creatinine, mg/dL	0.9 [0.71, 1.13]	1.41 [0.9, 2.16]	<0.001
AST, U/L	41.5 [28, 60.3]	44 [27, 68]	0.82
ALT, U/L	31.5 [19.3, 49.8]	26 [19, 44.5]	0.25
Total bilirubin, mg/dL	0.5 [0.4, 0.7]	0.6 [0.4, 0.9]	0.030
Albumin, g/dL	3.4 [3.1, 3.7]	3.2 [2.8, 3.6]	0.003
LDH, U/L	374 [271, 509]	409 [380, 545]	0.15
CPK, U/L	174 [65, 376]	140 [69, 441]	0.83
Coagulation			
PT-INR	1.0 [1.0, 1.1]	1.1 [1.0, 1.3]	0.004
APTT (s)	31.4 [28.4, 36.2]	33.3 [29.6, 38.4]	0.27
D-dimer, μg/mL	0.82 [0.58, 1.33]	2.03 [0.97, 3.08]	<0.001
Inflammation marker			
CRP or hsCRP, mg/L	70.7 [37.8, 141]	99.3 [42.8, 167]	0.21
Procalcitonin, ng/mL	0.11 [0.05, 0.24]	0.42 [0.11, 0.74]	<0.001
Ferritin, ng/mL	525 [303, 1,315]	670 [253, 1,428]	0.66
IL-6, pg/L	47.1 [22.1, 110]	87.9 [48.0, 140]	0.020
Others			
pH (venous blood gas)	7.41 [7.38, 7.44]	7.40 [7.33, 7.42]	0.026
Lactate, mmol/L	1.31 [1.08, 1.80]	1.50 [1.15, 1.95]	0.11
BNP, pg/mL	13.6 [10.0, 40.4]	191 [78, 720]	<0.001
Troponin I, ng/mL	0.01 [0.01, 0.01]	0.096 [0.059, 0.14]	<0.001
ECG			
AF	1.7% (2/119)	13.2% (7/53)	0.002
PAC	2.5% (3/120)	5.7% (3/53)	0.30
PVC	0.0% (0/120)	13.2% (7/53)	<0.001
CRBBB	3.3% (4/120)	11.3% (6/53)	0.038
CLBBB	0.8% (1/120)	3.8% (2/53)	0.172
TWI	7.5% (9/120)	13.2% (7/53)	0.23
TWI inferior	0.8% (1/120)	3.8% (2/53)	0.17
TWI anterior	5.8% (7/120)	1.9% (1/53)	0.26
TWI lateral	1.7% (2/120)	7.5% (4/53)	0.051
TWI apex	0.0% (0/120)	5.7% (3/53)	0.009
Upright T wave in aVR	0.0% (0/120)	0.0% (0/53)	-
ST depression	0.0% (0/120)	1.9% (1/53)	0.13
ST elevation in aVR	0.0% (0/120)	0.0% (0/53)	-
Chest X-ray	96.9% (123/127)	98.1% (53/54)	1.00
Multifocal pneumonia	59.3% (73/123)	56.6% (30/53)	0.73
Pleural effusion	4.1% (5/123)	17.0% (9/53)	0.006

Abbreviations: BUN, blood urea nitrogen; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; CPK, creatine kinase; PT-INR, prothrombin time-international normalised ratio; APTT, activated partial thromboplastin time; CRP, C-reactive protein; hsCRP, high sensitivity C-reactive protein; IL-6, interleukin-6; BNP, B type natriuretic peptide; ECG, electrocardiogram; AF, atrial fibrillation; CRBBB, complete right bundle branch block; CLBBB, complete left bundle branch block; PAC, premature atrial contraction; PVC, premature ventricular contraction; TWI, T wave inversion.

Table 3 In-hospital treatment and outcomes.

	No Cardiac Injury Group N=127	Cardiac Injury Group N=54	P-value
ICU stay	23.6% (30/127)	40.7% (22/54)	0.020
Hydroxychloroquine	86.6% (110/127)	83.3% (45/54)	0.57
Azithromycin	66.1% (84/127)	63.0% (34/54)	0.68
Steroids	29.9% (38/127)	29.6% (16/54)	0.97
Vasopressor	10.2% (13/127)	20.8% (11/53)	0.058
Dobutamine	1.6% (2/127)	3.8% (2/53)	0.36
Milrinone	0.8% (1/127)	0.0% (0/53)	0.52
Oxygen	69.3% (88/127)	81.1% (43/53)	0.10
NPPV	2.4% (3/127)	9.4% (5/53)	0.036
IMV	12.6% (16/127)	32.1% (17/53)	0.002
New initiation of dialysis	1.6% (2/125)	7.7% (4/52)	0.041
Liver failure	0.8% (1/127)	1.9% (1/53)	0.52
Cardiac catheterisation	0.0% (0/127)	1.9% (1/53)	0.12
VF/Pulseless VT	0.0% (0/127)	1.9% (1/53)	0.12
CPR	3.1% (4/127)	13.2% (7/53)	0.010
Length of stay	6 [4, 11]	8 [4.5, 15]	0.13
In-hospital mortality	12.7% (16/126)	42.3% (22/52)	<0.001

Abbreviations: ICU, intensive care unit; NPPV, noninvasive positive pressure ventilation; IMV, invasive mechanical ventilator; VT, ventricular tachycardia; STEMI, ST elevation myocardial infarction; VF, ventricular fibrillation; CPR, cardiopulmonary resuscitation.

measurement of troponin I upon admission and were included in the study. Among the 181 patients, the mean age was 64.0±16.6 and 55.8% were male; 36.4% were Caucasian, 21.0% were African American, 43.9% were Hispanic. Fifty-four (54) patients were categorised into the cardiac injury group and 127 into the no cardiac injury group.

Detailed baseline characteristics are shown in Table 1. The cardiac injury group was more likely to be older and have hypertension, and end-stage renal disease on dialysis, coronary artery disease as a previously known comorbidity (all p-value <0.05). Prior coronary revascularisation and known atrial fibrillation and congestive heart failure were also more frequently compared to no cardiac injury group (all p-value <0.05). Notably, both groups had non-significantly different, relatively low rates of chest pain (cardiac injury group versus no cardiac injury group: 17.0% versus 22.5%, p=0.92).

Table 2 describes baseline laboratory, ECG and radiographic findings. The cardiac injury group had significantly higher blood urea nitrogen, creatinine, creatine kinase, brain natriuretic peptide, D-dimer and inflammatory markers such as procalcitonin and interleukin-6, and lower haemoglobin compared to the no cardiac injury group (all p-value <0.05). Electrocardiograph was obtained in 95.6% of individuals. Electrocardiograph findings including T wave inversion and ST depression appeared numerically more frequent in the cardiac injury group but were not statistically significantly different (cardiac injury group versus no cardiac injury group; T wave inversion: 13.2% versus 7.5%, p=0.23; ST depression: 1.9% versus 0.0%, p=0.13). There was no significant difference of multifocal pneumonia on chest X-ray in both groups (cardiac injury group versus no cardiac injury group: 56.6% versus 59.3%, p=0.73).

Table 3 shows in-hospital treatment and outcomes. 32.1% of patients in the cardiac injury group required invasive mechanical ventilation (18.2% of total cohort). The cardiac injury group had significantly higher in-hospital mortality than the no cardiac injury group (42.3% versus 12.6%, p<0.001). Only one patient died with ventricular fibrillation/pulseless ventricular tachycardia in the overall group (4.5% of deceased patients among cardiac injury, 1.9% of cardiac injury).

The multivariate logistic regression analysis with stepwise method was performed with age, cardiac injury, hypertension, diabetes mellitus, history of coronary artery disease, congestive heart failure, peripheral vascular disease, cerebrovascular disease, chronic obstructive pulmonary disease, and end-stage renal disease on dialysis as variables. The final variables were age and cardiac injury. Age (odds ratio (OR) [95% confidence interval (CI)]: 1.033 [1.002–1.065], p=0.034) and cardiac injury (3.25 [1.40–7.54], p=0.006) were both independent predictors of in-hospital mortality.

Discussion

The salient findings of this cohort study are the following: 1) the cardiac injury group had higher rates of prior cardiac history as compared to the no cardiac injury group and also had higher inflammatory markers and D-dimer; 2) the cardiac injury group had a relatively low proportion of chest

pain and ECG abnormality such as T wave inversion and ST depression; and 3) the cardiac injury group had a mortality rate of 42.3% and cardiac injury was an independent predictor of in-hospital mortality.

As of 12 April 2020, New York City has been named the epicentre of the COVID-19 global pandemic and better understanding of the disease was highly desired [15]. Most of the initial clinical data of COVID-19 came from China, including those regarding cardiac injury, which may not be applicable to a population with different ethnic and social backgrounds. In our study, the cardiac injury group was more likely to have a history of cardiovascular disease, similar to a prior study by Guo et al. [4]. That study also revealed that the cardiac injury group had a higher rate of coronary artery disease, and that a combination of cardiovascular disease and cardiac injury was associated with greater mortality. Our study validates that cardiac injury is a predictor of in-hospital mortality even in a diverse multiracial population. Several studies have reported the association of elevated troponin and poor outcomes in patients with COVID-19 [16], but there have not been robust reports of ECG findings to our knowledge. Our study contained a detailed analysis of presenting symptoms and ECG findings, and did not show statistically significant difference in presence of chest pain or ECG changes; although T wave inversion and ST depression appeared numerically more frequent in the cardiac injury group. The small sample may have failed to show an increase in large vessel coronary artery disease and ischaemia, but this result suggests that cardiac injury with COVID-19 may be from other mechanisms. For instance, there are reports of myocarditis caused by direct myocardial invasion by SARS-CoV-2 and of takotsubo cardiomyopathy [6,9,17,18]; also, cytokine storm caused by systemic inflammation leading to coagulopathy, which may contribute to thrombus formation and ischaemia in microcoronary circulation [19].

There are several limitations in our study. This study was conducted in a single centre hospital with a relatively small sample size with approximately 80% of total consecutive hospitalised patients with a measurement of troponin I. Despite adjustment, we could not eliminate all unmeasured confounding factors. Moreover, higher rates of chronic kidney disease in cardiac injury group might affect elevation of troponin I [20]. Nonetheless, our study shows detailed information regarding symptoms and ECG abnormalities among patients with COVID-19.

Conclusion

Patients with COVID-19 with elevated troponin I had relatively low proportion of chest pain and ECG abnormality, which might suggest cardiac injury is just a reflection of systemic inflammation and coagulopathy due to COVID-19. Cardiac injury was an independent predictor of in-hospital mortality.

Disclosures

There are no conflicts of interest to disclose.

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