

Correlation Between Baseline Troponin Level and The Severity of Pulmonary Hypertension 3 Months After COVID-19 Infection in Subjects without Previous Significant Cardiovascular Pathology

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ABSTRACT

Background: A serious medical condition linked to pulmonary hypertension (PH), right ventricular dysfunction (RVD), and cardiovascular consequences is coronavirus (Covid-19) infection.

Objective: This study aimed to assess the relation between the rise of cardiac troponin at base line investigation of COVID patients and the development of pulmonary hypertension 3 months after recovery from the infection.

Subjects and methods: The study was conducted as a cross-sectional descriptive study and included 104 non-critically ill patients 3 months after recovery from Covid-19 pneumonia. They were divided into 2 groups; mild cases and moderate/severe non-critically ill cases based on the WHO criteria at the time of admission. Troponin at the time of admission was measured. A full echocardiographic assessment of the right ventricle was done, and the pulmonary artery systolic pressure was calculated for every patient.

Results: In COVID-19, PH is a common complication encountered after recovery, especially in moderate/severe (non-critically ill) cases. We observed increased incidence of PH in the moderate/severe group 3 months' post-recovery, significantly correlated with the initial levels of the cardiac troponin at admission ($p < 0.001$).

Conclusions: There was a statistically significant positive correlation between troponin I levels at baseline admission and the development of pulmonary hypertension ($r^2 = 0.696$, $p < 0.001$) 3 months after recovery from the infection.

Keywords: Baseline troponin level, Pulmonary hypertension, Right ventricular dysfunction, COVID-19

INTRODUCTION

On February 14, 2020, Egypt confirmed its first case of the virus. COVID-19 can spread by microscopic particles and air droplets. Even if a person doesn't exhibit any symptoms, they could still transfer the infection for up to 20 days⁽¹⁾. Worldwide healthcare systems have faced many obstacles as a result of the COVID-19 epidemic⁽²⁾. Understanding the various clinical symptoms and long-term implications of COVID-19 has been one of the major concerns⁽³⁾. New research indicates that COVID-19 may have a major effect on the cardiovascular system, resulting in irregular coagulation, hypoxia, acute respiratory distress syndrome, direct myocardial damage, and a strong systemic inflammatory reaction⁽⁴⁾.

Bilateral lung involvement is observed, characterized by lung edema, thickening of the alveolar septa, vascular congestion, and significant interstitial and alveolar inflammatory infiltrates. In certain cases, these factors may contribute to the development of pulmonary fibrosis. Damage to the lung parenchyma and changes in pulmonary circulation can result in pulmonary hypertension (PH), which can thereafter cause right ventricular (RV) involvement and right heart failure⁽⁵⁾.

This kind of pulmonary hypertension has been suggested to be a combination of group 3 (caused by obstructive lung disease or fibrosis) and group 4 (caused by pulmonary artery blockages)⁽⁶⁾. According to actual investigations, most COVID-19 patients had comorbidities, primarily related cardiovascular disorders.

Another factor is that most published articles focus primarily on data from critically ill cases, giving patients with mild or moderate to severe (not on mechanical ventilation) infections, which account for 80% of infections less attention⁽⁷⁾.

Troponin is a protein found in cardiac muscle cells, and elevated levels of troponin in the blood can indicate damage to the heart. In the context of COVID-19, the presence of elevated troponin levels has been linked to a more severe disease course and poorer outcomes⁽⁸⁾. SARS-CoV-2 can cause direct harm to the heart in addition to pneumonia, which is the infection's primary complication. It can cause myocarditis, an infection of the heart that severely impairs cardiac contractility, or it can affect the pericardium (pericarditis), causing an effusion that can potentially worsen cardiac function⁽⁹⁾.

Understanding the relationship between troponin rise and pulmonary hypertension is crucial to determine whether COVID-19 individuals are more likely to experience cardiovascular problems. With regards to the sources provided, there is no specific research paper mentioned that directly addresses the relationship between troponin rise and pulmonary hypertension in individuals with COVID-19.

This study aimed to search for correlations between troponin concentration upon admission and the development of pulmonary hypertension 3 months following the resolution of COVID-19 lung infection in individuals who had no prior major pulmonary or

cardiovascular conditions that might have contributed to the development of PH.

METHODOLOGY

We enrolled 104 persons with COVID-19 pneumonia who were at least 18 years old (Confirmed by PCR) 3 months after recovery referred from the Chest and Cardiology Clinic to The Non-invasive Cardiology Unit in Suez Canal University Hospital (SCUH) to undergo transthoracic echocardiogram (TTE). A patient's clinical presentation of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection can vary, ranging from no symptoms to life-threatening disease. Adults infected with SARS-CoV-2 can generally be classified into the following severity of illness categories ⁽¹⁰⁾:

- **Mild illness:** People who exhibit any of the COVID-19 signs and symptoms (fever, cough, sore throat, malaise, headache, muscle pain, nausea, vomiting, diarrhea, loss of taste and smell), but do not require hospitalization because they do not experience dyspnea, shortness of breath, or abnormal chest imaging.
- **Moderate illness:** Patients requiring hospitalization who exhibit signs of lower respiratory disease during clinical evaluation or imaging and whose pulse oximetry (SpO₂) reading is less than 94% on room air at sea level.
- **Severe illness:** Those with a respiratory rate greater than 30 breaths per minute, lung infiltrates greater than 50%, arterial partial pressure of oxygen to fraction of inspired oxygen (PaO₂/FiO₂) ratio less than 300 mm Hg, or SpO₂ less than 94% on room air at sea level and they need ward hospitalization.
- **Patients who are critically ill:** those who have severe respiratory failure who need mechanical ventilation and ICU admission.

Based on the clinical presentation history, we separated the study population into two groups: With the exception of the seriously ill group, mild and moderate/severe.

Inclusion criteria: Patients who had been diagnosed with COVID-19 within the last three months, as confirmed by a positive result of a real-time reverse transcriptase–polymerase chain reaction (RT-PCR) assay of nasal and pharyngeal swabs; they also had to have mild, moderate, or severe pneumonia, not have required mechanical ventilation due to respiratory insufficiency, and not have a history of cardiovascular diseases.

Exclusion Criteria: Patients with critical COVID-19 pulmonary infection, patients with idiopathic PH or other forms classified under group 1, patients with significant cardiovascular pathology resulting in group 2 PH, patients with respiratory diseases causing group 3 PH, and patients with a history of pulmonary thromboembolism that may result in group 4 PH. Patients receiving treatment with vasodilators used in PH therapy (such as phosphodiesterase type 5 inhibitors, guanylate cyclase

stimulators, prostacyclin analogues or endothelin receptor antagonists), metabolic, systemic and hematological, or other pathologies that could result in a PH of group 5.

A structured survey was employed to gather the fundamental clinical information about the patients, and examination was done to all patients enrolled in the study including general and local cardiac examination. Resting 12 lead ECG and echocardiography were performed on all patients. Troponin at baseline (Cut value less than 0.04 ng/mL) was reviewed, which indicates myocardial injury ⁽¹¹⁾. Echocardiography was performed (General Electric Healthcare Company, Vivid iq) with a 2.5-MHz phased array probe. The likelihood of pulmonary hypertension, valvular disease, and chamber size and function were evaluated. Measurements were taken offline, retroactively, with the help of the preserved photos by 2 independent observers blinded to the clinical data.

In order to evaluate the RV, the ED's apical four-chamber image was used to determine the basal diameter. RV wall thickness was assessed in the emergency department's subcostal view. On the lateral tricuspid annulus, the tricuspid annular plane systolic excursion (TAPSE) was assessed using M-mode. After tracing the RV endocardial boundaries in ES and ED, the RV fractional area change (FAC) was computed. Color flow was used to document the TR grade. The diameter of the inferior vena cava (IVC) was measured when it was seen in the subcostal view ^(12, 13).

Doppler Echocardiography: The tricuspid valve (TV) was then measured using PW Doppler from the apical 4-chamber view, and the pulmonary valve was measured to determine the peak pulmonary valve velocity. During silent breathing in end-expiration, these characteristics were measured. Lastly, the estimated right atrial pressure was added based on the size and collapsibility of the IVC, and a continuous-wave (CW) Doppler was used on the TV to determine the RV systolic pressure (RVSP) ^(14, 15).

According to the latest international recommendations, a tricuspid annular plane systolic excursion (TAPSE) of less than 17 mm, a tissue Doppler imaging S wave (S' wave) of less than 9.5 cm/s, or a basal diameter measurement of more than 41 mm were used to define RV systolic dysfunction ⁽¹⁶⁾. Systolic pulmonary artery pressure (SPAP) was estimated with the following formula: $SPAP = 4 \times \text{tricuspid regurgitation (TR) peak velocity}^2 + \text{right atrial pressure (RAP)}$ ⁽¹⁷⁾; PH was defined as $SPAP > 35$ mm Hg with the severity ranging from mild (35–44 mm Hg), to moderate (45–60 mm Hg) and severe (> 60 mm Hg) ^(18, 19). As per the latest guidelines, the central venous pressure (CVP) estimation and TR grading were carried out ⁽¹⁷⁾.

Ethical consideration: The Suez Canal University Ethical Committee amended and approved the study protocol. Written informed consent was provided by each study participant. The World Medical Association's (Declaration

of Helsinki) Code of Ethics for human subjects research has been adhered to in the conduct of this work.

Statistical analysis: The data was collected using Microsoft EX-CEL and analysis was done using Statistical Package for Social Sciences (SPSS) version 27.0. Statistical significance tests will be used and a probability value (P value) of less than or equal (0.05) will be considered statistically significant (At 95% level of confidence). Descriptive statistics will be presented as (Means ± Standard Deviation) for quantitative variables and as (Percentage) for qualitative variables. Quantitative variables were compared using unpaired t-test between the two groups. Qualitative variables were compared using the Chi-Square test/Fisher’s exact test. Pearson correlation coefficient was used to find out the association of E/e’ with various parameters. Personal, clinical, and imaging data will be collected and relationships between different factors will be done then the results of management will be represented in tables and graphs.

RESULTS

This study is part of a subgroup analysis of another study about the relation of global longitudinal strain and RV affection 3 months after recovery of COVID-19 infection of non-critically ill patients. 104 of the patients that were monitored for SARS-CoV-2 pneumonia met the requirements for inclusion, making them a part of this analysis. They were divided into 2 groups based on the WHO criteria mentioned earlier, the mild group which included 50 patients, and the moderate/severe one which included 54 patients. The presentation included baseline demographics, risk factors, and clinical, laboratory, and echocardiographic features (Table 1). The mean age was 48.8 ± 10.7 years, male predominance (55.8 %), mean BMI was 23.7 ± 0.71 kg/m², 37.5 % were diabetics and 44.2 % were hypertensive. The two populations did not differ in terms of age, gender, or cardiovascular risk factors.

Table (1): Description of demographic data in all studied patients

	TOTAL (N=104)		Mild (N = 50)		Moderate/ severe (N = 54)		P-value
	N	%	N	%	N	%	
Sex (males)	58	59.8%	27	54%	31	57.4%	0.727 NS
Age (years)	48.8 ± 10.7		48.5 ± 11.0		49.2 ± 10.6		0.739 NS
BMI (Kg/m²)	23.7 ± 0.71		23.6 ± 0.82		23.8 ± 0.59		0.1 NS
HTN	46	44.2%	22	44%	24	44.4%	0.964 NS
DM	39	37.5%	16	32%	23	42.6%	0.265 NS
Smoking	48	46.2%	24	48%	24	44.4%	0.716 NS

t: independent sample t-test. X²: Chi-square test. NS: p-value > 0.05 is considered non-significant

Table (2) showed that there was a statistically noteworthy distinction between the 2 studied groups regarding the elevation of cardiac troponin at baseline admission indicating myocardial injury.

Table (2): Comparison of baseline troponin between mild group and moderate/severe group

	MILD (N = 50)	Moderate to severe (N = 54)	p-value
Troponin (ng/ml)	5.1 ± 1.9	53.3 ± 9.4	0.032*

p- value < 0.05 is considered significant.

Table (3) showed that all RV echocardiographic parameters did not show statistically noteworthy distinction between the two groups 3 months after recovery from COVID infection apart from PASP and TR velocity confirming that pulmonary hypertension is a frequent sequel of COVID infection.

Table (3): Description of studied ECHO data in all studied patients

	TOTAL (N=104)	Mild (N = 50)	Moderate/severe (N = 54)	T	P-value
TAPSE	2.23 ± 0.25	2.28 ± 0.13	2.22 ± 0.31	1.3	0.193 NS
RV FAC	39.1 ± 6.02	42.2 ± 3.1	39.7 ± 4.1	3.4	0.081 NS
RV basal	3.45 ± 0.35	3.19 ± 0.13	3.30 ± 0.31	2.3	0.21 NS
RV MID	2.60 ± 0.36	2.43 ± 0.33	2.6 ± 0.38	2.44	0.16 NS
TR velocity	3.2 ± 0.30	2.97 ± 0.15	3.08 ± 0.27	2.4	0.014 S
PASP	39.8 ± 6.58	35.5 ± 3.64	37.6 ± 6.3	2.1	0.036 S

P-value < 0.05 is considered significant.

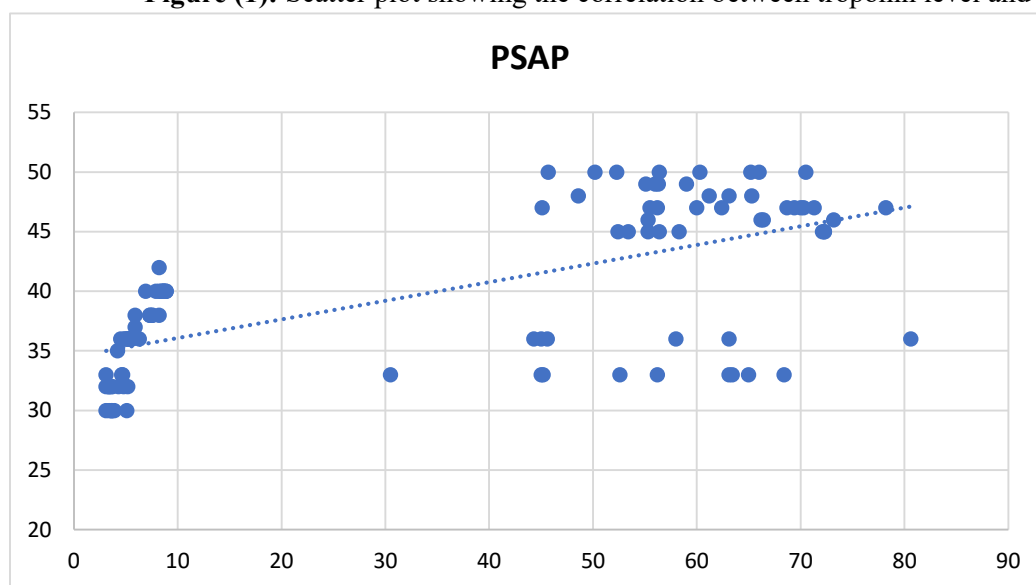
Table (4) and figure (1) showed a statistically significant positive correlation between troponin levels at baseline admission and the development of pulmonary hypertension ($r = 0.696, p = < 0.001$) 3 months after recovery from the infection.

Table (4): Spearman’s correlation coefficient for the relation between troponin level and PSAP levels among study participants (n=104)

	PSAP	
	R	p-value
Troponin	0.696	< 0.001*

*Statistically significant at $p < 0.05$.

Figure (1): Scatter plot showing the correlation between troponin level and PSAP



DISCUSSION

Patients with COVID-19 frequently have elevated troponin levels, which are strongly associated with catastrophic outcomes. A statistically significant difference between the studied mild and moderate/severe group regarding elevation of cardiac troponin at baseline admission indicating myocardial injury. This phenomena of myocardial injury could be explained by a number of processes, including unmasked CAD, cytokine-driven myocardial damage, viral myocarditis, and microangiopathy. As of right now, none of these processes have been shown to be the primary cause of troponin increase and/or cardiac injury in COVID-19 individuals⁽²⁰⁾. A study of 416 hospitalized COVID-19 patients from China found that 19.7% of patients had myocardial injury, and the mortality rate among these patients was 51.2%⁽²¹⁾.

Elevated cardiac troponin (cTn) levels were also linked to a poor prognosis in COVID-19. Specifically, patients with significantly elevated cardiac troponin I (cTnI) levels had higher mortality rates, and non-survivors consistently had elevated cTnI levels, which further increased as their condition worsened⁽²²⁾.

Another study reported that 27.8% of patients’ cardiac damage was evident by elevated Troponin T

levels (TnT), with an in-hospital mortality rate of 59.6%⁽²³⁾.

The death rate for patients with increased TnT levels and underlying cardiovascular disease was 69.4%. Conversely, the death rate for those with increased TnT levels but no history of cardiovascular disease was 37.5%. These results highlight the link between myocardial damage and worse outcomes in COVID-19 patients⁽²³⁾.

The research by **Xu et al.** revealed a low cardiac index, high pulmonary artery pressure, and high pulmonary vascular resistance were associated with higher troponin levels⁽²⁴⁾. According to another study, there may be a connection between this rise in troponin levels and heightened intramural right ventricular strain, elevated pulmonary vascular resistance, and eventually decreased cardiac output. These elements may be responsible for the ischemia and necrosis of the right ventricle⁽²⁴⁾.

Our research indicated a positive statistically significant association between the levels of troponin I at baseline admission and the development of pulmonary hypertension ($r2 = 0.696, p = < 0.001$) 3 months after recovery from the infection. Extensive pulmonary damage (caused by interstitial and alveolar

inflammation), as in group 3's PH, and changes to the pulmonary vasculature (caused by thrombotic/thromboembolic processes, endothelial injury, or at least, hypoxic vasoconstriction), as in group 4's PH, are thought to be the primary causes of PH associated with COVID-19^(5, 6).

According to our research, the frequency of pulmonary hypertension correlated with the severity of individual instances. This is consistent with **Tudoran et al.**⁽²⁵⁾ findings that the moderate group's pulmonary artery pressure was significantly higher than that of the mild group. In line with these findings, **D'Andrea et al.**⁽¹⁵⁾ carried out a prospective, single-center study involving 79 individuals to thoroughly examine the right ventricle (RV) in COVID-19 recovery patients using echocardiography. The patients were divided into four groups based on the degree of pneumonia or lack thereof: severe pneumonia, mild to moderate pneumonia, no pneumonia, and the control group. Pulmonary artery pressure (PAP) was higher in the groups with mild-to-moderate and severe pneumonia than in the control and non-pneumonia groups.

Study Limitation: The primary limitation of our research was the small sample size, but it was challenging to find additional COVID-19-recovered patients without concomitant pathology who were willing to participate in our study. The second limitation was the absence of precise baseline values for the TTE parameters due to the absence of a precise measurement during hospitalization. Therefore, we are unable to determine the exact esPAP values prior to the infection. The third limitation was that, because of pandemic constraints, we were unable to do right cardiac catheterization regularly, therefore we were forced to rely solely on the TTE assessment of esPAP in this investigation instead of validating our results with invasive procedures. The last limitation was that we did not keep an eye on our patients for a longer period of time, but this will be done later.

CONCLUSION

Even in circumstances where patients are not severely ill, pulmonary hypertension is a common COVID-19 consequence. Although its historical evolution is still unclear, it appears to have lasted longer than previously thought. A statistically significant positive connection was observed between the levels of troponin at baseline admission and the development of pulmonary hypertension ($r_2 = 0.696, p = < 0.001$) 3 months after recovery from the infection.

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