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Research Paper

Troponin as a Prognostic Biomarker for Mortality in COVID-19 Patients: A Retrospective Study

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ABSTRACT

Background: Troponin has been widely recognized as a key prognostic biomarker for predicting mortality in patients with severe COVID-19. This study aimed to assess the relationship between troponin concentration and mortality in patients infected with the Delta variant of COVID-19.

Methods: This retrospective, single-center study included 40 hospitalized COVID-19 patients aged 18 to 65 years, classified as having moderate or severe disease. Patients with pre-existing cardiovascular or muscular disorders, prior use of anticoagulants, or non-standard treatment protocols were excluded to reduce confounding. All participants had documented serum troponin levels measured at admission (troponin 0) and repeated six hours later (troponin 6). Data on demographics, comorbidities, laboratory findings, and clinical outcomes were extracted from medical records. Statistical analyses were performed to assess correlations between troponin levels and disease severity.

Results: Serum troponin levels showed a significant positive correlation with age ($r = 0.482$, $P = 0.002$) and were notably higher among retired individuals ($H = 6.45$, $P = 0.040$). No significant association was found between troponin levels and clinical symptoms ($P > 0.05$). A significant negative correlation was observed between diastolic blood pressure (DBP) and troponin ($r = –0.421$, $P = 0.008$). Patients who died had significantly elevated troponin levels ($U = 0.62$, $P = 0.001$). A troponin cutoff value of 15.5 ng/L predicted mortality with 70.6% sensitivity and 90.9% specificity.

Conclusion: The findings of this report indicate that elevated troponin concentrations were more frequently observed in older individuals and in patients with lower DBP. Moreover, higher troponin levels were significantly associated with increased mortality, likely due to direct cardiac damage from SARS-CoV-2 or the body's inflammatory response to the infection.

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Introduction

COVID-19 is a disease that causes severe acute respiratory syndrome and is caused by the novel coronavirus SARS-CoV-2. According to the WHO report, by mid-2021, COVID-19 had infected nearly 105 million people worldwide and caused 2.3 million deaths [1, 2].

Cardiac complications in COVID-19 patients are a common concern [3]. Viral-induced cardiac complications are severe, and mortality rates in these patients are high [4]. Biomarkers play an important role in treatment plans and clinical management. One of the most important biomarkers in COVID-19 patients is troponin, which is often associated with cardiac injury [5]. Cardiac troponin is a protein complex consisting of troponin T, troponin C, and troponin I. Troponin and calcium ions regulate muscle contraction in striated muscle tissue and the heart [6].

Evidence demonstrates that about 31% of COVID-19 patients have elevated troponin concentrations. Furthermore, high troponin levels are associated with a fivefold increased risk of death compared to patients with normal troponin concentrations. Notably, this is independent of age, gender, blood pressure, diabetes, or history of heart disease. Therefore, elevated troponin may be a strong indicator of disease prognosis, suggesting that COVID-19 patients with high troponin levels have a much lower chance of survival [7].

Given troponin's prognostic value in severe COVID-19 and the virus's mutational landscape, it is necessary to evaluate its prognostic utility across different stages of the outbreak. This is essential for effective treatment planning. This study investigated the relationship between troponin levels and mortality among patients hospitalized during the fifth wave of the pandemic, related to the Delta variant outbreak.

Materials and Methods

This retrospective, descriptive-analytical study examined the association between troponin levels and mortality in COVID-19 patients hospitalized at Shahid Beheshti Hospital in Kashan, Iran, during the fifth pandemic wave. COVID-19 diagnosis was confirmed via PCR testing. Disease severity was evaluated by a certified specialist based on clinical symptoms, SpO_2 levels, inflammatory markers, pulmonary involvement, and oxygen needs. Severe cases were defined as $SpO_2 < 90\%$, while moderate cases had SpO_2 between 90–94%. All patients received treatment according to national COVID-19 protocols.

Patient data were extracted from medical records stored in the hospital information system of Shahid Beheshti Hospital, using simple random sampling. Data collection was performed using a structured checklist that included demographic information, disease severity, clinical symptoms, troponin level (ng/L): Troponin levels were measured and reported based on a standard laboratory kit with a defined and fixed cutoff, disease outcome, vital signs (SpO_2, temperature, respiratory rate, heart rate, blood pressure), and underlying diseases.

Clinical Criteria for patients: Moderate Covid-19 criteria; Fever less than 37.8 axillary - Shortness of breath during less than usual activities - No chest pain. Severe Covid-19 criteria: Fever greater than 37.8 axillary - Shortness of breath even at rest - Not drinking or eating - Inability to speak due to shortness of breath - Chest pain, especially when talking or changing position

Inclusion criteria

Aged 18–65 years diagnosed with the Delta variant of COVID-19; documented troponin test results; moderate or severe disease severity; and treatment according to national COVID-19 protocols.

Exclusion criteria

missing troponin data; use of non-standard medications; pre-existing cardiovascular or muscular disorders; prior anticoagulant therapy; and incomplete medical records.

Results

This study evaluated 39 participants infected with the Delta variant of COVID-19. Patients were divided into two groups based on oxygen saturation (SpO_2): those with moderate disease ($SpO_2 > 90\%$) and those with severe disease ($SpO_2 < 90\%$). The average age of patients was 59.30 ± 19.72 years. Demographic and clinical details are provided in Table 1.

Table 2 illustrates the distribution of clinical symptoms and average vital signs among COVID-19 patients categorized by disease severity. The analysis showed a uniform distribution of symptoms, with no statistically significant association between symptom type and severity ($P<0.05$). However, oxygen saturation differed significantly between groups, with severe cases showing a lower mean value ($P=0.001$), suggesting its potential utility as an indicator of disease severity.

Table 1. Baseline characteristics of patients.

Variable		Moderate COVID-19 (N=19)	Severe COVID-19 (N=20)	P-value
Age		55.73±22.82	62.70±16.12	0.281*
Gender	Male	9 (45%)	11 (55%)	0.634**
	Female	10 (52.6%)	9 (47.4%)	
Occupation	Housewife	9 (72.7%)	3 (27.3%)	0.147**
	Retired	4 (33.3%)	8 (66.7%)	
	Worker	7 (43.8%)	9 (56.3%)	
Underlying disease	Yes	15 (46.9%)	17 (53.1%)	0.652**
	No	4 (57.1%)	3 (42.9%)	

*Independent T test, **Chi-square test

Table 2. Distribution of clinical symptoms and mean vital signs of patients.

Variable	Moderate COVID-19 (N=19)	Severe COVID-19 (N=20)	P-value
Fatigue and weakness	9(56.3%)	7(43.8%)	0.523*
Shortness of breath	13(50.0%)	13(50.0%)	1*
Cough	6(35.3%)	11(64.7%)	0.200*
Gastrointestinal symptoms	3(25.0%)	9(75.0%)	0.082*
Musculoskeletal symptoms	1(33.3%)	2(66.7%)	1*
Decreased level of consciousness	0 (0%)	2(100%)	0.157**
Respiratory rate (breaths/min)	18.21 ± 1.18	20.15 ± 6.68	0.221***
Heart rate (beats/min)	91.15 ± 13.45	93.65 ± 38.97	0.793***
Temperature (°C)	38.27 ± 0.71	37.28 ± 0.73	0.996***
Systolic blood pressure (mmHg)	123.31 ± 30.52	123.85 ± 22.85	0.951***
Diastolic blood pressure (mmHg)	71.42 ± 10.95	75.14 ±14.35	0.369***
Oxygen saturation percentage SpO_2 (%)	94.84 ±3.16	82.95 ± 6.54	0.001***

*Fisher's exact test, **Chi-square test, *** Independent T test

Table 3. Distribution of troponin levels and outcome in COVID-19 patients.

Variable		Moderate COVID-19 (N=19)	Severe COVID-19 (N=20)	P-value
Troponin (ng/ L)		5(479.75 – 3.25)	4(32.75– 4)	0.607*
Final outcome	**Death**	7(41.2%)	10(58.8%)	0.523**
	Recovery	12(54.5%)	10(45.5%)	
Variable		**Median troponin level**	**U**	**P-value**
Final outcome	**Recovery**	4.0(5.0-3.0)	62.0	0.001*
	Death	339.0(1734.6 – 4.5)		

*Mann-Whitney Test (Findings are reported as median (first quartile - third quartile)), **Fisher's exact test

Table 4. Distribution of baseline characteristics of COVID-19 patients by median troponin level.

Variable		Median troponin level	H	P-value
Occupation	**Housewife**	0.4 (0.8 - 0.2)	6.45	0.040*
	Retired	5.15 (7.1378 – 2.4)		
	Freelancer	0.4 (.032 – 25.3)		
Variable		**Correlation coefficient**		**P-value**
Age		482/0		0.002**

*The Kruskal-Wallis one-way ANOVA, ** Spearman's correlation test (Findings are reported as median (first quartile - third quartile))

As shown in Table 3, troponin levels were not significantly associated with disease severity (P=0.607), and no significant difference in outcomes was observed between severity groups (P=0.523). However, a significant relationship was found between median troponin levels and patient outcomes, with deceased individuals exhibiting higher troponin levels (P=0.001), indicating its potential as a prognostic marker for mortality.

Table 4 demonstrates a significant positive correlation between age and troponin levels (r=0.482, P=0.002), indicating that older patients tend to have higher troponin concentrations. Additionally, occupation was found to be associated with troponin levels, with retired individuals exhibiting significantly elevated troponin levels (H=6.45, P=0.040), suggesting that age-related factors may contribute to increased cardiac vulnerability.

Table 5 reveals a statistically significant negative correlation between DBP and troponin levels (r=–.421, P = 0.008). This suggests that lower diastolic pressure is associated with higher troponin concentrations, indicating a potential link between reduced coronary perfusion and myocardial injury.

Table 5. Correlation of vital signs of COVID-19 patients with median troponin levels.

Variable	Correlation coefficient	P-value
Respiratory rate	0.065	0.696
Heart rate	0.066	0.688
Temperature	0.001	0.997
Systolic blood pressure	0.084	0.612
Diastolic blood pressure	0.421	0.008
Oxygen saturation percentage	0.023	0.889

*Spearman's correlation test (Findings are reported as median (first quartile - third quartile))

Table 6 and Figure 1 indicate that a troponin cutoff value of 15.5 ng/L provides a sensitivity of 70.6% and a specificity of 90.9% for predicting mortality in COVID-19 patients. This threshold effectively distinguishes high-risk individuals from those with a greater likelihood of recovery, reinforcing troponin's role as a reliable prognostic marker.

Table 6. Sensitivity and specificity of troponin level cutoff points for mortality in COVID-19 patients.

Area under the Curve	Cutoff point	Sensitivity (%)	Specificity (%)
0.833	15.5	70.6	90.9

Discussion

Cardiovascular diseases (CVD) are the main reason for mortality and disability [8]. Cardiac complications of COVID-19 are common and are related to worse outcomes [9]. The exact mechanisms underlying the cardiac complications of COVID-19 are unknown; however, some studies have suggested mechanisms including microvascular thrombosis, direct viral damage to heart cells, systemic inflammation, and a cytokine storm [10, 11]. High troponin levels, underlying cardiovascular disease, and related risk factors are significantly associated with higher mortality in COVID-19 patients [12-14].

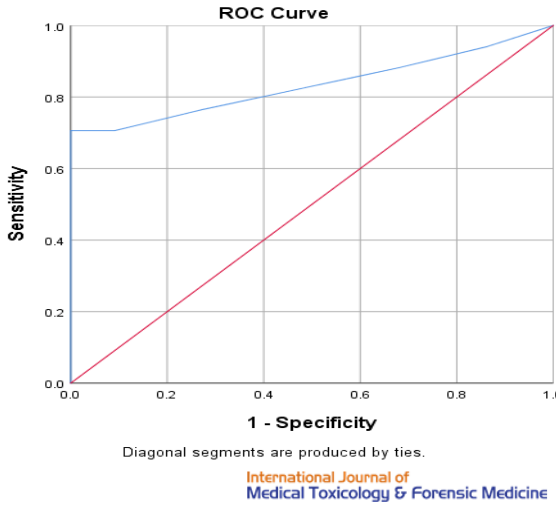

Figure 1. ROC curve of troponin level in terms of mortality.

According to recent research, elevated troponin levels are more common in older patients [15] who have pre-existing cardiovascular diseases. These patients also have more risk factors, including hypertension, diabetes, dyslipidemia, smoking, chronic kidney disease, and dialysis [16]. Shi S et al. found cardiac injury in 19.7% of hospitalized COVID-19 patients; also, they reported that troponin levels increase with age and serve as an independent predictor of mortality [17]. Bürgi JJ et al. reported that in men aged 54 and older with mild to moderate COVID-19, prolonged elevation of troponin I for 14 months indicates persistent cardiac injuries, even without severe symptoms [18]. These findings highlight the strong relationship between aging and cardiovascular vulnerability, emphasizing the importance of early cardiac monitoring in elderly COVID-19 patients.

This study demonstrates an inverse relationship between troponin levels and DBP, indicating that lower DBP is associated with higher troponin levels. This supports previous findings that systolic blood pressure and pulse pressure are positively associated with troponin, suggesting that unstable hemodynamic status may lead to myocardial injury, even in the absence of obvious cardiovascular disease [19]. Since blood flow in the coronary arteries occurs primarily during diastole, decreased DBP may lead to myocardial hypotension and myocardial injury, especially in patients with systemic inflammation, such as those with COVID-19 [19]. In severe cases, sepsis and septic shock can cause significant hypotension, which can

raise the possibility of low DBP as a marker of disease severity and a contributing factor to cardiac injury [20]. Therefore, high troponin in COVID-19 patients may reveal reduced myocardial perfusion, ischemia, and secondary cardiac injury due to severe illness.

This study emphasizes the strong association between elevated troponin levels and mortality, showing higher troponin concentrations in deceased patients. Giustino G et al. (2020) identified cardiac injury in COVID-19 as an independent predictor of in-hospital death, and highlighted the importance of troponin as a prognostic marker [12]. Similarly, Metkus TS et al. (2021) confirmed the association between myocardial damage and increased mortality, whether caused by direct viral effects or systemic inflammation [13]. Previous studies have reported mortality (20 to 61 percent) in patients with elevated troponin levels, which is higher than in those with normal levels. Despite variations due to patient demographics and disease severity, troponin remains a reliable marker of cardiac injury and poor outcomes in COVID-19 [12-14]. Since troponin is an important biomarker for diagnosing heart muscle damage or cardiotoxicity, troponin levels have been measured in studies of other patients with ECG abnormalities and cardiac complications [21].

Cardiac injury in COVID-19 arises from several mechanisms. One involves the SARS-CoV-2 spike protein binding to ACE2 receptors, which are highly expressed in cardiac tissue, allowing the virus to enter heart cells directly and causing inflammation and dysfunction [22, 23]. Another key factor is systemic inflammation, where a cytokine storm and immune activation may damage the myocardium [24-27]. Also, COVID-19 has been linked to myocardial infarction and acute coronary syndrome. In addition, stressors associated with COVID-19 infection [28-31]. Furthermore, COVID-19 induces a prothrombotic state characterized by endothelial dysfunction and increased clotting. These events lead to thromboembolic states such as deep vein thrombosis, pulmonary embolism, and arterial thrombosis, which are linked to increased risks of MI and stroke [32-37].

This study has several important limitations that should be acknowledged. First, the retrospective design inherently limits the ability to establish causal relationships. It may be subject to selection bias, as data were collected from existing records rather than through prospective enrollment. Second, the relatively small sample size limits the analysis's statistical power and may reduce the reliability of subgroup comparisons and the detection of subtle associations. Third, the study was conducted at a single center, which may limit the generalizability of the findings to broader populations with different demographic or clinical characteristics. Lastly, potential measurement biases may have influenced the results. These factors could introduce inconsistencies that affect the interpretation of clinical correlations. Future multicenter, prospective studies with larger and more diverse cohorts are recommended to validate and expand upon these findings. It is also suggested that future studies evaluate additional cardiovascular biomarkers.

Conclusion

Overall, this study demonstrates that elevated troponin levels are more common in older patients and those with lower DBP, and are significantly linked to increased mortality. The findings demonstrate the prognostic utility of troponin in COVID-19 as an early marker for risk stratification and to guide preventive interventions, thereby reducing cardiovascular complications and mortality at the optimal time.

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Ethical Approval

This study was approved by the Ethics Committee of KAUMS, Kashan, Iran (IR.KAUMS.MEDNT.REC.1402.018).

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Conflicts of Interest

The authors report there are no competing interests to declare.

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