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Prognostic value of elevated cardiac and inflammatory biomarkers in patients with severe COVID-19: a single-center, retrospective study

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Abstract

Background: The outbreak of coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 in India has been declared a public health emergency. Many patients with COVID-19 experience cardiac injury. Patients with COVID-19 admitted to the intensive care unit (ICU) with acute myocardial injury showed increased high-sensitivity troponin levels. Abnormal troponin levels may indicate myocardial injury and are commonly associated with COVID-19.

Methods: We conducted a retrospective observational study of 44 patients with severe COVID-19 in ICU during the second wave. The primary end point of our retrospective study was 28-day mortality, and the time of ICU admission was designated as day 0. We extracted and analyzed cardiac biomarkers, such as creatine kinase (CK), creatine kinase-MB (CK-MB), B-type natriuretic peptide (BNP), and high-sensitivity cardiac troponin I (hs-cTnI), and various inflammatory markers such as C-reactive protein (CRP) level, interleukin 6 (IL-6), D-dimer, ferritin, lactate dehydrogenase, IL-6, and procalcitonin in patients with severe COVID-19 at ICU admission and 72 hours after ICU admission from our electronic medical record system.

Results: The best cutoff of BNP were 326.8 and 398.5 pg/mL, CK were 195.95 and 180.12 U/L, CK-MB were 112.10 and 108.5 U/L, and hs-cTnI were 0.035 and 0.025 ng/mL, at ICU admission and 72 hours after ICU admission for predicting 28-day mortality among nonsurvivors.

Conclusion: In patients with severe COVID-19, CK and hs-cTnI may be considered effective and valuable predictive cardiac biomarkers among nonsurvivors and predict poor prognosis.

Keywords: Cardiac biomarkers, COVID-19, Intensive care unit, Mortality, Prognosis, Retrospective study

Introduction

The coronavirus disease 2019 (COVID-19) pandemic, caused by severe acute respiratory syndrome coronavirus 2, has become a global threat.[1]

Specific drug treatment for COVID-19 is still not wholly established; therefore, early identification of severe patients needs attention, and necessary interventions should be considered. Patients with severe COVID-19 may have more complications, such as acute respiratory distress syndrome, acute cardiac injury, acute kidney injury, and shock, and have poorer adverse clinical outcomes.[2,3]

Many patients with COVID-19 have cardiac injury. Patients with COVID-19 admitted to intensive care unit (ICU) with acute myocardial injury mainly showed increased high-sensitivity troponin levels.[2] The abnormal level troponin values may indicate a myocardial injury, and they are commonly associated with COVID-19 infection. Elevated levels of high-sensitivity cardiac troponin (hs-cTn) were observed in many patients, and high-sensitivity cardiac troponin I (hs-cTnI) levels were significantly elevated in more than half of the patients who died of COVID-19.

A few important prognostic markers may play an important role in the early initiation of therapeutic strategies, especially in patients with severe COVID-19.

Guo et al.[4] concluded that COVID-19 patients with elevated troponin T levels had higher mortality. The overall prevalence of acute myocardial injury ranges from 5% to 38% in COVID-19 patients.[5] There are limited studies that analyze the association between the prognostic value of elevated cardiac biomarkers for patients with severe COVID-19 and 28-day mortality. Therefore, this retrospective study aimed to observe any change in cardiac biomarkers, including creatine kinase (CK), creatine kinase-MB (CK-MB), B-type natriuretic peptide (BNP), and hs-cTnI in patients with severe COVID-19 and determine the association between elevated cardiac biomarkers and poor outcomes. We have also analyzed the various inflammatory markers, including C-reactive protein (CRP), interleukin 6 (IL-6), lactate dehydrogenase (LDH), D-dimer, ferritin, and procalcitonin whether these markers act as an effective and valuable prognostic predictor for patients with COVID-19.

The main aims of this study were to determine the role of cardiac-specific biomarkers with other inflammatory markers and their association with COVID-19 mortality and to evaluate their use in predicting prognosis.

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

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Methods

Study design

This retrospective observational study reviewed data obtained from an electronic register system of patients with severe COVID-19 admitted to the ICU of the All India Institute of Medicine Sciences Patna, Bihar, India, during the second wave, that is, from March 31 to April 15, 2021. This study was approved, and written informed consent was waived by the departmental research committee of the All India Institute of Medical Sciences Patna, Bihar, India (141/T&E/AIIMS/Pat/2022, dated March 10, 2022), because of the retrospective nature of the study. The study complied with international ethical guidelines for human research, such as the Declaration of Helsinki. The accessed data were anonymized.

Study population

This observational study included 44 patients with severe COVID-19 according to the Ministry of Family Health and Welfare, New Delhi, guidelines. All patients with the following characteristics were included: (1) age greater than or equal to 18 years; (2) real-time reverse transcriptase–polymerase chain reaction assay positive; (3) respiratory rate greater than 30 breaths per minute; (4) oxygen saturation less than 92% on room air or PaO_2/FiO_2 less than or equal to 300 mm Hg (P/F) ratio (arterial partial pressure of oxygen/inspired oxygen concentration); and (5) patients with greater than 50% lesion progression within 24 to 48 hours on pulmonary imaging. Patients younger than 18 years, with incomplete electronic medical records, history of acute myocardial infarction, cerebral stroke, pregnancy, or any form of malignancy were excluded from this study.

Data collection

The electronic register system was a part of our hospital information system, in which ICU physicians and residents have recorded the information of patients. For all included cases, the following data were retrieved: demographic characteristics, preexisting comorbidities, various modes of ventilation, P/F ratio, cardiac biomarkers like CK, CK-MB, BNP, and hs-cTnI in patients with severe COVID-19 at the time of ICU admission and 72 hours after ICU admission.

Outcome analysis

The primary end point of our retrospective study was 28-day mortality; the time of ICU admission was designed as day 0. The upper limit of the normal range of serum cardiac biomarkers and other inflammatory biochemical markers was used to define myocardial injuries based on our local hospital criteria. There may be variations of different techniques, reagents, and instruments in performing these analyses at different hospitals. Myocardial injury was defined as an increase in CK, CK-MB, BNP, and hs-cTnI levels above the upper limit of the normal.

Statistical methods

Data were analyzed using Statistical Package for Social Sciences (SPSS) Statistics (version 26.0, IBM, Armonk, NY). Normally distributed continuous variables were presented as median interquartile range (IQR) compared using an independent *t* test. The receiver operating characteristic (ROC) analysis was conducted to assess the overall performance of increased levels of myocardial Injury parameters to identify the risk of mortality in patients with COVID-19. The area under the receiver operating characteristic (AUC) curve was computed to evaluate the performance of each marker. Two-sided α error ($P < 0.05$) was considered statistically significant.

Results

Fifty-one patients with severe COVID-19 were initially recruited for this retrospective study. Of these 3 patients who had incomplete electronic medical records, 2 who were younger than 18 years and 2 who were pregnant were excluded. Therefore, 44 patients were enrolled, of which 25 were survivors and 19 were nonsurvivors

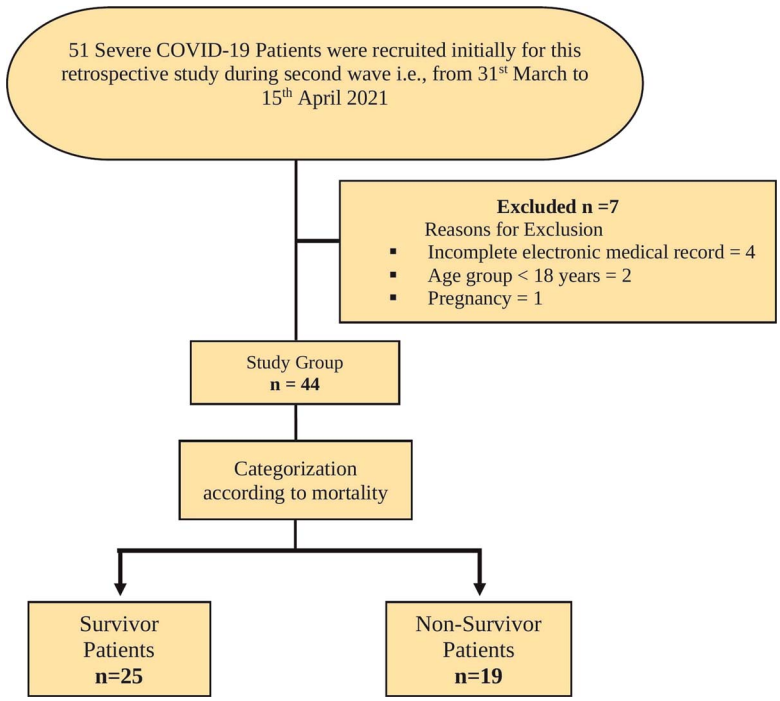

Figure 1. Flow diagram. COVID-19, coronavirus disease 2019; ICU, intensive care unit.

(Fig. 1). We measured the best cutoff values for CK, CK-MB, BNP, and hs-cTnI at ICU admission and 72 hours after ICU admission and various inflammatory biomarkers, such as CRP, IL-6, D-dimer, ferritin, LDH, and procalcitonin at ICU admission and 72 hours after ICU admission in the 2 study groups.

Association of cardiac biomarkers with 28-day mortality of patients with severe COVID-19

We evaluated the association between cardiac and inflammatory biomarkers and COVID-19 outcomes. All myocardial biomarkers CK, CK-MB, BNP, and hs-cTnI levels were significantly associated with 28-day all-cause death in patients with COVID-19.

Receiver operating characteristic curve

The ROC curve was used to demonstrate the ability of each cardiac biomarker and inflammatory biomarker at ICU admission and 72 hours after ICU admission in the discrimination of high risk of COVID-19 mortality; AUC is shown in Fig. 2. We analyzed and calculated the median IQR values for cardiac and inflammatory biomarkers at ICU admission and 72 hours after ICU admission between

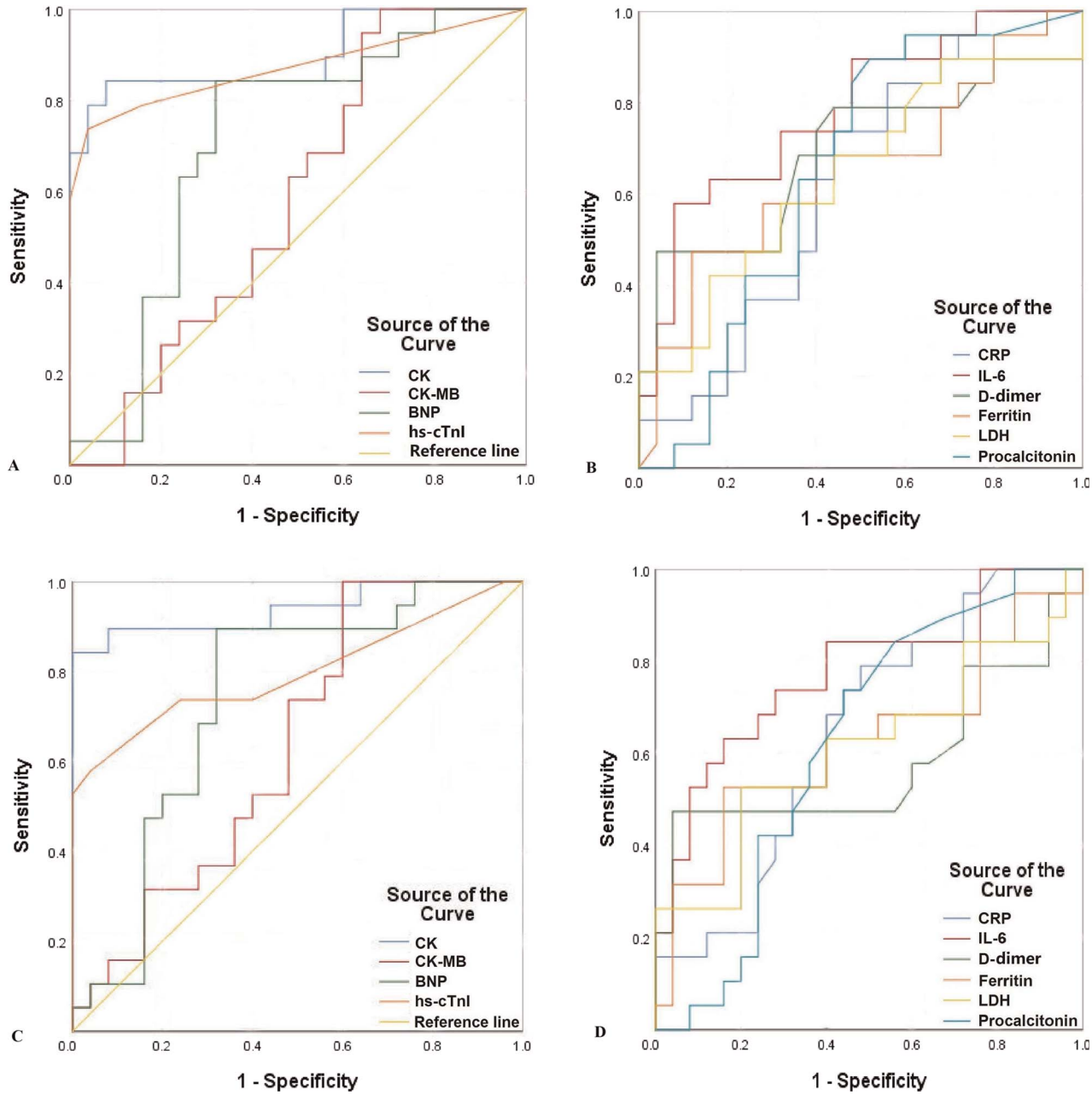

Figure 2. (A) and (B), ROC curve analyses for the prognostic role of the cardiac and inflammatory biomarkers in patients with severe COVID-19 at ICU admission. (C) and (D), ROC curve analyses for the prognostic role of the cardiac and inflammatory biomarkers in patients with severe COVID-19 at 72 hours after ICU admission. BNP, B-type natriuretic peptide; COVID-19, coronavirus disease 2019; CK, creatine kinase; CK-MB, creatine kinase-MB; CRP, C-reactive protein; hs-cTnI, high-sensitivity cardiac troponin I; ICU, intensive care unit; IL-6, interleukin 6; LDH, lactate dehydrogenase; ROC, receiver operating characteristic.

Table 1

Median IQR Value Between Survivors and Nonsurvivors

Biomarkers	Survivors, Median (IQR)	Nonsurvivors, Median (IQR)	*P*	Mean Difference	95% CI
At the time of ICU admission					
CK	100.56 (51.27–139.54)	332.6 (225.9–413.70)	<0.01*	–204.96	–264.80 to –145.12
CK-MB	72.60 (30.17–194.15)	93.20 (55.90–198.45)	0.71	–9.01	–58.70 to –40.68
BNP	302.70 (98.65–478.75)	451.90 (390.30–655.0)	0.21	–265.52	–691.07 to 160.02
hs-cTnI	0.01 (0.01–0.02)	0.08 (0.03–0.13)	<0.01*	–0.11	–0.18 to –0.04
CRP	123.00 (62.45–259.75)	198.30 (119.60–242.01)	0.12	–82.06	–187.99 to 23.87
IL-6	95.00 (39.61–147.70)	224.83 (107.90–224.83)	<0.01*	–164.32	–269.09 to –59.55
D-dimer	0.78 (0.54–1.27)	1.11 (0.89–3.32)	<0.01*	–1.64	–2.78 to –0.49
Ferritin	816.90 (611.40–1004.40)	1000.30 (698.50–1453.00)	<0.05*	–237.69	–461.06 to –14.31
LDH	980.60 (763.35–1173.20)	1123.50 (890.40–1338.50)	0.05	–193.81	–393.41 to 5.79
Procalcitonin	0.06 (0.02–0.20)	0.12 (0.90–0.246)	0.81	0.03	–0.21 to 0.26
At 72 h after ICU admission					
CK	98.65 (48.15–135.60)	349.60 (228.60–487.56)	<0.01*	–242.83	–301.99 to –183.67
CK-MB	73.45 (34.05–189.50)	108.60 (63.21–211.40)	0.27	–27.98	–79.03 to 23.07
BNP	298.50 (93.90–489.50)	498.20 (402.60–668.90)	0.30	–256.72	–752.82 to 239.37
hs-cTnI	0.01 (0.01–0.03)	0.06 (0.01–0.13)	<0.01*	–0.08	–0.14 to –0.26
CRP	167.90 (90.28–267.30)	211.40 (176.30–289.40)	0.05	–98.59	–198.54 to 1.37
IL-6	110.30 (82.95–165.50)	201.23 (119.60–345.00)	<0.01*	–149.59	–247.24 to –51.94
D-dimer	1.09 (0.76–1.26)	0.98 (0.78–2.93)	0.01	–0.92	–1.65 to –0.20
Ferritin	898.59 (744.22–990.48)	1005.00 (703.45–1346.00)	0.11	–145.81	–327.64 to 36.01
LDH	903.80 (796.60–1.54.30)	1104.90 (805.70–1308.60)	0.05	–191.85	–387.95 to 4.24
Procalcitonin	0.05 (0.02–0.23)	0.13 (0.05–0.22)	0.59	0.06	–0.17 to 0.29

*$P < 0.05$ was considered statistically significant.

BNP, B-type natriuretic peptide; CI, confidence interval; CK, creatine kinase; CK-MB, creatine kinase-MB; CRP, C-reactive protein; hs-cTnI, high-sensitivity cardiac troponin I; IL-6, interleukin 6; LDH, lactate dehydrogenase.

survivors and nonsurvivors (Table 1). We considered an AUC of 0.90 to 1.00 as excellent, 0.80 to 0.90 as good, 0.70 to 0.80 as fair, 0.60 to 0.70 as poor, and 0.50 to 0.60 as fail. The ability to predict unfavorable outcomes was fair both at ICU admission with BNP (cutoff value = 326.8 pg/mL, AUC = 0.707, $P = 0.02$) and at 72 hours of admission (cutoff value = 398.5 pg/mL, AUC = 0.739, $P < 0.01$) and was statistically significant (Tables 2, 3). We obtained a good association for CK level at admission (cutoff value = 195.95 U/L, AUC = 0.899, $P < 0.01$) and excellent correlation at 72 hours after admission (cutoff value = 180.12 U/L, AUC = 0.939, $P < 0.01$), which was statistically significant (Tables 2, 3). We also obtained a good correlation for hs-cTnI level at admission (cutoff value = 0.035 ng/mL, AUC = 0.871, $P < 0.01$), which was statistically significant (Table 2). However, a fair correlation at 72 hours after admission was obtained for hs-cTnI level (cutoff value = 0.025 ng/mL, AUC = 0.798, $P < 0.01$) and was statistically significant (Table 3). Similarly, the cutoff values for various inflammatory markers, such as CRP, IL-6, D-dimer, ferritin, LDH, and procalcitonin at admission and 72 hours after ICU admission to predict 28-day mortality were also obtained by analyzing the ROC. The ability to predict unfavorable outcomes was fair both at admission for IL-6 (cutoff value = 114.5 mg/mL, AUC = 0.781, $P < 0.01$; Table 4) and at 72 hours admission (cutoff value = 132.1 mg/mL, AUC = 0.777, $P < 0.01$) and was statistically significant (Table 4). The D-dimer level at ICU admission was poorly associated (cutoff value = 0.975 mg/mL, AUC = 0.689, $P = 0.03$) but was statistically significant (Table 4). The D-dimer level at 72 hours after ICU admission failed to predict the appropriate correlation under AUC (cutoff value = 2.03 mg/mL, AUC = 0.582, $P = 0.35$) and was not statistically significant (Table 5).

Comparison of cardiac biomarkers among survivors and nonsurvivors

The median (IQR) of the cardiac markers (CK, CK-MB, BNP, and hs-cTnI) is shown in Table 1. At admission, the median values of CK and hs-cTnI were significantly higher in nonsurvivors than in survivors ($P < 0.01$ for both cardiac biomarkers; Table 2). At 72 hours after admission, the median values of CK and hs-cTnI were higher and

Table 2

Cutoff Value of Cardiac Biomarkers at ICU Admission

Cardiac Biomarkers	Normal Reference Range	Cutoff Value	AUC	Sensitivity, %	Specificity, %	*P*
CK	24–180 U/L	195.95	0.899	84.2	92	<0.01*
CK-MB	<25 IU/mL	112.10	0.585	47.4	60	0.33
BNP	<100 pg/mL	326.80	0.707	84.2	64	<0.05*
hs-cTnI	0.02–0.06 ng/mL	0.035	0.871	73.7	96	<0.01*

*$P < 0.05$ was considered statistically significant.

AUC, area under the receiver operating characteristic curve; BNP, B-type natriuretic peptide; CK, creatine kinase; CK-MB, creatine kinase-MB; hs-cTnI, high-sensitivity cardiac troponin I; ICU, intensive care unit.

Table 3

Cutoff Value of Cardiac Biomarkers 72 Hours after ICU Admission

Cardiac Biomarkers	Normal Reference Range	Cutoff Value	AUC	Sensitivity, %	Specificity, %	*P*
CK	24–180 U/L	180.12	0.939	89.5	92	<0.01*
CK-MB	<25 IU/mL	108.5	0.638	52.6	60	0.12
BNP	<100 pg/mL	398.5	0.739	89.5	68	<0.01*
hs-cTnI	0.02–0.06 ng/mL	0.025	0.798	73.7	76	<0.01*

*$P < 0.05$ was considered statistically significant.

AUC, area under the receiver operating characteristic curve; BNP, B-type natriuretic peptide; CK, creatine kinase; CK-MB, creatine kinase-MB; hs-cTnI, high-sensitivity cardiac troponin I; ICU, intensive care unit.

Table 4

Cutoff Value of Inflammatory Biomarkers at ICU Admission

Inflammatory Biomarkers	Normal Reference Range	Cutoff Value	AUC	Sensitivity, %	Specificity, %	*P*
CRP	0–5 mg/mL	190.5	0.625	63.2	60	0.16
IL-6	<6.4 mg/mL	114.5	0.781	73.7	68	<0.01*
D-dimer	<0.2 mg/mL	0.975	0.689	68.4	64	<0.05*
Ferritin	22–322 ng/mL	881.7	0.652	68.4	60	0.08
LDH	230–460 U/L	991.3	0.641	68.4	56	0.11
Procalcitonin	<0.2 ng/mL	0.112	0.648	68.4	60	0.09

*$P < 0.05$ was considered statistically significant.

AUC, area under the receiver operating characteristic curve; CRP, C-reactive protein; ICU, intensive care unit; IL-6, interleukin 6; LDH, lactate dehydrogenase.

statistically significant in nonsurvivors than in survivors ($P < 0.01$ for both cardiac biomarkers, respectively; Table 2).

Comparison of inflammatory biomarkers among survivors and nonsurvivors

The median (IQR) of the inflammatory markers (CRP, IL-6, D-dimer, ferritin, LDH, and procalcitonin) are shown in Table 1. At admission, the median values of IL-6, D-dimer, ferritin, and hs-cTnI were higher and statistically significant in nonsurvivors than in survivors ($P < 0.01$, $P < 0.01$, and $P = 0.03$, respectively; Table 1). At 72 hours after admission, the median values of IL-6 and D-dimer were higher and statistically significant in nonsurvivors than in survivors ($P < 0.01$ and $P = 0.01$, respectively; Table 1).

Discussion

This retrospective observational study summarizes the evidence regarding biomarkers as prognosticators of outcomes in COVID-19 and outlines the utility of cardiac biomarkers in predicting mortality as a primary outcome among nonsurvivors. This study is unique because it is the first study done within the second wave time frame and analyzed the various cardiac and inflammatory biomarkers among survivors and nonsurvivor as separate subgroups. Our study demonstrated a statistically significant association between disease severity and cardiac injury. Our analysis showed that BNP, CK-MB, CK, CRP, IL-6, and LDH levels were higher among nonsurvivors than survivors (Table 3).

Our study showed a relationship between plasma BNP levels and the risk of ICU death in patients with severe COVID-19 in the second wave. All enrolled patients with severe COVID-19 have high BNP (cutoff value = 398.5 pg/mL at 72 hours) and high CK levels (cutoff value = 180.12 U/L at 72 hours) are either in younger or older age groups, and they also have higher levels of systemic inflammation markers except procalcitonin and D-dimer.

Wang et al. (2020) published a case series study of 138 patients with COVID-19 and affirm that acute cardiac injury (22.2%) and arrhythmia (44.4%) were higher in patients with severe COVID-19, and they have elevated CK-MB and hypersensitive troponin I levels.[6] The mechanism underlying severe acute respiratory syndrome coronavirus 2–induced cardiac injury remains unclear. Xu et al.[7] (2020) observed inflammation-induced cardiac injury, and postmortem heart biopsy revealed few interstitial mononuclear inflammatory infiltrates. Another proposed mechanism by Donoghue et al.[8] (2000) suggested the invasion of cardiomyocytes via the binding site of angiotensin-converting enzyme–related carboxypeptidase (ACE2) in COVID-19. The sudden decrease in myocardial oxygen supply after pulmonary infection due to COVID-19, and the presence of cytokine storm syndrome may also contribute to cardiac injury.[9]

This study had several limitations. First, the sample size is small. Second, we evaluated patients with severe COVID-19 admitted to the ICU; therefore, our results cannot be generalized to all patients with COVID-19, specifically those with less severe forms of the disease. Moreover, the heterogeneous population in the present study was another disadvantage. Further studies using larger sample sizes will provide a higher level of evidence for assessing these cardiac markers. However, this study assessed a single test of cardiac biomarkers, such as CK, CK-MB, BNP, and hs-cTnI, and various inflammatory markers, such as CRP level, IL-6, D-dimer, ferritin, LDH, IL-6, and procalcitonin, in patients with severe COVID-19 at admission. Finally, our single-center study may have caused selection bias. Further multicenter studies are required to validate the performance of these biomarkers.

Conclusion

In patients with severe COVID-19, CK-MB and hs-cTnI may be considered effective and valuable predictive cardiac biomarkers among nonsurvivors and predict poor prognosis. Inflammatory markers, such as CRP, IL-6, and D-dimer, had a better performance for prognosis prediction among nonsurvivors at ICU admission. Therefore, early determination of CK-MB and hs-cTnI may help predict the possibility of adverse outcomes and improve patient prognosis.

Table 5

Cutoff Value of Inflammatory Biomarkers 72 Hours after ICU Admission

Inflammatory Biomarkers	Normal Reference Range	Cutoff Value	AUC	Sensitivity, %	Specificity, %	*P*
CRP	0–5 mg/mL	194.9	0.646	68.4	60	0.10
IL-6	<6.4 mg/mL	132.1	0.777	73.7	72	<0.01*
D-dimer	<0.2 mg/mL	2.03	0.582	47.4	96	0.35
Ferritin	22–322 ng/mL	902	0.625	63.2	60	0.15
LDH	230–460 U/L	992.8	0.613	63.2	60	0.20
Procalcitonin	<0.2 ng/mL	0.12	0.623	57.9	64	0.16

*$P < 0.05$ was considered statistically significant.

AUC, area under the receiver operating characteristic curve; CRP, C-reactive protein; ICU, intensive care unit; IL-6, interleukin 6; LDH, lactate dehydrogenase.

Conflict of interest statement

The authors declare no conflict of interest.

Author contributions

Kumar N contributed to the conception and design of the study; Ahmad S and Kumar N collected the patient's medical information; Mahto M and Kumar A contributed to data cleaning and analysis. Kumar A performed the statistical analysis. Singh PK and Ahmad S drafted the manuscript. All authors contributed to manuscript revision and read and approved the submitted version.

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Ethical approval of studies and informed consent

This study was approved and written informed consent was waived by the departmental research committee of the All India Institute of Medical Sciences Patna, Bihar, India (141/T&E/AIIMS/Pat/2022, dated March 10, 2022) because of the retrospective nature of the study.

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