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ORIGINAL ARTICLE

NT PROBNP'S Prognostic Value Among Patients of Covid-19 without Heart Failure

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ABSTRACT

Objective: If Nt Pro BNP's prognostic value increased the mortality rate and hospitalization of covid-19 patients.
Study Design: Retrospective study
Place and Duration: Tehsil Headquarter Hospital Minchinabad. April 2021-Dec 2021.
Methods: This research included 85 patients of both sexes with ages 18-65 years had coronavirus disease. After obtaining informed written permission, demographic data such as age, gender, height, weight, and place of residence were collected on all recruited participants. Comorbidities among all cases were assessed. NT proBNP> 88.64 pg/mL was considered as high and low was NT-proBNP≤88.64 pg/mL. Hospitalization and death rate among all cases were calculated.
Results: There were 52 (61.2%) males and 33 (38.8%) females among all cases. The mean age of the enrolled cases was 55.7±17.76 years and had mean BMI 26.4±11.44 kg/m^2. Hypertension was found in 35 (41.2%), chronic pulmonary disease in 20 (23.5%) cases, diabetes mellitus in 18 (21.2%) cases and 12 (14.1%) patients had chronic heart disease. Among 85 cases, 49 (57.6%) patients had high NT proBNP> 88.64 pg/mL and 36 (42.3%) patients had NT-proBNP≤88.64 pg/mL. We found higher level of CRP, creatinine and urea among patients of higher Nt ProBNP's value. Mortality in patients of higher Nt ProBNP's was found among 20 (40.8%) cases and no any death case found among patients of lower Nt ProBNP's value.
Conclusion: We concluded in this study that increase in Nt ProBNP's value among patients of coronavirus cause higher number of deaths.
Keywords: Covid-19, HTN, Nt ProBNP, Mortality

INTRODUCTION

The SARS-CoV-2-caused coronavirus illness 2019 (COVID-19) in China has been deemed a public health emergency of international concern. However, SARS-CoV2 has killed a greater number of individuals than either SARS or MERS.[2]. In epidemiological studies, the intensity of COVID19 sickness has been shown to be linked to poor outcomes. Predictive markers for people with severe disease are critical to designing an effective treatment strategy. [3,4]

Patients diagnosed with SARS-CoV-2 infections have a higher risk of experiencing cardiac damage, which in turn increases the likelihood that they will have a more dire prognosis. [5]. Several cardiac biomarkers, such as cardiac troponin I (cTnI), alpha-hydroxybutyrate dehydrogenase, myoglobin, lactate dehydrogenase (LDH), creatine phosphokinase (CPK), creatinine phosphokinase–muscle/brain CPK-MB, and aspartate aminotransferease (AST)/N-terminal of the prohormone brain natriuretic peptid (COVID-19). [6] Although there is a chance that indicators of heart injury may be elevated, this is not always the case in every patient. Patients diagnosed with COVID-19 who are being treated in the intensive care unit have a greater risk of having elevated levels of cardiac biomarkers such as cTnI and CPK-MB. Arrhythmia, myocarditis, cardiogenic shock, substantial myocardial damage, or heart failure are all possible signs that the heart is implicated in some manner. [7] According to the findings of another research, acute cardiac damage affected 7.2% of the 138 COVID-19 patients who were investigated, and arrhythmia affected 16.7% of them. When there is serious illness present, these percentages go up to 22.2 percent and 44 percent, respectively [8]. There are a number of different pathways that might lead to myocardial injury, but the origin of the SARS-CoV-2-induced cardiac damage is not yet understood. An arrhythmia may develop as a consequence of a direct viral infection brought on by intracellular replication. This causes the cardiomyocytes to deteriorate and eventually die. In addition, the influence that the virus has on angiotensin-converting enzyme 2 (ACE2), which is extensively expressed in the heart and lungs, may be mediated via this enzyme. [9] The immune system of the body itself is another another route that the SARS-CoV-2 virus may use to cause harm to the heart. [10] In multiple different examinations, patients with COVID-19 have been shown to have increased levels of inflammatory markers. Because these processes are reflected in prognostic cardiac biomarkers, having a solid grasp of these indicators is essential to properly navigating these pathways. It is possible to predict heart failure, acute coronary syndromes, valvular aortic stenosis, and stable coronary artery disease by analysing the amount of BNP and NT-proBNP that is released from the cardiac myocardium in response to increased wall stress [13, 14].

At least 22.2 percent and 44.4% of severe patients were found to have acute heart damage or arrhythmias. C-reactive protein (CRP) and hypersensitivity troponin I (HS-TnI) levels were greater in individuals with severe COVID-19 [15].

A recent study found that the heart failure marker NT-proBNP increased significantly during hospitalisation in patients who died [6]. No research have shown if NT-proBNP can predict the outcome of severely ill COVID-19 patients.

MATERIAL AND METHODS

This retrospective study was conducted at Tehsil Headquarter Hospital Minchinabad and comprised of 85 patients. After obtaining informed written permission, demographic data such as age, gender, height, weight, and place of residence were collected on all recruited participants. In the absence of NT-proBNP testing, patients were not considered. Stroke and acute myocardial infarction patients, cancer patients and pregnant women were excluded from the study.

The chi-square test, the T-test (for regularly distributed continuous variables), and the Mann-Whitney U test (for nonnormally distributed continuous variables) were all unable to identify any statistically significant differences between the NT-proBNP high and low groups (categorical variables). The NT-proBNP cut-off value that was considered to be the most optimal was the one that had the highest level of both sensitivity and specificity. The survival curves of patients who passed away while they were still being treated at the hospital were constructed using a Kaplan-Meier product-limit estimate as well as log-rank testing. Spearman's coefficients of correlation were used in order to

investigate any relationships that may have existed between NT-proBNP and the other variables. Independent analysis of the influence of NT-proBNP on in-hospital mortality was performed with the use of Cox proportional hazards models. These models were used to exclude potentially relevant risk factors. The analysis of all the data was done using SPSS 21.0.

RESULTS

There were 52 (61.2%) males and 33 (38.8%) females among all cases. The mean age of the enrolled cases was 55.7±17.76 years and had mean BMI 26.4±11.44 kg/m^2.Mostly cases were from urban area and were not educated.(table 1)

Table-1: Baseline details of enrolled cases

Variables	Frequency	Percentage
Mean age (years)	55.7±17.76	
Mean BMI (kg/m^2)	26.4±11.44	
Gender		
Male	52	61.2
Female	33	38.8
Residency		
Urban	46	54.1
Rural	39	45.9
Education status		
Literate	33	38.8
Non-educated	52	61.2

Hypertension was found in 35 (41.2%), chronic pulmonary disease in 20 (23.5%) cases, diabetes mellitus in 18 (21.2%) cases and 12 (14.1%) patients had chronic heart disease.(fig 1)

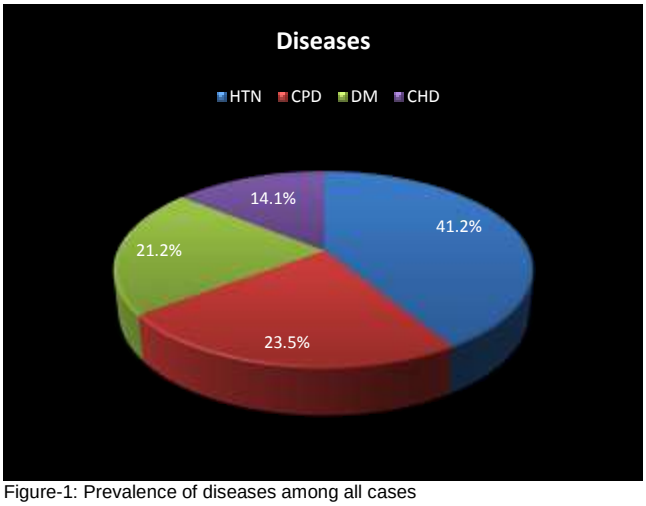

Figure-1: Prevalence of diseases among all cases

Among 85 cases, 49 (57.6%) patients had high NT proBNP> 88.64 pg/mL and 36 (42.3%) patients had NT-proBNP≤88.64 pg/mL.(table 2)

Table-2: Association of NT-proBNP value among cases

Variables	Frequency	Percentage
NT proBNP > 88.64 pg/mL	49	57.6
NT proBNP ≤88.64 pg/mL	36	42.3
Total	85	100

Table-3: Laboratory and adverse outcomes among cases

Variables	NT proBNP > 88.64 pg/mL	NT proBNP ≤88.64 pg/mL
CRP (mg/L)	52.3±10.32	9.13±8.56
Creatinine (umol/L)	78.15±6.45	53.6±3.64
Urea (umol/L)	8.5±6.16	3.18±7.31
Mortality		
Yes	20 (40.8%)	0
No	29 (59.2%)	36 (100%)

We found higher level of CRP, creatinine and urea among patients of higher Nt ProBNP's value. Mortality in patients of higher Nt ProBNP's was found among 20 (40.8%) cases and no any death case found among patients of lower Nt ProBNP's value.(table 3)

DISCUSSION

An connection between plasma NT-proBNP levels, which were measured in COVID-19 patients, and death while in the hospital was found in this study. COVID-19 patients with high NT-proBNP levels had an increased risk of being older, having raised heart damage indicators, and having higher levels of systemic inflammatory mediators. [16] The cumulative survival rate was worse in individuals with high NT-proBNP levels (> 88.64 pg/mL). An independent risk factor for death in the hospital in patients with severe COVID-19 has been discovered to be NT-proBNP, even in the presence of potential confounding factors.

In this study 85 patients of both genders were presented. There were 52 (61.2%) males and 33 (38.8%) females among all cases. The mean age of the enrolled cases was 55.7±17.76 years and had mean BMI 26.4±11.44 kg/m^2.Mostly cases were from urban area and were not educated. These were in line with the previous studies.[16,17] Hypertension was found in 35 (41.2%), chronic pulmonary disease in 20 (23.5%) cases, diabetes mellitus in 18 (21.2%) cases and 12 (14.1%) patients had chronic heart disease. An rise in hypersensitive troponin I (hs-cTnI) (>28 pg/mL) in 5 of 41 COVID-19 patients (12 percent) was the most common symptom of a virus-related cardiac damage, according to a recent research. [18] Among 85 cases, 49 (57.6%) patients had high NT proBNP> 88.64 pg/mL and 36 (42.3%) patients had NT-proBNP≤88.64 pg/mL. The number of patients with NT-proBNP levels below 300 pg/ml was double that of those with aberrant hs-cTnI values in earlier studies. This finding suggested that cardiac dysfunction, rather than cardiac damage, was more frequent in COVID-19 patients than previously thought. More over one-third of patients with pneumonia exhibited temporary left ventricular dysfunction, according to a previous research, even though they had no prior history of cardiac illness. [19,20]

We found higher level of CRP, creatinine and urea among patients of higher Nt ProBNP's value. Mortality in patients of higher Nt ProBNP's was found among 20 (40.8%) cases and no any death case found among patients of lower Nt ProBNP's value. Researchers recently found that COVID-19 patients with cardiac injury had higher BNP levels and a greater death rate than non-COVID-19 patients [21]. NT-proBNP levels and early detection of the possibility for HF may be important in COVID-19 to prevent and treat cardiac issues. In addition, we discovered that patients on the COVID-19 who had increased NT-proBNP concentrations had worse clinical results.

The release of NT-proBNP occurs when the heart is under stress. [22] Interleukin 1 (IL-1), C-reactive protein (CRP), and cardiotrophin I (CTI) have been shown to impact chronic kidney disease (CKD).[23,24] Non-heart-controlled functions According to the findings, high levels of NT-proBNP are linked to cardiovascular disease, kidney damage, and generalised inflammation. There was additional evidence from the Cox proportional-hazards analysis indicating that these variables led to an increased risk of dying while in the hospital. The NT-proBNP risk factor was not shown to be significantly different by Cox multivariate analysis. As a potential indicator of the overall severity of SARS-CoV-2 infection, NT-proBNP may have a predictive value of some significance. In individuals with severe COVID-19, NT-proBNP levels may be able to identify patients with unfavourable prognoses early on.

CONCLUSION

We concluded in this study that increase in Nt ProBNP's value among patients of coronavirus cause higher number of deaths.

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