Role of Troponin I in Prediction of Mortality in Covid 19 Patients

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Abstract

While most COVID-19 cases present with mild respiratory symptoms resembling a common cold, the situation is globally serious. Severe outcomes are more prevalent in older male patients with underlying health conditions. Although, most COVID-19 cases present with mild respiratory symptoms resembling a common cold, yet severe outcomes are more prevalent in older male patients with underlying health conditions. As this is novel infection and its cardiac manifestations and outcomes are not fully determined yet in any population so there is a need to conduct a study to known prevalence of raised cardiac markers in Covid-19 infection in the absence of any cardiac defect so that further management can be planned. Our study showed that there is not only a raise in levels of Troponin-I but also has a strong association with increased mortality

1. Introduction

On December 26, 2019, a patient in Wuhan, Hubei province, China, a 41-years old man was admitted to the hospital with symptoms resembling severe pneumonia(1). Similar cases emerged in the province, and by early January 2020, it was determined that a novel subtype of the Coronaviridae family, named severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), was the cause(1-3) In late January 2020, the World Health Organization declared SARS-CoV-2 a Public Health Emergency of International Concern and officially named the associated illness "coronavirus disease 2019" (COVID-19) (4). Severe outcomes were reported more in older male patients with secondary comorbidities (5), stacking a mortality of over a million by November 2020 (6).

While most COVID-19 cases present with mild respiratory symptoms resembling a common cold, the situation is globally serious. Severe outcomes are more prevalent in older male patients with underlying health conditions. SARS-CoV-2, a betacoronavirus with a 29,903 bp single-stranded RNA, targets angiotensin-converting enzyme (ACE)-2 receptors to enter host cells(7). Frequently-reported symptoms of patients admitted to the hospital with COVID-19 infection include fever, cough, myalgia, fatigue, and shortness of breath (3–31%) at illness onset. Approximately 20–30% of patients hospitalized with COVID-19 and pneumonia have required respiratory support in intensive care units (ICUs) (8).

Other manifestation includes cardiac involvement including myocarditis and even myocardial infraction. Human coronavirus-associated myocarditis is known, and a number of coronavirus disease 19 (COVID-19)–related myocarditis cases have been reported. The pathophysiology of COVID-19–related myocarditis is thought to be a combination of direct viral injury and cardiac damage due to the host's immune response. The clinical findings include changes in electrocardiogram and cardiac biomarkers, and impaired cardiac function (9, 10).

Myocardial injury, detected by elevated plasma troponin levels, has been associated with mortality in patients hospitalized with coronavirus disease 2019 (COVID-19) (11, 12). 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 (13, 14). Huang et al reported that 12% of patients with COVID-19 were diagnosed as having acute myocardial injury, manifested mainly

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by elevated levels of high-sensitive troponin I (15). Gou et al. found that 27.8% of covid-19 patients exhibited myocardial injury as indicated by elevated Troponin levels. The mortality during hospitalization was 7.62% for patients without underlying CVD and normal Troponin levels, 13.33% for those with underlying CVD and normal Troponin levels, 37.50% for those without underlying CVD but elevated Troponin levels, and 69.44% for those with underlying CVD and elevated Troponin (16).

COVID-19, as a global pandemic, poses a significant healthcare challenge, with ongoing research focusing on its pathophysiology, disease progression, and causes of mortality. The increasing prevalence of COVID-19 and delays in its management contribute to higher mortality rates. Although multiple biomarkers assess disease progression and severity, the accuracy of these markers remains debated. Present data suggests that Troponin-I is more specific to COVID-19, prompting this study to investigate its significance in the early prediction of COVID-19-related mortality (12).

As this is novel infection and its cardiac manifestations and outcomes are not fully determined yet in any population so there is dire need to conduct a study to known prevalence of raised cardiac markers in Covid-19 infection in the absence of any cardiac defect so that further management can be planned.

2. Objective

To determine frequency of mortality in patients with raised Troponin-I during COVID-19 infection.

3. Material And Methods

The study was Retrospective cross-sectional study, conducted at Emergency medicine department, Ziauddin hospital, Karachi. All the patients admitted in the hospital in during September 2021 to August 2022. Non-probability convenient sampling. Technique was used.

Patients having age range from 18 year and above of both genders, having COVID -19 infection admitted to the hospital with covid rapid antigen or covid PCR positive. Patients excluded in the study were those having S-T segment elevation or depression in ECG, having multiple organ dysfunction / failure, previous history of cardiac disease. (will be determined on history and medical record) and patient not given consent

Patients who fulfil the inclusion criteria were enrolled and written informed consent was taken from them or guardians. Demographic data including age, gender, duration of symptoms, diabetes (BSR more than 200mg/dl), hypertension (BP more than 130/90mmHg), dyslipidemia (fasting cholesterol more than 200mg/dl), obesity (BMI more than 25Kg/m2) and smoking (more than 3 pack years) were noted. All the patients were tested for cardiac markers.

Data was analyzed using SPSS. Mean and standard deviation were calculated for all quantitative variables like age and duration of symptoms. Frequency and percentage were calculated for all qualitative variables like gender, dyslipidemia, hypertension, diabetes mellitus, smoking, obesity, raised cardiac markers and hospital outcomes. Patients with raised cardiac markers and normal cardiac markers were compared for hospital outcomes using paired sample T-test, a p-value ≤ 0.05 was taken as significant.

4. Results

A total of 223 patients were included in the study after applying the selection criteria. Amongst these 123 were males and 91 were males while 9 patients asked not to disclose their gender. Mean age of the study participants was reported to be 61.00+18.25 years (Discharge group=53.46+18.69, mortality group= 68.92+14.12)

Amongst vitals, blood pressure and pulse rate showed a non-significant effect on outcome, while respiratory rate (RR), oxygen saturation (SO2) and temperature showed a significant association with worsening in outcome. and Table 1: Vitals of the study participants on the basis of outcome

Vitals	Death	Discharge	p-value
BP. Sys	131.77+31.96	132.58+23.61	0.837
BP. Dys	76.74+22.70	77.38+16.19	0.819
Pulse	98.40+22.28	89.88+14.79	0.002
RR	22.63+2.12	21.49+2.00	<0.001
SO2	83.37+9.44	92.75+7.88	<0.001
Temp	101.01+1.61	100.02+1.67	<0.001

Our study found that there is a strong association between the levels of Troponin (keeping cut off at 0.1) I with outcome (p=<0.001) (table 2)

Outcome	Mean	Std. Error Mean	p-value
Death	6.62+9.73	0.907	<0.001
Discharge	1.10+3.17	0.305	

5. Discussion

Our study showed that males over age of 60 showed poor out come and prognosis as compared to other age groups and gender, our findings were in concordance with the studies published before (6, 8, 15). Our study showed a non-significant association with blood pressure and pulse rate, which are in similar to that reported by Ali et al (17).

COVID-19 infection was not only reported to be affecting respiratory tract but not only physically to other organs such as cardiovascular (12), liver (18), kidney (19), brain (5), but also psychologically (20). Our study aimed to assess the effect of the infection on heart excluding myocardial infarction or any such condition that might affect electric activity of the heart (i.e., ECG changes).

Various patterns of cardiovascular involvement have been identified in the context of COVID-19. Firstly, pre-existing cardiovascular diseases may not only surface but also become more intricate and decompensated during the course of COVID-19. Secondly, the cardiovascular

system's susceptibility to systemic inflammatory responses during the progression of COVID-19. Thirdly, treatment-induced effects on the cardiovascular system, particularly stemming from the side effects of medications or complications arising from secondary hospital-acquired infections (16). Although the pathology usually is focal within the myocardium, but there is a risk of arrhythmia as well as progression to fulminant heart failure and cardiogenic shock (8)

Our study concluded that there is a significant association with raise in troponin levels with the patient outcome, i.e., discharge or mortality. This raised troponin in absence of any change in ECG can be interpreted as early phase of cardiomyocyte injury which leads to release of troponins in the blood stream (13, 21).

6. Conclusion

Our study concluded that there is a strict monitoring of cardiac activity not only by ECG changes but also by troponin levels should be done in COVID patients to improve the outcome of the patients.

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