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Correlation Between Severe Covid-19 and Serum Troponin Regarding Significant Cardiac Disease

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Abstract— Background*: Coronavirus disease 2019 (COVID-19) is a viral disease that affected multiple organs in the body. Identification of predictors for elevated serum troponin and significant cardiac disease involvement can help in managing the disease.* ***Aims****: to correlate between severe Covid-19 and S.troponin regarding significant cardiac disease.* ***Patients and Methods****: This was a cross-sectional study including 90 patients with SARS-CoV-2, Patients demographics and comorbidities were collected through direct interview. Laboratory parameter (C- reactive protein, D-Dimer) were obtained from patient 's record during days of admission, Laboratory parameter (serum Troponin I) which was ordered with daily routine investigations. Electrocardiography and Echocardiography were ordered when S.troponin is above (> 0.04 ng/ml). Echocardiography was done by a echocardiologist or a cardiologist at Al- Shiffa Hospital.* ***Results****: Serum Troponin Positive and Significant Cardiac disease in patients having a comorbidity in general was (15[21.74%] and 9[13.04%], respectively) compared with (2[18.88%] and 2 [18.22%], respectively) in patients with no comorbidity Although patients with comorbidity had higher percentage of cardiac disease than those without comorbidity, the difference statistically was not significant. In particular, patients with hypertension, DM, IHD and HF as comorbidity, had more Positive Serum Troponin and more Significant Cardiac disease (15[30.61%] and 9[18.37%],13[36.11%] and 8[22.22%], 12[26%] and 6[50%],6[75%] and 4[50%], respectively) than those without these comorbidities, the difference statistically was significant. Positive serum troponin had a positive significant correlation with each of serum CRP (r=0.35, p= 0.002), D-dimer (r=0.22, p= 0.048) and age (r= 0.31, p= 0.006) Although patients with elevated, D-dimer and older age had higher percentage of cardiac disease, the difference statistically was not significant.* ***Conclusions****: Older age patients and those with comorbidities, especially DM, IHD, HF and hypertension, are more prone for positive serum troponin and greater risk for significant cardiac disease in COVID-19. Inflammatory markers, in particular, CRP and D-dimer are significantly correlate with positive serum troponin and greater risk for significant cardiac disease. NSTEMI is more frequent than other cardiac diseases*.

Keywords*— Covid-19, s.troponin, echocardiography, cardiac disease*.

I. BACKGROUND

As a result of the coronavirus disease 2019 (COVID-19) pandemic, hospitalizations for multiorgan-involved pneumonia have abruptly increased significantly. In this case, COVID-19 is due to the new coronavirus (SARS-CoV-2). Infection with SARS-CoV-2 may cause no symptoms at all or a wide range of them, including mild signs of an upper respiratory infection and potentially deadly sepsis (1).

When a cluster of individuals in Wuhan, China, were diagnosed with pneumonia of uncertain origin in December 2019, COVID-19 initially became known. SARS-CoV-2 has affected more than 200 nations as of July 1, 2020, resulting in more than 10 million cases that have been identified and 508 000 deaths that have been confirmed. (2). Patients admitted for COVID-19 often exhibit increased cardiac troponin levels, indicative of myocardial injury, however the etiology of this phenomenon remains ambiguous (3).

Hospitalized COVID-19 patients have varying rates of myocardial damage, with reported rates ranging from 7 to 36 percent (4, 5), which is indicated by an increase in cardiac troponin levels. Patients with COVID-19 who only have modest symptoms seem to experience troponin increase less frequently (6). The variability in troponin assays, 99th percentile upper reference limit thresholds, and sampling periods complicates the comparison of troponin elevation frequencies across various COVID-19 studies (7). On the prevalence of troponin increases in individuals with SARS-CoV-2 infection who are asymptomatic or just slightly symptomatic, there is a paucity of information.

Troponin increases have been found to occur more frequently and to a higher extent in hospitalized patients with more severe illnesses and poorer prognoses (4, 8). An increased troponin level was one of the clinical characteristics most significantly correlated with worse outcomes; OR 10.58, 95% CI 5.00-22.40, as per a systematic review and meta-analysis (9). Acute kidney injury, history of cardiovascular disease, elevated procalcitonin, elevated D-dimer, and thrombocytopenia were additional clinical characteristics linked with a high risk of the composite outcomes. One of the clinical characteristics linked with in-hospital mortality was an increased troponin level.

Aim of the Study

To identify correlation between sever COVID-19 and serum troponin in face of significant cardiac disease.

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II. PATIENTS AND METHODS

This was a cross-sectional study including 90 patients infected with SARS-CoV-2 who were admitted and treated at Baghdad City Complex (Al-Shiffa Hospital) during the period from 7th October 2021 to 22nd July 2022. Real-time polymerase chain reaction analysis of SARS-CoV-2 RNA from nasopharyngeal swabs provided the diagnosis for the patients. according to WHO guidelines 27th May 2020. The study was approved by the Arab board of Health Specializations.

Inclusion Criteria

All adult patients confirmed with SARS-CoV-2 infection (severe and critical) with laboratory features associated with severe Covid-19 infection.

Exclusion criteria

Patients who refused to participate in the study.
Age <18 years
Patients on Hemodialysis or on vasopressors (10 patients)
Ethical consideration: Prior to collecting any data, each participant gave their verbal agreement after being informed of the study's objectives. The option to decline participation was completely unrestricted for each patient. Patients were given the assurance that their information would only be utilized for research purposes throughout the trial, which guaranteed the confidentiality of their data.
Data Collection: Patients demographics (age, gender) comorbidities (diabetes, hypertension, ischemic heart disease, chronic kidney disease, Asthma and others) were collected by direct interview. Laboratory parameter (C-reactive protein, D-Dimer) were collected from patient 's record during days of admission, Laboratory parameter (serum Troponin I) which was ordered with daily routine investigations. (measurement done by Roche/Cobas C311 approved by Ministry of Health and Environment of Iraq).

Electrocardiography and Echocardiography were ordered when S.troponin is above (> 0.04 ng/ml) . Echocardiography was done by a echocardiologist or a cardiologist at Al Shiffa Hospital.

Quantitative data was expressed statistically using the mean and standard deviation. Binomial data was presented using frequency percentages. To investigate the potential associations between demographic and laboratory data and major heart disease and blood troponin level, point-biserial correlation was used. Every piece of data was examined using IBM Corp. Armonk's SPSS which is based in New York, USA.

III. RESULTS

The mean age of the patients was 57.41±16.89 years (range 19-95 years). Slightly less than half of the patients (47.5%) were males. Less than fifteen of patients (13.75%) had no past medical history of comorbid diseases. Hypertension and DM were common comorbidities accounting for 61.25% and 45% of the patients, respectively whereas minority of the patients (15%),(10%) and (6.25%) had ischemic heart disease (IHD) ,Heart failure (HF) and chronic kidney disease (CKD) ,respectively (Table 1).

Other comorbidities include 8 cases of cerebrovascular accident (CVA), five cases of asthma, one case of each of epilepsy, IgA nephropathy, Hellp syndrome, psoriasis, acute pancreatitis, renal transplant, lung cancer and pulmonary fibrosis.

TABLE 1: Patients 'characteristics, laboratory and demographic data (n=80)

Variables	Values
Age, years	
Mean±SD	**57.41±16.89**
Range	19-95
Gender	
Male	38(47.5%)
Female	42(52.5%)
Comorbidities*	
None	11(13.75%)
Hypertension	**49(61.25%)**
Diabetes mellitus	36(45%)
Ischemic heart disease	12(15%)
Chronic kidney disease	5(6.25%)
Heart failure	8(10%)
Others	21(26.25%)
C-reactive protein level (mg/L)	
Mean±SD	167.91±31.23
Range	108-220
D-dimer level (ng/mL)	
Mean±SD	6511.65±3157.3
Range	1266-11977

*Many patients had more than one comorbidity.

Diagnostic tool and Laboratory Findings Table 2 illustrates the biochemical and Diagnostic tool of the studied patients. The Troponin I level was 0.10 ±0.26ng/mL (Normal value range 0-0.04 nanograms per milliliter (ng/mL)). 63 (78.75%) of patients had ≤0.04ng/mL cTnI level and 17 (21.25%) of patients had >0.04ng/mL cTnI level which 11 (64.70%) of patients had significant cardiac disease by ECG or Echocardiography and 6 (35.30%) of patients nothing significant were found by serial cTnI, ECG or echocardiography.

TABLE 2: Clinical and laboratory findings (n=80)

Variables	Values
Troponin Ilevel (ng/mL)	
Mean±SD	0.10±0.26
Median	0.025
Range	0.01-1.65
cTnI level Positive (> 0.04 ng/mL)	
≤0.04 ng/mL	63(78.75%)
>0.04 ng/mL	17(21.25%)
cTnI > 0.04 and cardiac disease	
Significant cardiac disease	11(64.70%)
Nothing significant after serial cTnI , ECG or echocardiography	6(35.30%)

Association of Patients 'Characteristics with Serum Troponin , Positive Serum Troponin and Significant Cardiac disease The Serum Troponin Positive and Significant Cardiac disease in patients having a comorbidity in general was (15[21.74%] and 9[13.04%],respectively) compared with (2[18.88%] and 2[18.22%],respectively) in patients with no comorbidity Although patients with comorbidity had higher

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percentage of Cardiac disease than those without comorbidity , the difference statistically was not significant . In particular, patients with hypertension, DM, IHD and HF as comorbidity, had more Positive Serum Troponin and more

Significant Cardiac disease (15[30.61%] and 9[18.37%], 13[36.11%] and 8[22.22%], 12[26%] and 6[50%], 6[75%] and 4[50%], respectively) than those without these comorbidities, the difference statistically was significant (Table 3)

TABLE 3: Association of Patients 'Characteristics with Serum Troponin, Positive Serum Troponin and Significant Cardiac disease

Variables	CTnI Level ng/mL	CTnI positive (+ve)	Significant Cardiac disease
Age	0.11±0.26	-ve (54.73±17) +ve (67.35±12.35)	-ve (56.17±17.28) +ve (65.18±12.16)
P-value	0.59	**0.006**	0.10
Gender			
Male (n=38)	0.09±0.25	7(18.42%)	5(11.9%)
Female (n=42)	0.11±0.27	10(23.8%)	6(15.78%)
P-value	0.79	0.55	0.88
Comorbidity			
Yes (n=69)	0.09±0.21	15(21.74%)	9(13.04%)
No (n=11)	0.19±0.49	2(18.18%)	2(18.18%)
P-value	0.54	0.78	0.64
Hypertension			
Yes (n=49)	0.12±0.24	15(30.61%)	9(18.37%)
No (n=31)	0.08±0.29	2(6.45%)	2(6.45%)
P-value	0.61	0.01	0.13
Diabetes mellitus			
Yes (n=36)	0.14±0.27	13(36.11%)	8(22.22%)
No (n=44)	0.08±0.26	4(9.09%)	3(6.82%)
P-value	0.35	**0.003**	**0.047**
Ischemic heart disease			
Yes (n=12)	0.15±0.1	12(26%)	6(50%)
No (n=68)	0.10±0.28	5(14.7%)	5(7.35%)
P-value	0.56	**0.001**	**0.001**
Heart failure			
Yes (n=8).	0.2±0.18	6(75%)	4(50%)
No (n=72)	0.1±0.27	11(15.28%)	7(7.35%)
P-value	0.296	**0.001**	**0.002**
Chronic kidney disease			
Yes (n=5)	0.11±0.24	2(40%)	1(20%)
No (n=75)	0.10±0.29	15(20%)	10(13.33%)
P-value	0.56	0.29	0.67
Others			
Yes (n=21).	0.06±0.12	3(14.29%)	2(9.52%)
No (n=59)	0.12±0.3	14(23.73%)	9(15.25%)
P-value	0.399	0.36	0.51

Correlation of Laboratory Findings with Positive Serum Troponin and Significant Cardiac disease point-biserial correlation was used to explore the possible correlation of demographic and laboratory findings with Positive serum troponin, had a positive significant correlation with each of serum CRP (r=0.35, p= 0.002), D-dimer (r=0.22, p= 0.048) and age (r= 0.31, p= 0.006) as shown in table 4, figure 3, 4, 5, 6, 7 and 8.

TABLE 4: point-biserial correlation of demographic and laboratory findings positive serum Troponin and significant cardiac disease.

Variable	Positive Serum Troponin		Significant Cardiac disease	
	Coefficient	p-value	Coefficient	p-value
Age	0.31	**0.006**	0.18	0.101
D-dimer	0.22	**0.048**	0.1	0.397
CRP	0.35	**0,002**	0,18	0,105

TABLE 5: positive serum troponin and diagnosis of cardiac disease. see figure 2.

Variables	Values
cTnI > 0.04 and cardiac disease (ECG and echocardiograph)	9(52.94%)
NSTEMI	1(5.88%)
STEMI	1(5.88%)
Atrial Fibrillation	6(35.30%)
No definitive diagnosis	

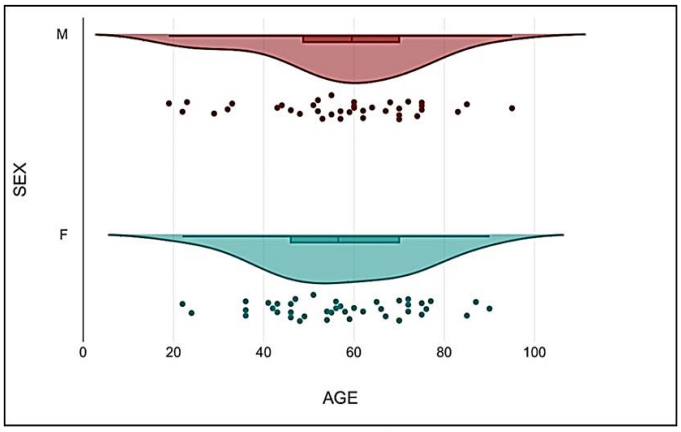

Figure 1: Raincloud Plot for sex and age

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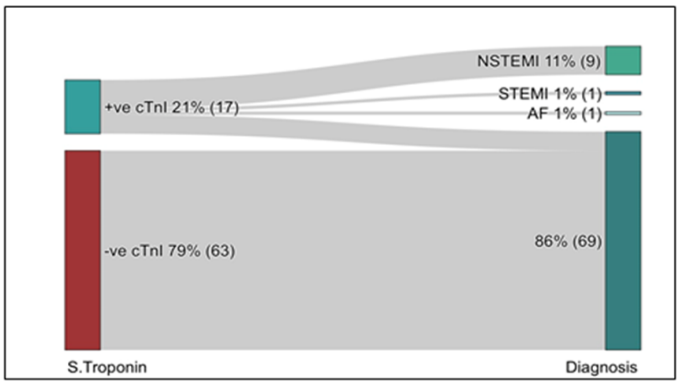

Figure 2: Sankey diagram for positive serum troponin and diagnosis Point biserial correlation (positive correlation) r=0.6 p value =0.001

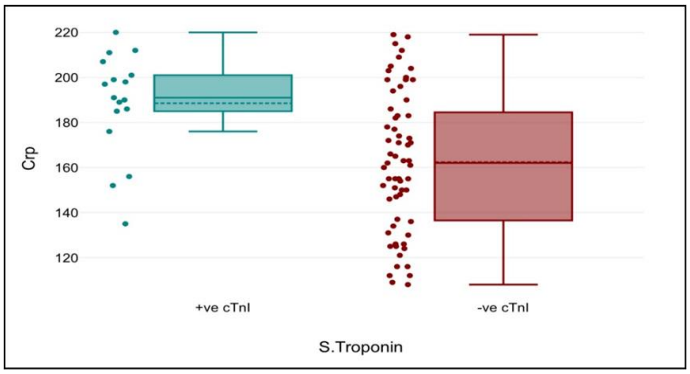

Figure 3: Box plot between CRP and Serum Troponin

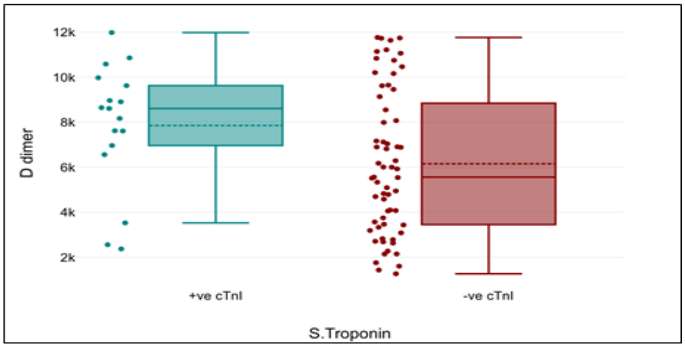

Figure 4: Box plot between D-dimer and Serum Troponin

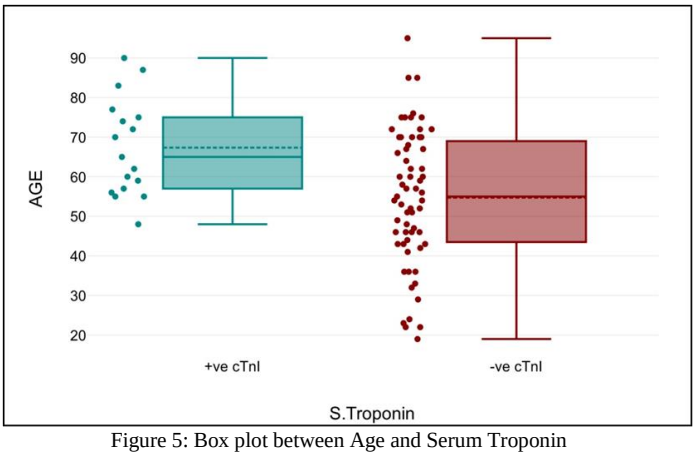

Figure 5: Box plot between Age and Serum Troponin

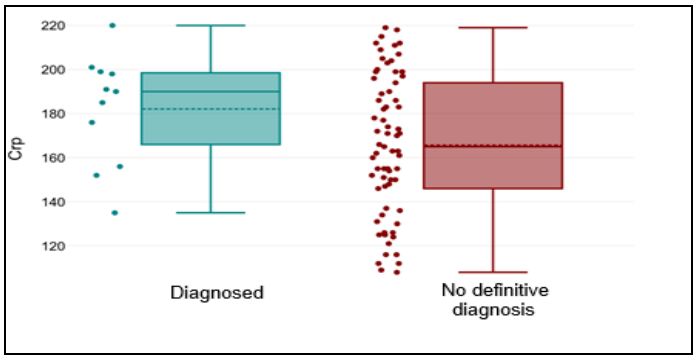

Figure 6: Box plot between CRP and cardiac disease

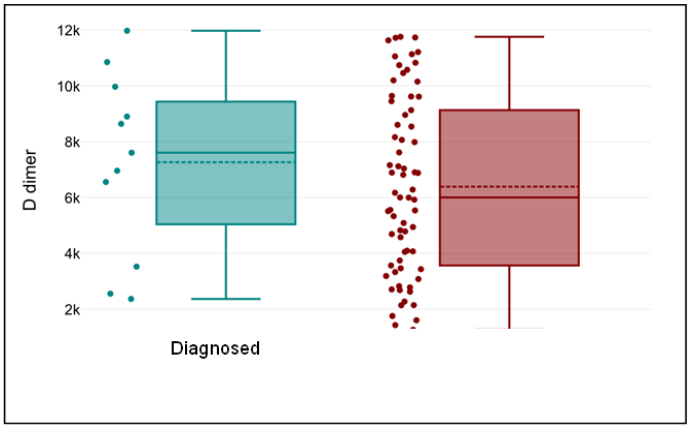

Figure 7: Box plot between D-dimer and cardiac disease

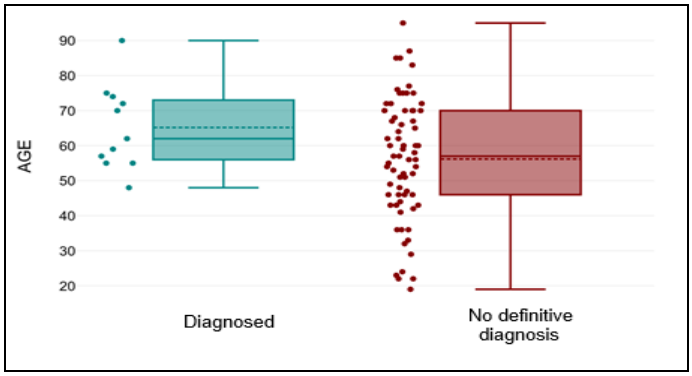

Figure 8: Box plot between Age and cardiac disease

IV. Discussion

Although the majority of research has been on COVID-19 and serum troponin mortality predictions, our work sheds light on novel and clinically plausible risk variables for positive serum troponin and substantial heart illness.

Assessment of these risk variables may aid in better patient triage and prompt treatment with suitable medication to limit disease development by using serum troponin as an intermediate result that could not be performed until deterioration or symptoms may emerge.

According to the results of the present study, the same factors that had a significant impact on serum troponin, had also a higher risk for significant cardiac disease.

Demographically, older age, the present of comorbidities, especially DM, IHD, HF and hypertension significant increase

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positive serum troponin level and significant cardiac disease. These results are in accordance with many previous studies. A research conducted in New York involving 2736 hospitalized patients with COVID-19 revealed that 36 percent exhibited higher hs-cTnI levels (5). Troponin increase was more common in those with established cardiovascular disease or risk factors. At Renmin Hospital in Wuhan, 416 COVID-19 patients were hospitalized, and 19.7% of them were admitted with hs-cTnI levels that were higher than the 99th percentile URL (4). Compared to individuals without myocardial injury, those exhibiting this indication of myocardial injury were older and had a greater prevalence of comorbidities, including chronic heart failure at 14.6% versus 1.5%. Another study discovered that the link between CVD and mortality was significantly influenced by the presence of acute injury as indicated by troponin increase (10). A history of CVD (defined as HTN or cardiomyopathy) was present in 66 (35%) of the 187 individuals with confirmed COVID-19, and troponin levels were raised in 52 (28%) of them. Patients with cardiovascular diseases were more likely to experience troponin increase (55 percent, 36 of 66). Mortality was 69% (25 of 36) among individuals with CVD and high troponin.

The pathophysiology of the association between the severity of COVID-19 and serum troponin, myocardial injury remains insufficiently understood. Hypoxic damage is one of the potential causes of cardiac injury in COVID-19 patients also; stress cardiomyopathy (11, 12); Ischemic damage brought on by small vessel cardiac vasculitis and cardiac microvascular dysfunction (13), immune response within the endothelium in blood vessels (14), or Epicardial coronary artery disease; pulmonary embolism is one of the reasons of right heart strain (acute cor pulmonale). (15-19), ARDS (20), and acute respiratory infection; inflammation of the myocardium (4, 21); and SIRS (cytokine storm) (22-25). In this setting, it is unclear whether or not these variables contribute to myocardial damage and worse cardiovascular outcomes. In a COVID-19 patient, cardiac symptoms and signs might develop as a result of an acute illness process, an abrupt worsening of chronic diseases, or an increase in hemodynamic demands due to underlying heart disease. Hypertension, coronary artery disease, and other cardiovascular diseases are known to increase the likelihood of contracting and the severity of complications from COVID-19 (9).

Possible causes include problems with cardiovascular and pulmonary physiological reserves, a weakened immune system, an overactive inflammatory response, an increased risk of induced dysfunction of endothelial cells, and effects via the angiotensin-converting enzyme 2 receptor (26).

In the present study there was a positive significant correlation between positive serum troponin and significant cardiac disease ($r= 0.6$, $p<0.001$). Consistent with numerous prior investigations, Patients hospitalized with COVID-19 often have myocardial damage, as shown by troponin increase, however the exact origins of this condition are still unclear (3). Myocardial damage may occur for many different reasons in COVID-19 patients, and elevated cardiac troponin levels do not help narrow down the possible causes. However, the exact cause in each patient is often unknown. Myocardial infarction (MI), stress cardiomyopathy, and myocarditis are all clinical disorders associated with injury to the heart muscle.

The results of the present study showed a significant positive correlation between inflammatory markers (CRP and D-dimer) with positive serum troponin and higher risk for significant cardiac disease. The findings align with numerous prior studies; specifically, a study involving 311 hospitalized COVID-19 patients from Wuhan identified independent risk factors for mortality, including hs-cTnI concentration, C-reactive protein concentration, and D-dimer concentration (8). In a cohort of 416 COVID-19 patients from Wuhan, 19.7 percent exhibited hs-cTnI levels exceeding the 99th percentile upper reference limit at admission (4). Patients exhibiting this myocardial damage marker demonstrated more significant laboratory abnormalities, including elevated C-reactive protein levels.

Study Limitation

This study has several limitations

1. The relatively small sample size does not allow the generalization of the results. However, there is a consensus agreement between almost all studies regarding the effect of age, comorbidities and inflammatory markers with the Serum Troponin and significant cardiac disease.
2. Some inflammatory markers such as LDH, Ferritin and neutrophil count, were not included in the study.
3. Serum Troponin was not with the routine daily investigation.
4. Limited access to echocardiography.

V. Conclusions

Older patients with comorbidities like diabetes, IHD are at higher risk for elevated troponin levels and serious cardiac issues in COVID-19. Inflammatory markers correlate significantly.

References

1. Wiersinga WJ, Rhodes A, Cheng AC, Peacock SJ, Prescott HC. Pathophysiology, transmission, diagnosis, and treatment of coronavirus disease 2019 (COVID-19): a review. Jama. 2020;324(8):782-93.
2. Sharma A, Tiwari S, Deb MK, Marty JL. Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2): a global pandemic and treatment strategies. International journal of antimicrobial agents. 2020;56(2):106054.
3. Sandoval Y, Januzzi Jr JL, Jaffe AS. Cardiac troponin for assessment of myocardial injury in COVID-19: JACC review topic of the week. Journal of the American college of cardiology. 2020;76(10):1244-58.
4. Shi S, Qin M, Shen B, Cai Y, Liu T, Yang F, et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. JAMA cardiology. 2020;5(7):802-10.
5. Lala A, Johnson KW, Januzzi JL, Russak AJ, Paranjpe I, Richter F, et al. Prevalence and impact of myocardial injury in patients hospitalized with COVID-19 infection. Journal of the American college of cardiology. 2020;76(5):533-46.
6. Velavan TP, Meyer CG. Mild versus severe COVID-19: Laboratory markers. International Journal of Infectious Diseases. 2020;95:304-7.
7. Sandoval Y, Bielinski SJ, Daniels LB, Blaha MJ, Michos ED, DeFilippis AP, et al. Atherosclerotic cardiovascular disease risk stratification based on measurements of troponin and coronary artery calcium. Journal of the American College of Cardiology. 2020;76(4):357-70.
8. Nie S-F, Yu M, Xie T, Yang F, Wang H-B, Wang Z-H, et al. Cardiac troponin I is an independent predictor for mortality in hospitalized patients with COVID-19. Circulation. 2020;142(6):608-10.

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9. Figliozzi S, Masci PG, Ahmadi N, Tondi L, Koutli E, Aimo A, et al. Predictors of adverse prognosis in COVID-19: a systematic review and meta-analysis. European journal of clinical investigation. 2020;50(10):e13362.
10. Guo T, Fan Y, Chen M, Wu X, Zhang L, He T, et al. Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19). JAMA cardiology. 2020;5(7):811-8.
11. Giustino G, Croft LB, Oates CP, Rahman K, Lerakis S, Reddy VY, et al. Takotsubo cardiomyopathy in COVID-19. Journal of the American College of Cardiology. 2020;76(5):628-9.
12. Tsao CW, Strom JB, Chang JD, Manning WJ. COVID-19–associated stress (Takotsubo) cardiomyopathy. Circulation: Cardiovascular Imaging. 2020;13(7):e011222.
13. Fox SE, Lameira FS, Rinker EB, Vander Heide RS. Cardiac endotheliitis and multisystem inflammatory syndrome after COVID-19. Annals of internal medicine. 2020;173(12):1025-7.
14. Varga Z, Flammer AJ, Steiger P, Haberecker M, Andermatt R, Zinkernagel AS, et al. Endothelial cell infection and endotheliitis in COVID-19. The Lancet. 2020;395(10234):1417-8.
15. Creel-Bulos C, Hockstein M, Amin N, Melhem S, Truong A, Sharifpour M. Acute cor pulmonale in critically ill patients with Covid-19. New England Journal of Medicine. 2020;382(21):e70.
16. Danzi GB, Loffi M, Galeazzi G, Gherbesi E. Acute pulmonary embolism and COVID-19 pneumonia: a random association? European heart journal. 2020;41(19):1858-.
17. Poissy J, Goutay J, Caplan M, Parmentier E, Duburcq T, Lassalle F, et al. Pulmonary embolism in patients with COVID-19: awareness of an increased prevalence. Circulation. 2020;142(2):184-6.
18. Klok FA, Kruip M, Van der Meer NJM, Arbous MS, Gommers D, Kant KM, et al. Incidence of thrombotic complications in critically ill ICU patients with COVID-19. Thrombosis research. 2020;191:145-7.
19. Wichmann D, Sperhake J-P, Lütgehetmann M, Steurer S, Edler C, Heinemann A, et al. Autopsy findings and venous thromboembolism in patients with COVID-19: a prospective cohort study. Annals of internal medicine. 2020;173(4):268-77.
20. Huette P, Beyls C, Guilbart M, Haye G, Najid F-Z, Mestan B, et al. Acute cor pulmonale in COVID-19-related ARDS: improvement with almitrine infusion. Case Reports. 2020;2(9):1311-4.
21. Caforio AL, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB, et al. European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2013;34(33):2636-48.
22. Zheng Y-Y, Ma Y-T, Zhang J-Y, Xie X. COVID-19 and the cardiovascular system. Nature reviews cardiology. 2020;17(5):259-60.
23. Tersalvi G, Vicenzi M, Calabretta D, Biasco L, Pedrazzini G, Winterton D. Elevated troponin in patients with coronavirus disease 2019: possible mechanisms. Journal of cardiac failure. 2020;26(6):470-5.
24. Libby P, Lüscher T. COVID-19 is, in the end, an endothelial disease. European heart journal. 2020;41(32):3038-44.
25. Akhmerov A, Marbán E. COVID-19 and the heart. Circulation research. 2020;126(10):1443-55.
26. Chatterjee NA, Cheng RK. Cardiovascular disease and COVID-19: implications for prevention, surveillance and treatment. BMJ Publishing Group Ltd and British Cardiovascular Society; 2020. p. 1119-21.

Mosab G. T. Almilhem, Jaafar Hayder Abdulateef Al-Yousuf, Ali Zuhair Abdulhussein Al-Ghazal, and Kamil Hayder Mohammed Alkendi, "Correlation Between Severe Covid-19 and Serum Troponin Regarding Significant Cardiac Disease," *International Research Journal of Pharmacy and Medical Sciences (IRJPMS)*, Volume 7, Issue 6, pp. 41-46, 2024.