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Original Research Article

Prevalence of Cardiac Manifestations of COVID-19 at a Tertiary Centre in Western Rajasthan

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Abstract:

Introduction: The coronavirus disease 2019 (COVID-19) pandemic has brought unprecedented challenges to global healthcare systems, with the respiratory system being predominantly affected. However, emerging evidence suggests that COVID-19 can also lead to various cardiovascular complications. This retrospective study aims to investigate the prevalence of cardiac manifestations in COVID-19 patients and its association with disease severity.

Methods: We conducted a retrospective analysis of medical records from 350 patients admitted with COVID-19 infection at a tertiary care hospital between June 2020 and July 2022. Patients were categorized into severe and non-severe groups based on clinical, lab parameters. Various cardiac manifestations, including heart failure, arrhythmias, myocarditis, acute coronary syndrome (ACS), and pulmonary embolism, were evaluated.

Results: Our findings revealed a clear correlation between the severity of COVID-19 and the prevalence of cardiovascular complications. Heart failure was observed in 7.4% of patients, with a significantly higher incidence rate in the severe COVID-19 group (10.9% versus 2.7% in non-severe group, $p < 0.01$). Similarly, the overall incidence of cardiac arrhythmias was 7.1%, with a higher prevalence in the severe COVID-19 group (10.5% versus 2.7% in non-severe group, $p < 0.01$). Myocarditis and ACS were reported in 6.6% and 10.3% of patients, respectively, with significantly higher rates in severe COVID-19 cases ($p < 0.01$). Additionally, pulmonary embolism was identified in 1.4% of patients, predominantly in the severe COVID-19 group ($p < 0.01$).

Conclusion: Our study provides valuable insights into the diverse cardiac complications associated with COVID-19. The findings underscore the importance of vigilance among clinicians to recognize and manage these manifestations, particularly in severe cases. By understanding the impact of COVID-19 on cardiovascular health, healthcare providers can take proactive measures to optimize patient outcomes. Further research is needed to explore the underlying mechanisms and develop targeted interventions for these cardiac complications.

Keywords: COVID-19, cardiovascular complications, heart failure, arrhythmias, myocarditis, acute coronary syndrome, pulmonary embolism.

Introduction

The COVID-19 pandemic caused by the novel coronavirus, SARS-CoV-2, continues to pose significant challenges to global health. It is well-established that the primary mode of virus transmission occurs through respiratory droplets, with infected individuals shedding virus particles in their respiratory secretions. Consequently, direct contact with the mucous membranes of the nose, eyes, or mouth of a contagious individual plays a critical role in disease spread. [1] The virus's outer surface is characterized by distinctive S-spikes, enabling its binding to angiotensin-converting enzyme 2 (ACE2) receptors as an entry point into host cells. These ACE2 receptors are expressed on various body cells, including type 1 and type 2 pneumocytes and the outer surface of endothelial cells. Interestingly, ACE2 receptor activation inversely regulates the renin-angiotensin system, potentially influencing disease severity.[2]

While COVID-19 predominantly affects the respiratory system, it has become increasingly evident that the disease can also lead to cardiovascular manifestations.[3]

Although the exact underlying mechanisms of these cardiovascular complications remain incompletely understood, several theories have been proposed to shed light on this phenomenon.

One prominent theory suggests that SARS-CoV-2 primarily gains entry into host cells through ACE2 receptors present on both cardiac and pulmonary cells. Consequently, viral invasion directly damages tissues, while the release of cytokines and inflammatory markers contributes to further tissue destruction. The resulting exaggerated inflammatory response, often referred to as a "cytokine storm," plays a pivotal role in the multiorgan involvement seen in severe cases of COVID-19.[4]

Notably, COVID-19 induces the release of various cytokines and chemokines, leading to vascular inflammation, plaque instability, and myocardial inflammation, culminating in a spectrum of cardiovascular complications.

Recent research has also uncovered evidence of direct viral injury to cardiac myocytes, potentially initiating inflammation and cardiac damage, further exacerbating the development of cardiovascular complications.[5]

Given the evolving nature of the pandemic and the pressing need to understand the disease's diverse manifestations, this study aims to delve deeper into the mechanisms underpinning COVID-19-related cardiovascular complications. By elucidating the role of ACE2 receptor-mediated pathways and the subsequent inflammatory response, we aim to contribute valuable insights to the understanding and management of COVID-19-induced cardiovascular pathology. This research could ultimately lead to improved patient outcomes and guide the development of targeted therapeutic interventions.

Aims and Objectives

The primary aim of this study is to determine the prevalence of cardiovascular complications in COVID-19 patients admitted to MDM Hospital between June 2022 and July 2023. The specific objectives are as follows:

1. To assess the incidence of cardiovascular complications, including myocardial injury, myocardial infarction (STEMI and NSTEMI), and myocarditis, arrhythmias in COVID-19 patients.
2. To investigate the association between disease severity and the occurrence of cardiovascular complications.

Materials and Methods

This retrospective study was conducted at MDM Hospital, which is affiliated with Dr. S. N. Medical College, Jodhpur. A total of 350 patients diagnosed with COVID-19 and admitted to the hospital between June 2022 and July 2023 were enrolled in the study. Patients with positive RT-PCR COVID reports were included, while those with pre-existing cardiovascular disorders were excluded.

Data pertaining to various parameters were collected from the hospital records of the included patients. The parameters included:

1. Clinical history related to signs and symptoms, exposure history, medical history, and any previously known chronic diseases.
2. Laboratory data, including complete blood picture, total bilirubin, SGOT, SGP, blood urea, serum creatinine, serum ferritin, C-reactive protein, troponinI,NT-pro-BNP, serum creatinine phosphokinase-MB, D-dimer, and prothrombin time.
3. Radiological investigations, such as 2D transthoracic echocardiography, HRCT, and CT pulmonary angiography.

The patients were categorized into severe and non-severe disease groups based on the following criteria:

- Severe disease: Spo2 ≤ 94% and PaO2/FiO2 < 300.
- Non-severe disease: Spo2 > 94% and PaO2/FiO2 > 300.

Cardiovascular complications were classified as follows:

1. STEMI (ST-segment elevation myocardial infarction) and NSTEMI (non-ST-segment elevation myocardial infarction) based on the Universal definition of myocardial injury and myocardial infarction.
2. Myocarditis in patients with positive cardiac markers and echocardiographic features of global LV hypokinesia and Heart failure on basis of clinical finding as per record and NT-proBNP level.
3. Arrhythmias on the basis of ECG finding.
4. Pulmonary embolism and DVT on basis of CT pulmonary angiography , Doppler scan respectively.

The treatment regimens, including antiviral therapy, antibiotics, corticosteroid therapy, and oxygen support, administered to all COVID-19 patients during hospital admission, were also evaluated.

By comprehensively examining these parameters, we aim to provide valuable insights into the prevalence and characterization of cardiovascular complications in COVID-19 patients, thereby contributing to improved management and care for this vulnerable patient population.

Inclusion Criteria

Admitted patients with a positive RT-PCR COVID report, confirming the presence of SARS-CoV-2 infection.

Exclusion Criteria

Patients with pre-existing cardiovascular disorders, such as a history of heart disease, coronary artery disease, heart failure, arrhythmias, or any other significant cardiovascular condition, were excluded from the study.

Results

Table 1: Demographic features, Comorbidities and Laboratory parameters in Patients with severe and non- severe COVID 19

Total patients=350	Severe COVID(n=210)	Non-severe COVID(n=140)	P value
Age	53±17	51±16	0.360
Gender (male/female ratio)	114/96	90/50	0.063
Co-morbidity			
DM	113	24	<.01
HTN	79	12	<.01
CKD	21	0	<.01
COPD	27	4	<.01
PAD	4	1	<.01
DVT	4	1	<.01
Lab Parameters			
TLC	10027±4285	6759±2987	<.001
Urea	75±51	50±18	<.001
Creatinine	1.6±.9	1.1±.4	<.001
Sodium	140±10	142±10	.119
Potassium	4.3±.9	4.2±.7	.922
SGOT	46±28	45±19	.042
SGPT	45±25	46±16	.006
TC	214±24	214±24	.625
LDL	122.1±42.1	121.1±42.1	.898
HDL	47±12	49±12	.070
TG	137.1±41.1	142.2±46.2	.466
IL-6	13.7±11.4	6.1±5.3	<.001
D-DIMER	791±699	473±423	<.001
Cardiac Troponin	0.803±1.375	0.133±.593	<.001
NT-pro-BNP	10876 ±347	346±34.8	<.001

Table 1 presents the demographic features, comorbidities, and laboratory parameters of the 350 patients with COVID-19, categorized into severe (n=210) and non-severe (n=140) groups.

Demographic Features and Comorbidities

The mean age of patients in the severe COVID-19 group was 53 years ±17, while in the non-severe group, the mean age was 51 years ±16. The difference in age between the two groups was not statistically significant ($p = 0.360$).

In terms of gender distribution, the severe COVID-19 group had 114 male and 96 female patients, whereas the non-severe group had 90 male and 50 female patients. The difference in gender distribution between the groups with no statistical significance ($p = 0.063$).

The prevalence of comorbidities was significantly higher in the severe COVID-19 group compared to the non-severe group. Specifically, the presence of diabetes mellitus (DM), hypertension (HTN), chronic kidney disease (CKD), chronic obstructive pulmonary disease (COPD), peripheral arterial disease (PAD), and deep vein thrombosis (DVT) was notably higher in the severe group ($p < 0.01$ for all comorbidities).

Laboratory Parameters

Several laboratory parameters were analyzed, and the results showed statistically significant differences between the severe and non-severe COVID-19 groups:

Total leukocyte count (TLC) was significantly higher in the severe group (10,027 ± 4,285) compared to the non-severe group (6,759 ± 2,987; $p < 0.001$).

Serum urea levels were significantly elevated in the severe group (75 ± 51) compared to the non-severe group (50 ± 18; $p < 0.001$).

Serum creatinine levels were also significantly higher in the severe group (1.6 ± 0.9) compared to the non-severe group (1.1 ± 0.4; $p < 0.001$).

Serum sodium and potassium levels did not show significant differences between the two groups (p = 0.119 and p = 0.922, respectively).

SGOT (Serum Glutamic Oxaloacetic Transaminase) levels were slightly higher in the severe group (46 ± 28) compared to the non-severe group (45 ± 19; p = 0.042).

SGPT (Serum Glutamic Pyruvic Transaminase) levels were slightly higher in the non-severe group (46 ± 16) compared to the severe group (45 ± 25; p = 0.006).

Total cholesterol (TC), LDL (low-density lipoprotein), HDL (high-density lipoprotein), and triglyceride (TG) levels did not show statistically significant differences between the two groups (p > 0.05 for all).

Interleukin-6 (IL-6) levels were significantly higher in the severe group (13.7 ± 11.4) compared to the non-severe group (6.1 ± 5.3; p < 0.001).

D-dimer levels were significantly elevated in the severe group (mean ± standard deviation: 791 ± 699) compared to the non-severe group (473 ± 423; p < 0.001).

Cardiac troponin levels were significantly higher in the severe group (0.803 ± 1.375) compared to the non-severe group (0.133 ± 0.593; p < 0.001).

NT-pro-BNP levels were also higher in severe COVID group (10876±347) as compared to non-severe COVID group (346±34.8; p<0.001) due to high proportion of heart failure patient in severe group.

These findings suggest that severe COVID-19 is associated with older age, a higher prevalence of comorbidities, and significantly altered laboratory parameters, including increased TLC, urea, creatinine, SGOT, SGPT, IL-6, D-dimer, and cardiac troponin levels.

These results provide valuable insights into the clinical characteristics and potential risk factors for severe cardiovascular complications in COVID-19 patients.

Table 2: Comparison of Cardiac Parameters in Patients of Severe and non-severe COVID 19

	Severe COVID	Non-Severe Covid	P value
ECG Findings			
Sinus Tachycardia (ST)	45	5	<0.01
MI Findings (STEMI, NSTEMI)	29	3	
Atrial Fibrillation	11	2	
Wide QRS Morphology (SVT, VT)	7	0	
Sinus Bradycardia (BC)	27	2	
1° AV Block	8	1	
Mobitz Type 1	10	0	
Mobitz Type 2	2	0	
CHB	0	0	
S1Q3T3	5	0	
2D Echo Findings (mean SD)			
LVEF	41.6(8.8)	59.8(13.3)	<0.001
E/A	0.9(0.2)	1.2(0.4)	0.06
E/e’	9.9(4.0)	9.8(3.4)	0.94
RA/RV Dilated	15	1	<0.01
TAPSE<17	13	0	<0.01

Table 2 presents a comparison of cardiac parameters between patients with severe and non-severe COVID-19.

ECG Findings

- Sinus Tachycardia (ST) was significantly more prevalent in the severe COVID group, observed in 45 (21.4%) cases compared to only 5 (3.5%) cases in the non-severe COVID group.
- Myocardial Infarction (MI) findings, including STEMI and NSTEMI, were more frequent in the severe COVID group, with 29 (13.8%) cases, compared to 3 (21.4%) cases in the non-severe COVID group.
- Atrial Fibrillation was observed in 11 (5.2%) cases in the severe COVID group, whereas it was seen in 2 (1.4%) cases in the non-severe COVID group.
- Wide QRS Morphology was present in 7 (3.34%) cases in the severe COVID group and was absent in the non-severe COVID group.
- Sinus Bradycardia was noted in 29 (13.8%) cases in the severe COVID group and only 2 (1.42%) cases in the non-severe COVID group.

- First-degree AV Block was seen in 8 (3.8%) cases in the severe COVID group and 1 (0.7%) case in the non-severe COVID group.
- Mobitz Type 1 AV Block was observed in 10 (4.7%) cases in the severe COVID group, while it was absent in the non-severe COVID group.
- Mobitz Type 2 AV Block was noted in 2 (0.9%) cases in the severe COVID group and was not seen in the non-severe COVID group.
- Complete Heart Block (CHB) was not observed in either group.
- S1Q3T3 pattern, suggestive of acute cor pulmonale, was present in 5 (2.3%) cases of severe COVID, while it was absent in the non-severe COVID group.

2D Echo Findings

- Mean Left Ventricular Ejection Fraction (LVEF) was significantly lower in the severe COVID group, measuring 41.6% with a standard deviation of 8.8, compared to 59.8% with a standard deviation of 13.3 in the non-severe COVID group ($p < 0.001$).
- The E/A ratio (early to late ventricular filling ratio) did not show a statistically significant difference between the two groups ($p = 0.06$).
- The E/e' ratio (early transmitral velocity to early diastolic mitral annular velocity ratio) was comparable between the severe COVID group (mean ± SD: 9.9 ± 4.0) and the non-severe COVID group (mean ± SD: 9.8 ± 3.4; $p = 0.94$).
- Right atrial and right ventricular dilation was more prevalent in the severe COVID group, with 15 (7.14%) cases, whereas it was observed in only 1 (0.7%) case in the non-severe COVID group ($p < 0.01$).
- Tricuspid Annular Plane Systolic Excursion (TAPSE) < 17mm was noted in 13 (6.1%) cases of the severe COVID group, while it was absent in the non-severe COVID group ($p < 0.01$).

Overall, the occurrence of various cardiac events based on ECG findings, as well as the 2D Echo parameters, significantly differed between the severe and non-severe COVID-19 groups. Patients with severe COVID-19 demonstrated a higher incidence of abnormal ECG findings, reduced LVEF, and a higher prevalence of right atrial and ventricular dilation, indicating a higher burden of cardiac involvement in severe cases of COVID-19. These findings underscore the importance of monitoring cardiac parameters and assessing cardiovascular health in patients with severe COVID-19 to provide timely and appropriate management.

Table 3: Cardiac Manifestations of COVID 19

Cardiac Manifestation	Severe COVID(n=210)	Non-severe COVID(n=140)	P value
Pulmonary Embolism	5	0	<0.01
Myocardial Infarction (STEMI/NSTEMI)	33	3	
Arrhythmias	22	3	
Myocarditis	21	2	
Heart Failure (HF)	23	3	
None	129	132	
Total	210	140	

Table 3 presents the cardiac manifestations observed in patients with severe and non-severe COVID-19.

Cardiac Manifestations:

- Pulmonary Embolism: The incidence of pulmonary embolism was significantly higher in the severe COVID-19 group, with 5 (2.4%) cases, compared to the non-severe COVID-19 group, where no cases were reported ($p < 0.01$).
- Myocardial Infarction (STEMI/NSTEMI): Myocardial infarction, including both ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI), was significantly more frequent in the severe COVID-19 group, with 33 (15.7%) cases, compared to 3 (2.7%) cases in the non-severe COVID-19 group.
- Arrhythmias: The incidence of arrhythmias was significantly higher in the severe COVID-19 group, with 22 (10.5%) cases, compared to 3 (2.7%) cases in the non-severe COVID-19 group.
- Myocarditis: Myocarditis was more prevalent in the severe COVID-19 group, with 21 (10%) cases, compared to 2 (1.8%) cases in the non-severe COVID-19 group, and this difference was found to be significant.
- Heart Failure (HF): Heart failure was observed in 23 (11%) cases in the severe COVID-19 group and in 3 (2.7%) cases in the non-severe COVID-19 group. All heart failure patients have raised NT-pro-BNP levels which is

diagnostic and prognostic marker of heart failure.

- None: A total of 129 patients in the severe COVID-19 group and 132 patients in the non-severe COVID-19 group did not manifest any cardiac complications.

The data highlights that severe COVID-19 is associated with a higher incidence of significant cardiac manifestations, including pulmonary embolism, myocardial infarction (both STEMI and NSTEMI), arrhythmias, myocarditis, and heart failure. In contrast, a substantial number of patients in both groups did not exhibit any cardiac complications.

These findings underscore the importance of closely monitoring COVID-19 patients, particularly those with severe disease, for potential cardiovascular complications, which may warrant specialized management and interventions to improve patient outcomes.

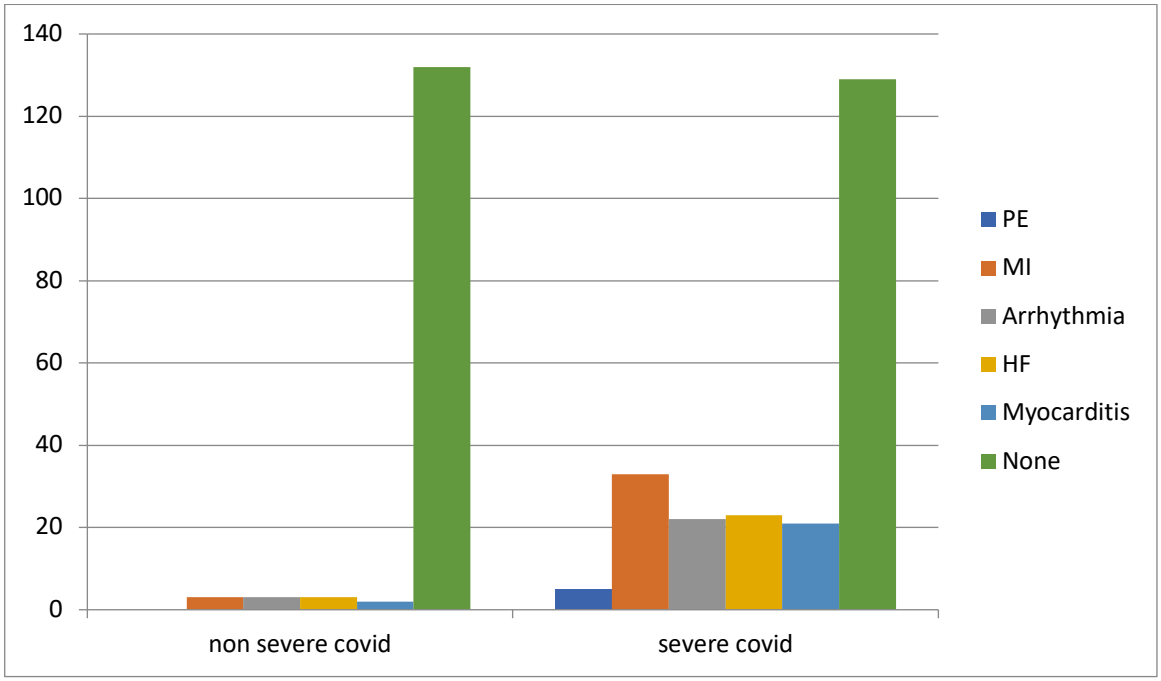

Figure 1:

Discussion

There is paucity of sufficient literature regarding the range of cardiac manifestations and their impact on outcomes in COVID-19. In this retrospective study, we examined the medical records of 350 patients admitted with COVID-19 infection between June 2020 and July 2022, focusing on the occurrence of cardiac manifestations, including non-specific ECG changes and myocarditis.

Our observations revealed that the most common ECG finding in these patients was sinus tachycardia (N=50), although it was non-specific in nature. Additionally, thirteen patients exhibited atrial fibrillation, while twenty-nine patients presented with sinus bradycardia, and seven patients showed a wide QRS tachycardia.

It is important to note that troponin levels can be elevated in various conditions other than myocardial injury, particularly in critically ill patients. This may have led to a potential overestimation of myocardial injury in our cohort, especially in patients with renal failure. However, we only considered cases as myocarditis when myocardial injury was accompanied by global myocardial dysfunction. Therefore, it is unlikely that the prevalence of myocarditis in our study was affected by a potential overestimation of myocardial injury.

By conducting this study, we aimed to contribute valuable insights into the cardiac complications associated with COVID-19. However, the limited existing literature in this area emphasizes the significance of further research to better understand the spectrum of cardiac manifestations and their implications on patient outcomes in the context of COVID-19.

Cardiac Manifestations of COVID-19

Heart Failure (HF)

In our study, the overall incidence of heart failure was 7.4%, with a significantly higher rate observed among severe COVID-19 cases compared to non-severe cases (10.9% in severe COVID-19 category versus 2.7% in non-severe COVID-19 category, p-value <0.01). Numerous studies have also highlighted heart failure as a significant manifestation of COVID-19. For instance, Chen et al. (6) in a

study conducted in China reported a prevalence of heart failure as 24% in COVID-19 infection, which was higher than what we observed.

The occurrence of heart failure in COVID-19 patients is believed to be associated with a severe immune system overreaction, leading to a cytokine storm. The downregulation of angiotensin-converting enzyme-2 by the COVID-19 virus results in increased levels of Angiotensin II, which in turn leads to heightened inflammation, hypertension, and thrombosis.

Arrhythmias

In our study, the overall incidence of cardiac arrhythmias was 7.1%, with a significantly higher rate observed in severe COVID-19 cases compared to non-severe cases (10.5% in severe COVID-19 category versus 2.7% in non-severe COVID-19 category, p-value <0.01). Wang et al. [18], in a study conducted in China, reported the prevalence of cardiac arrhythmias as 11.6% in COVID-19 infection, which was higher than our findings.

COVID-19 patients are more susceptible to arrhythmias due to underlying comorbidities, multiple drugs used in treatment, and the severity of the disease itself. In a study by Liu et al. [7], among 137 patients, 7.3% reported palpitations as one of their symptoms. Several other studies have also shown a higher prevalence of cardiac arrhythmias in critically ill COVID-19 patients compared to non-critically ill patients. Tachyarrhythmias, such as atrial fibrillation, atrial flutter, and ventricular tachycardia/ventricular fibrillation, were commonly observed in COVID-19 patients, particularly in severe cases. The increased prevalence of arrhythmias in severe COVID-19 cases could be explained by the heightened systemic inflammatory response, which makes patients more prone to cardiac arrhythmias and electrolyte abnormalities.

Myocarditis

In our study, the overall incidence of myocarditis was 6.6% (23/350), with a significantly higher rate observed in severe COVID-19 cases compared to non-severe cases (10% in severe COVID-19 category versus 1.8% in non-severe COVID-19 category, p-value <0.01). Deng et al. [8], in a study conducted in China, found the prevalence of myocarditis as 12.5% in COVID-19 infection, which was higher than our findings.

Myocarditis is characterized by myocardial injury or inflammation without an ischemic cause and can be due to various factors, including viral infection. The mechanism of viral myocarditis involves stimulation of interleukin-6 (IL-6), leading to a cytokine storm and direct myocardial injury. Human coronaviruses, including Sars-Cov-1, have been isolated in mammalian cardiac tissue, indicating the possibility of direct toxic effects on the heart. The presence of ACE-2 receptors on the heart, upregulated in heart failure, allows Sars-Cov-2 to enter cardiac cells.

ACS (Acute Coronary Syndrome)

In our study, the overall incidence of ACS was 10.3% (36/350), with a significantly higher rate observed in severe COVID-19 cases compared to non-severe cases (15.7% in severe COVID-19 category versus 2.7% in non-severe COVID-19 category, p-value <0.01). Huang et al. [9], in a study conducted in China, found the incidence of ACS as 12% in COVID-19 infection, which was higher than our findings.

ACS is a well-recognized complication of COVID-19 and may be related to the hypercoagulable state induced by the virus, leading to thrombosis of coronary arteries. Prothrombotic factors like prothrombin time and D-dimer values were significantly elevated in severely ill COVID-19 patients, particularly those requiring ICU care, and were associated with lower left ventricular ejection fraction.

Pulmonary Embolism

In our study, the overall incidence of pulmonary embolism was 1.4% (5/350), with a significantly higher rate observed in severe COVID-19 cases compared to non-severe cases (2.4% in severe COVID-19 category versus 0% in non-severe COVID-19 category, p-value <0.01). Rupak Desai et al. [10], in a meta-analysis of 9 studies, reported a pooled prevalence of PE as 15.8% among 3066 COVID-19 patients, which was much higher than our findings.

The incidence rate of VTE (venous thromboembolism) and arterial thrombosis, along with related thromboembolic complications, is considerably higher in COVID-19 patients. The mechanisms responsible for this phenomenon include the cytokine storm induced by interleukin-1, interleukin-6, tumor necrosis factor-alpha, and other inflammatory markers, leading to widespread thrombosis similar to what is seen in pulmonary vessels in ARDS. The affinity of the COVID-19 virus for ACE-2 receptors in alveolar epithelial cells and endothelial cells of extrapulmonary tissues also contributes to thrombosis by causing microvascular injury and multiorgan failure. Furthermore, lupus anticoagulant antibodies can lead to thrombosis by the "two-hit" thrombosis hypothesis.

Our study provides valuable insights into the cardiac manifestations of COVID-19. Heart failure, arrhythmias, myocarditis, ACS, and pulmonary embolism are significant complications associated with COVID-19, with higher incidence rates observed in severe cases. Understanding these manifestations can aid in the management and treatment of COVID-19 patients and may have implications

for future research and improvements in cardiovascular health during the pandemic.

Conclusion

In conclusion, our study sheds light on the various cardiac complications seen in COVID-19 patients. We have identified a significant association between the severity of COVID-19 and the prevalence of cardiovascular diseases. By uncovering these cardiac manifestations, our study aims to increase awareness among clinicians about the potential cardiovascular impact of COVID-19. This understanding will assist in better managing and treating COVID-19 patients and may have implications for future cardiovascular health.

In summary, our findings emphasize the importance of considering the cardiovascular implications of COVID-19 in clinical practice. By doing so, we can be better prepared to tackle the challenges posed by this viral infection and protect the cardiovascular health of affected individuals. Through continued research and collaboration, we can gain a more comprehensive understanding of COVID-19's impact on the cardiovascular system and develop strategies to mitigate its long-term effects on cardiovascular health.

Limitations of the study

1. Retrospective Design: The study's retrospective nature may introduce selection bias and limit the control over the investigations performed on the patients.
2. Single-Center Study: Being a single-center study, the findings may not fully represent the broader population, potentially limiting the generalizability of the results.
3. Lack of Examination of Cardiovascular Comorbidity: The study did not explore the significant association of cardiovascular comorbidity with COVID-19 severity and mortality, which could have provided further insights into the interplay between pre-existing cardiovascular conditions and COVID-19 outcomes.
4. Possible Confounding Effects: As with any observational study, confounding factors that were not accounted for might influence the observed associations between COVID-19 severity and cardiac manifestations.

These limitations should be considered when interpreting the results of the study and highlight the need for further research, including larger multicenter studies and investigations into the impact of pre-existing cardiovascular conditions on COVID-19 outcomes. Despite these limitations, the study contributes valuable information to our understanding of cardiac complications in COVID-19, emphasizing the importance of cardiovascular health management during the pandemic.

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