Journal of Cardiovascular Disease Research
ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 08, 2024

ACUTE MYOCARDIAL INFARCTION IN YOUNG ADULTS IN POST COVID ERA IN SSIMS, BHILAI

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Abstract

Introduction: The corona virus disease 2019 (COVID-19) pandemic is caused by severe acute respiratory syndrome corona virus 2 (SARS-CoV-2) and is associated with various disease manifestations across multiple organ systems, presenting with a spectrum of disease severity that is only partially explained by age, sex and comorbidities. Of note, the manifestations of acute cardiovascular injury associated with SARS-CoV-2 infection are diverse, including acute myocardial infarction, myocarditis, stress cardiomyopathy, pericarditis, arrhythmias, multisystem inflammatory syndrome in both adults (MIS-A) and children (MIS-C), stroke, macrothrombotic disease including arterial and venous thromboembolism, microthrombotic disease and bleeding diathesis.

Materials and Methods: Total 750 acute myocardial infarction patients were identified; The present study included 150 young patients with acute myocardial infarction in post COVID-19 era in Shri Shankaracharya Institute of Medical Sciences (SSIMS), Bhilai district Durg, Chhattisgarh. All patients treated with a primary percutaneous coronary intervention (PPCI) during the period from January 2021 to June 2023. The study was approved by the local ethical committee. As our study is retrospective and data were collected after patients discharged from the hospital, there is no consent for participation in our study.

Results: Total 750 acute myocardial infarction patients were identified; The present study included 150 young patients with acute myocardial infarction in post COVID-19 era. All patients treated with a primary percutaneous coronary intervention (PPCI) during the period from January 2021 to June 2023. Male patients represented 86.66% (n=130), while female patients represented 13.13% (n = 20). The mean age of the patients were 35.75 years ± 8.76 with range 20-45 years. Regarding traditional risk factors the incidence of diabetes, hypertension, current smoking status, dyslipidemia and obesity were 16.16% (n=25), 8.66% (n=13), 2% (n =3), 9.33% (n = 14), 0.66 (n=1) respectively. The mean heart rate (beat/min), oxygen saturation%, Serum creatinine, CRP, SBP and D-Dimer of our patients were 80, 92.76, 1.3, 86.51, 118, and 650.12 respectively.

Conclusion: After covid era (after 2021) young myocardial infarction patients have increased. Especially young patients who are smoker and tobacco chewers, these were not confirmed cases of Covid. A non-obstructive CAD was found in about one third of patients; on the other hand, patients who had a total occlusion of their culprit artery, the thrombus burden was high. Identification of the underlying mechanism responsible for the high thrombus burden in these patients is important as it may result in changes in their primary management strategy, either primary PCI, fibrinolytic therapy, or a pharmacoinvasive strategy. Furthermore, adjunctive anticoagulation and antiplatelet therapy may need to be revised.

Key Words: corona virus disease, myocardial infarction, Serum creatinine, CRP, SBP and D-Dimer.

INTRODUCTION

The corona virus disease 2019 (COVID-19) pandemic is caused by severe acute respiratory syndrome corona virus 2 (SARS-CoV-2) and is associated with various disease manifestations across multiple organ systems, presenting with a spectrum of disease severity that is only partially explained by age, sex and comorbidities.[1] Of note, the manifestations of acute cardiovascular injury associated with SARS-CoV-2 infection are diverse, including acute myocardial infarction, myocarditis, stress cardiomyopathy, pericarditis, arrhythmias, multisystem inflammatory syndrome in both adults (MIS-A) and children (MIS-C), stroke, macrothrombotic disease including arterial and venous thromboembolism, microthrombotic disease and bleeding diathesis. The pathogenic mechanisms underlying these clinical manifestations are not well understood and are likely to be multifactorial, including primary causes such as elevated local (endotheliitis) and systemic (cytokine storm) inflammation, resulting in coagulopathy, myocardial infarction, stroke, MIS-A, MIS-C and arrhythmias, direct viral cytopathic effects possibly resulting in myocarditis, and autoantibodies.[2]

Acute coronavirus disease 2019 (COVID-19) can lead to various cardiovascular complications, including arrhythmia, myocardial infarction, deep-vein thrombosis and pulmonary embolism, left and/or right ventricular systolic dysfunction, pericarditis and stroke. These complications can have profound long-term implications for the health of survivors of COVID-19.[3]

Many studies have documented substantial increases in serum troponin levels as a manifestation of acute myocardial injury and, in general, these biomarkers have been shown to be correlated with the severity of acute illness as well as SARS-CoV-2 viraemia.[4] However, among these patients, the degree of myocardial injury observed is inconsistent and the mechanisms are often unclear; whether the degree of troponin level elevation is simply reflective of more severe systemic illness or whether it is a causal factor of worsened prognosis has not yet been determined.

In the current study, we aim to evaluate the angiographic and clinical characteristics of COVID-19 patients presented by myocardial infarction (MI).

MATERIALS AND METHODS

Total 750 acute myocardial infarction patients were identified; The present study included 150 young patients with acute myocardial infarction in post COVID-19 era in Shri Shankaracharya Institute of Medical Sciences (SSIMS), Bhilai district Durg, Chhattisgarh. All patients treated with a primary percutaneous coronary intervention (PPCI) during the period from January 2021 to June 2023.

The study was approved by the local ethical committee. As our study is retrospective and data were collected after patients discharged from the hospital, there is no consent for participation in our study.

Confirmed acute STEMI was defined based on the presence of typical anginal pain more than 20 min, associated with new left bundle branch block or new ST-elevation at the J-point in two contiguous leads with the cut-point: > 1 mm in all leads other than leads V2–V3 where the following cut-points apply: > 2 mm in men > 40 years, > 2.5 mm in men <40 years, or > 1.5 mm in women regardless of age. The diagnosis was confirmed by the elevation in troponin levels.

Patients who have symptoms suspicious for COVID-19, e.g., progressively worsening shortness of breath, cough, fever, body aches, and unexplained hypoxemia were referred for chest CT without contrast. Laboratory tests (complete blood count, C-reactive protein (CRP), and serum ferritin) were done. COVID-19 was confirmed with reverse transcription-polymerase chain reaction assays.

Angiographic procedure

Coronary angiography and percutaneous coronary intervention were done through the femoral or radial approach. All patients received the following regimen: (1)ticagrelor 180 mg initial dose followed by a maintenance dose of 90 mg twice daily or clopidogrel 600 mg loading dose orally followed by a maintenance dose of 75 mg/day if ticagrelor is contraindicated, (2) aspirin 300 mg followed by 75–100 mg/day, and (3) during the procedure patients received unfractionated heparin (100 IU/kg), the dose was reduced to (70 IU/kg) in case of administration of glycoprotein IIb/IIIa inhibitor (eptifibatide).

Thrombolysis in myocardial infarction (TIMI) flow rate [7] was assessed before and at the end PPCI.

Obstructive coronary artery disease was defined as a ≥ 50% stenosis in the coronary artery lumen.

Statistical Analysis: All statistical studies were carried out using Statistical Package for Social Sciences software (SPSS 20.0 for Windows, SPSS Inc., Chicago, IL). The quantitative variables are expressed as mean ± standard deviation (SD). Normally distributed scale variables were expressed as mean ± standard deviation. Non-normally distributed variables were expressed as median and range. Categorical variables were expressed in numbers and percentages.

RESULTS

Total 750 acute myocardial infarction patients were identified; The present study included 150 young patients with acute myocardial infarction and confirmed diagnosis of COVID-19. All patients treated with a primary percutaneous coronary intervention (PPCI) during the period from January 2021 to June 2023.

Male patients represented 86.66% (n=130), while female patients represented 13.13% (n = 20). The mean age of the patients were 35.75 years ± 8.76 with range 20-45 years.

Regarding traditional risk factors the incidence of diabetes, hypertension, current smoking status, dyslipidemia and obesity were 16.16% (n=25), 8.66% (n=13), 2% (n =3), 9.33% (n = 14), 0.66 (n=1) respectively.

The mean heart rate (beat/min), oxygen saturation%, Serum creatinine, CRP, SBP and D-Dimer of our patients were 80, 92.76, 1.3, 86.51, 118, and 650.12 respectively.

	N	Range	Mean	SD
Age	150	20-45	35.75	8.76
Oxygen saturation	150	80-98	92.76	3.75
BMI (kg/m2)	150	2235	26.52	2.80
Serum creatinine (mg/dl)	150	0.7-2.0	1.3	0.42
CRP (mg/dl)	150	18-200	86.51	30.12
D-Dimer (ng/ml)	150	100-2000	650.12	342.13
SBP (mmHg)	150	80-140	118	12.0
Heart beat (beat/min)	150	60-120	80	11.0

Table 1: Basal clinical characteristics

Gender	Number of patients	Percentage
Male	130	86.66
Female	20	13.33

Table 2: Gender distribution

Risk factors	Number of patients	Percentage
Diabetes mellitus	25	16.66
Hypertension	13	8.66
Dyslipidemia	14	9.33

Obesity	1	0.66
Smoker and tobacco chewer	60	40

Table 3: Risk factors

Types	Number of patients	Percentage
SVD-Single vessel disease	100	66.66
DVD- Double vessel disease	30	20
TVD-Triple vessel disease	20	13.33

Table 4: Type of coronary artery involved

Artery	Number of patients	Percentage
LAD (left anterior descending (*LAD*) *arter)*	70	46.66
RCA (right coronary artery)	32	21.33
RAMUS	2	1.33
LCx	10	6.66
LMCA (Left main coronary artery)	1	0.66
LAD/LCx	8	5.33
LAD/RCA	7	4.66
LAD/RCA/LCx	3	2
LAD/PDA	1	0.66

Table 5: Artery involved

Percentage of stenosis	Number of patients	Percentage
100%	41	27.33
99%	20	13.33
95%	2	1.33
90%	32	21.33
80%	25	16.66
70%	14	9.33
60%	2	1.33

Table 6: Percentage of stenosis

Outcome	Number of patients	Percentage
Discharge	142	95%
Death	8	5%

Table 7: Outcome

Out of 150 patients, 142 patients discharged from hospital, 8 patients were dead.

DISCUSSION

The novel corona virus, severe acute respiratory syndrome corona virus 2 (SARS-CoV-2), which causes corona virus disease 2019 (COVID-19), is highly contagious in the community and has resulted in a global pandemic.[6]

Myocardial injury, due to underlying ischemia, acute thrombotic occlusion, or myocarditis, is reported in 7–28% of hospitalized COVID-19-positive patients and is associated with higher mortality.[7]

Many countries, including India, have instituted measures to limit transmission of the virus. India was implementing a partial night-time curfew during the study time, and this may explain the delay we noticed in the presentation of patients with MI in the present study. Atypical symptoms and patients' avoidance of hospitals for fear of contracting COVID-19 infection may be other factors that enhanced the delayed presentation of acute myocardial infarction.[8]

Also, COVID-19 infection is associated with a pro-thrombotic state. The occurrence of venous thromboembolic complications appears to be an important manifestation of the disease and one which is related to disease severity and outcome. The participation of these mechanisms may explain the high thrombus burden in patients of COVID-19 and presented by MI.[9]

Guo et al. showed in their retrospective single center study that patients with cardiovascular co-morbidities had a more cardiac injury when get infected by (SARS-CoV-2). 54.5% of patients with cardiovascular co-morbidities had an elevated troponin level compared to 13.2% of those without; this may explain the relatively less mortality (5%) in our patients.[10]

CONCLUSION

After covid era (after 2021) young myocardial infarction patients have increased. Especially young patients who are smoker and tobacco chewers, these were not confirmed cases of Covid. A non-obstructive CAD was found in about one third of patients; on the other hand, patients who had a total occlusion of their culprit artery, the thrombus burden was high. Identification of the underlying mechanism responsible for the high thrombus burden in these patients is important as

it may result in changes in their primary management strategy, either primary PCI, fibrinolytic therapy, or a pharmacoinvasive strategy. Furthermore, adjunctive anticoagulation and antiplatelet therapy may need to be revised.

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