Original Article

Influence of Clinical, Angiographic, and Developmental Characteristics and COVID-19 Severity and Vaccination Status on Young Patients with Acute Myocardial Infarction

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ABSTRACT

Background: Atherosclerosis is the most common cause of ischemic cardiovascular disease (CVD). However, approximately 20%–40% of cases of acute myocardial infarction (AMI) in patients aged <50 years can be attributed to genetic factors, and coronavirus disease 2019 (COVID-19) is a risk factor for blood clots and AMI. We aimed to describe the clinical, angiographic, and developmental characteristics and COVID-19 severity and vaccination status in patients aged ≤45 years with AMI. **Methods:** We retrospectively analyzed 2624 patients with AMI by reviewing the results of coronary angiography and percutaneous coronary intervention and medical reports. The study included patients aged >18 years who met the universal definition of AMI but excluded those with missing medical records or coronary angiograms. **Results:** In total, 2624 patients with AMI (aged 18–85 years) were included in the study and divided into two groups based on age: ≤45 (n = 1286) and >45 years (n = 1338). Total cholesterol and triglyceride levels were significantly higher in patients aged ≤45 years (5.6 ± 2.1 and 3.2 ± 2.1 mmol/L, respectively, $P < 0.007$) than in those aged >45 years (6.3 ± 1.8 and 3.1 ± 2.0 mmol/L, respectively, $P < 0.001$). Overall, 1745 and 879 patients were unvaccinated and fully vaccinated, respectively; severe and critical COVID-19 infections were more common among unvaccinated patients in both age groups. **Conclusions:** Younger patients with AMI were more likely to be smokers with no hypertension, diabetes mellitus, or previous AMI. In contrast, they were more likely to have hypercholesterolemia and hypertriglyceridemia, single-vessel disease, Type C lesions, and a history of drug-eluting stent use.

KEYWORDS: *Acute myocardial infarction, atherosclerosis, cardiovascular disease, coronavirus disease 2019*

INTRODUCTION

Ischemic cardiovascular disease (CVD) is primarily due to atherosclerosis progression; however, it can also develop in childhood and progress until it reaches its most severe forms in adulthood.[1] Certain lifestyle factors such as an unhealthy diet, smoking, and alcohol consumption predispose individuals to CVD; these are acquired during adolescence and early adulthood and are difficult to modify later. Therefore, determining the risk factors present since the onset of illness is important, as their persistence constitutes a substantial portion of the future risk of CVD.[2]

Prior to 1979, acute myocardial infarction (AMI) was not considered prevalent in patients aged <45 years;

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however, in 1979, approximately 4% of all AMIs occurred in patients aged <40 years.[3] Genetic predisposition is estimated to contribute to 20%–40% of all AMIs in patients aged <50 years. The association between 4G/5G polymorphisms in the plasminogen inhibitor gene and susceptibility to AMI, a genetic alteration found in diverse families, may explain this predisposition. Nevertheless, several studies contradict these findings.[4]

Moreover, in young individuals with normal coronary arteries, viral infections can precede the onset of AMI. Other causes of AMI include the constriction of one or both coronary birth orifices due to syphilitic aortitis, clogging of the arteries due to Takayasu's arteritis, and blockage of the coronary arteries due to giant cell arteritis, polyarteritis nodosa, systemic lupus erythematosus, or mucocutaneous lymph node syndrome (Kawasaki syndrome).[5] Furthermore, therapeutic radiation doses in the mediastinum can thicken and hyalinize the coronary artery walls, eventually causing AMI.[6]

Earlier studies describing AMI in young patients aged <45 years have shown that men comprise the majority of patients with AMI at this age. Most women aged <50 years are still in their reproductive years; therefore, these results might be explained by the cardioprotective effects of estrogen.[7] Similar to younger patients, older individuals with AMI exhibit a higher incidence of smoking and a lower frequency of other conventional CVD risk factors such as hypertension (HTN) and diabetes mellitus (DM),[8-10] and varying angiographic results have been reported in these populations.

Occlusion of the left anterior descending (LAD) artery, a cause of AMI in many younger individuals, appears to have a higher lesion complexity and a lower disease load in younger patients. However, in approximately 6% of all patients with AMI and potentially as many as 4% of those aged <35 years who have been diagnosed with AMI, coronary atherosclerosis is not detectable on arteriography or at necropsy. Patients with AMI who have angiographically healthy coronary arteries are typically young and have less coronary risk factors.[11]

According to previous studies, coronavirus disease 2019 (COVID-19) is associated with a higher risk of thrombosis and a greater risk of AMI.[12] Moreover, vaccine-induced immune thrombotic thrombocytopenia, a condition similar to heparin-induced thrombocytopenia, may cause the thrombotic phenomenon that appears after vaccination.[13] After receiving the COVID-19 vaccine, AMI might result due to Kounis syndrome, a vaccine-induced allergic vasospastic response.[14,15] Although vaccinations against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) are effective in preventing COVID-19 from progressing into a serious illness,[16] whether vaccinations prevent further complications remains unknown.

In a previous systematic review and meta-analysis, AMI occurred in approximately 0.5% of patients who had recovered from COVID-19 during the follow-up period.[17] Moreover, those who survived had a 93% increased risk of AMI following recovery from COVID-19, which was negatively correlated with the duration of the follow-up. Furthermore, following an average follow-up period of 8.5 months, the prevalence and susceptibility to AMI were greater in patients who had recovered from COVID-19 than in controls.[17]

AMI is a public health issue in this patient population and is, therefore, significant in clinical research because it affects people during their productive age. This has implications for their socioeconomic status and contributes significantly to admissions to intensive care units. Therefore, we aimed to describe the clinical, angiographic, and developmental characteristics and COVID-19 severity and vaccination status of AMI patients aged ≤45 years.

Methods

Participants and design

We conducted a descriptive, longitudinal retrospective analysis on 2624 patients with AMI by reviewing coronary angiography and percutaneous coronary intervention reports and medical records from ten main Tertiary Care Hospitals in India between May 2022 and October 2023.

All patients aged >18 years who met the standards of the universal definition of AMI were included.[18] Patients were excluded if their medical records and/or coronary angiograms were missing critical information. The presence of AMI was determined by confirming the appearance of typical elevation and gradual decline or rapid elevation and decline of troponin with at least one of the following factors: typical ischemia-related symptoms, development of pathological Q waves in the electrocardiogram, changes in the ST segment that indicate ischemia, coronary artery intervention such as coronary angioplasty, and performance of invasive coronary angiography during admission.

Information from the collected data was incorporated into an anonymized database using Excel 2016, and each patient was assigned a sequential number. In this study, we observed and noted the following traits and variables: demographic (age and sex), clinical (smoking, HTN,

DM, dyslipidemia, body mass index [BMI], history of previous AMI, and type of AMI (ST-elevation myocardial infarction [STEMI] or non-ST-EMI [NSTEMI]), biochemical (total cholesterol, triglycerides, low-density lipoprotein cholesterol [LDL-C], and high-density lipoprotein cholesterol [HDL-C]), angiographic (number of vessels, type of vessel treated, type of lesion, number and type of stent, and stent diameter and length), the severity of COVID-19 (requirement for supplementary oxygen [severe], higher respiratory support [critical], and no requirement for respiratory support), COVID-19 vaccination status (unvaccinated and fully vaccinated), and evolution-related (immediate surgery results and unfavorable follow-up occurrences). The median follow-up duration was 90 day for unvaccinated patients diagnosed with COVID-19 within 30 days before the follow-up period started, whereas that for fully vaccinated patients was 84 day.

Statistical analyses

Qualitative and quantitative variables are presented as numbers with percentages and means with standard deviations, respectively. We performed Pearson's Chi-squared and Student's *t*-tests to evaluate the significance of the differences between qualitative and quantitative variables, as appropriate. All statistical analyses and processing were performed using Microsoft Excel, Statistica Excel, and Statistica 8.0. Differences were considered statistically significant at $P < 0.05$.

Results

In total, 2624 patients with AMI aged 18–85 years were included in this study and divided into the following two groups according to age: ≤45 years (n = 1286) and >45 years (n = 1338). We found that the distribution of patients according to age was similar and had no significant differences, with a similar predominance of males in both groups [65.8% vs. 68.2%, respectively, $P = 0.186$, Table 1].

Regarding comorbidities, compared to the younger group, patients aged >45 years had a higher prevalence of HTN (45.4% vs. 70.8%, $P < 0.001$), DM (24.6% vs. 28.7%, $P = 0.029$), and a history of previous AMI (16.5% vs. 25.2%, $P = 0.021$). In contrast, compared to patients aged >45 years, younger patients tended to have a higher BMI (30.7 ± 5.0 vs. 31.3 ± 5.1, $P = 0.184$); however, this difference was not significant. Additionally, the prevalence of dyslipidemia between the groups was not significantly different [Table 2].

Table 1: Demographic variables distribution of patients based on age and gender

Demographic variables	Age (years)		OR (95% CI)	*P*
	<45 (*n*=1286)	>45 (*n*=1338)		
Male	846 (65.8)	912 (68.2)	0.89 (0.69–1.46)	0.186
Female	440 (34.2)	426 (31.8)		

Data as *n* (%). OR: Odds ratio, CI: Confidence interval

Patients aged ≤45 years showed significantly lower levels of total cholesterol (5.6 ± 2.1 vs. 6.3 ± 1.8 mmol/L, $P < 0.007$) and significantly higher levels of triglycerides (3.2 ± 2.1 vs. 3.1 ± 2.0 mmol/L, $P < 0.001$), than those in the patients aged >45 years. However, HDL-C and LDL-C showed no significant differences between the groups [Table 3].

Regarding the number of diseased vessels, the group aged ≤45 years had a significantly higher percentage of patients with single-vessel disease (88.3% vs. 57.5%, $P < 0.001$) and significantly fewer patients with two diseased vessels (9.5% vs. 18.5%, $P < 0.001$) than the older group; however, no significant differences were observed for diseases involving three or more vessels. Moreover, those aged >45 years showed a significantly higher percentage of patients with coronary artery disease (CAD) (2.9% vs. 0.3%, $P = 0.020$) than that in younger patients. Conversely, patients aged ≤45 years had a significantly higher percentage of Type C lesions (37.2% vs. 27.2%, $P = 0.023$) than that of the older group.

Concerning the implanted stents, we analyzed 2852 stents as follows: 859 (1.84 ± 0.7) in patients aged ≤45 years and 1993 (1.92 ± 0.9) in patients aged >45 years. The stent diameter was significantly greater in patients aged ≤45 years than in older patients, whereas no significant differences in stent length were observed. Furthermore, while the frequency of drug-eluting stent usage was significantly higher in patients aged ≤45 years than in older patients (31.7% vs. 15.9%, $P < 0.001$), the number of stents was similar between the two groups [Table 4].

A higher percentage of successful procedures was observed in patients aged ≤45 years than in those aged >40 years (90.9% vs. 90.1%, $P = 0.087$). Moreover, compared to the group aged >45 years, the younger age group had a lower percentage of patients with complicated (5.0% vs. 6.9%, $P = 0.526$) failed procedures and a higher percentage of patients with uncomplicated (4.1% vs. 2.9%, $P = 0.413$) failed procedures; however, these differences were not significant [Table 5]. Furthermore, the percentage of adverse events during the 1st year of AMI presentation was significantly higher in patients aged >45 years than in those aged ≤45 years [5.6% vs. 3.4%, $P = 0.038$, Table 6].

Table 2: Clinical variables distribution of patients based on age

Clinical variables	Age (years)		OR (95% CI)	*P*
	<45 (*n*=1286)	>45 (*n*=1338)		
Smoking	795 (61.8)	869 (65.0)	2.61 (1.83–3.76)	<0.001*
HTN	584 (45.4)	947 (70.8)	1.14 (0.96–1.30)	<0.001*
Dyslipidemia	245 (19.0)	276 (20.6)	1.06 (0.98–1.18)	0.567
DM	316 (24.6)	384 (28.7)	1.03 (0.95–1.09)	0.029
BMI	31.3±5.1	30.7±5.0	-	0.184
Previous history of AMI	212 (16.5)	337 (25.2)	0.73 (0.58–0.98)	0.021*
STEMI	863 (67.2)	891 (66.6)	3.16 (2.06–4.85)	<0.001*
NSTEMI	423 (32.8)	447 (33.4)		

*Signifies highly significant *P*<0.001. Data as *n* (%). Data as mean±SD (25^{th}–75^{th} percentile). OR: Odds ratio, CI: Confidence interval, AMI: Acute myocardial infarction, STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non-ST-segment elevation myocardial infarction, HTN: Hypertension, DM: Diabetes mellitus, BMI: Body mass index, SD: Standard deviation

Table 3: Biochemical parameters distribution of patients based on age

Biochemical parameters	Age (years)		*P*
	<45 (*n*=1286)	>45 (*n*=1338)	
Total cholesterol (mmol/L)	5.6±2.1	6.3±1.8	<0.001*
Triglycerides (mmol/L)	3.2±2.1	3.1±2.0	<0.001*
HDL-C (mmol/L)	1.2±0.6	1.2±0.6	0.567
LDL-C (mmol/L)	4.3±0.9	4.1±0.8	0.029

*Signifies highly significant *P*<0.001. Data as mean±SD (25^{th}–75^{th} percentile). SD: Standard deviation, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol

Regarding vaccination status, 1745 patients were unvaccinated, and 879 were fully vaccinated [Table 7]. Unvaccinated patients were older and had more comorbidities [Table 7]. In both age groups, most patients were unvaccinated; however, the group aged ≤45 years had a larger proportion of unvaccinated patients than that of the older group (69.8% and 66.7%, $P = 0.002$). Moreover, in both age groups, most males were unvaccinated (45.5% vs. 47.8%, $P < 0.001$) [Table 8]. Relating to comorbidities, most of the unvaccinated groups of patients aged ≤45 years and >45 years had higher percentages of smokers (38.0% vs. 40.7%, $P < 0.001$) and those with HTN (24.9% vs. 42.0%, $P < 0.001$). Furthermore, a history of previous AMI was prevalent in 214 (16.0%) unvaccinated patients aged >45 years. In both age groups, both STEMI and NSTEMI were mainly observed in unvaccinated patients (STEMI: 47.5% vs. 44.4%, $P < 0.001$; NSTEMI: 18.7% vs. 22.3%, $P < 0.001$).

In both age groups, patients with severe and critical COVID-19 were more common among unvaccinated patients than those among fully vaccinated patients [Table 8], and 89 patients with severe or critical COVID-19 were male [Table 9]. Regarding comorbidities in patients with severe COVID-19, smoking was observed in 2.5% vs. 2.3% of patients aged ≤45 and >45 years, respectively. In addition, a history of a previous AMI was observed in 1.6% vs. 1.7% of patients aged ≤45 and >45 years, respectively. Moreover, in the group aged >45 years, 43 and 33 patients with severe COVID-19 had HTN and dyslipidemia, respectively. STEMI and NSTEMI were mainly observed in patients with severe and critical COVID-19 [2.0% vs. 1.6% and 1.7% vs. 1.9%, respectively, Table 9] in both age groups.

Discussion

Our results revealed a comparable sex distribution between the age groups, consistent with other studies reporting a predominance of males among patients aged >40 years who had AMI.[19-21] Moreover, androgen levels, which exhibit a negative correlation with the incidence of STEMI and are known to prevent atherosclerosis, are significantly associated with an increased incidence of CAD, and can predict AMI.[22] Similarly, as most women aged <50 years are still in their fertile years, they benefit from the cardioprotective effects of estrogen.

Smoking promotes atherosclerosis, increases LDL-C oxidation, and reduces HDL-C levels; therefore, it is widely regarded as the primary risk factor for infarction in patients aged <50 years.[23] Additionally, it impairs endothelium-dependent vasodilation of the coronary arteries, increases fibrinogen concentration and platelet aggregation, and increases the incidence of coronary artery spasm. A previous study of 6892 patients with STEMI who underwent percutaneous coronary intervention revealed that 46.4% of the participants were smokers.[24] Furthermore, in a Singaporean registry, 74% of patients with AMI aged >45 years were smokers, which is similar to the results for patients aged ≤45 years in the present study.[10]

Given the higher prevalence of smoking and single-vessel disease in young people, coronary occlusion in these

Table 4: Angiographic variables distribution of patients based on age

Angiographic variables	Age (years)		OR (95% CI)	*P*
	<45 (*n*=1286)	>45 (*n*=1338)		
Number of vessels				
1	1136 (88.3)	1019 (57.5)	1.77 (1.02–2.11)	0.001*
2	122 (9.5)	247 (18.5)	1.01 (0.53–1.49)	0.007*
>3	28 (2.2)	72 (5.3)	0.65 (0.32–0.98)	0.131
Type of vessels				
CAD	4 (0.3)	39 (2.9)	1.16 (0.74–1.38)	0.020*
LAD	813 (63.2)	734 (54.9)	0.41 (0.23–0.69)	0.072
Cx	158 (12.3)	198 (14.8)	1.03 (0.85–1.28)	0.324
RCA	311 (24.2)	367 (27.4)	2.13 (1.71–2.44)	0.217
Lesion characteristics				
A	311 (24.2)	287 (21.4)	0.83 (0.51–1.37)	0.613
B1	255 (19.8)	357 (26.7)	1.08 (0.77–1.43)	0.063
B2	242 (18.8)	330 (24.7)	1.15 (0.86–1.52)	0.305
C	478 (37.2)	364 (27.2)	0.96 (0.61–1.33)	0.023*
Number of stents	1.84±0.7	1.92±0.9	-	0.478
Type of stents				
BMS	878 (68.3)	1125 (84.1)	3.17 (2.45–4.89)	<0.001*
DES	408 (31.7)	213 (15.9)		
Stent diameter	2.81±0.5	2.67±0.4	-	0.008*
Stent length	2.08±7.2	19.4±6.7	-	0.432

*Signifies highly significant *P*<0.001. Data as *n* (%). Data as mean±SD (25th–75th percentile). OR: Odds ratio, CI: Confidence interval, CAD: Coronary artery disease, LAD: Left anterior descending artery, Cx: Circumflex artery, RCA: Right coronary artery, A: Type A, B1: Type B1, B2: Type B2, C: Type C, BMS: Bare metal stent, DES: Drug-eluting stent

Table 5: Immediate outcomes of the procedure distribution of patients based on age

Immediate outcomes	Age (years)		RR (95% CI)	*P*
	<45 (*n*=1286)	>45 (*n*=1338)		
Successful	1169 (90.9)	1206 (90.1)	1.26 (0.84–1.48)	0.087
Complicated	64 (5.0)	93 (6.9)	1.04 (0.59–1.27)	0.526
Uncomplicated	53 (4.1)	39 (2.9)	0.84 (0.36–1.68)	0.413

Data as *n* (%). RR: Relative risk, CI: Confidence interval

Table 6: Short-term adverse events distribution of patients based on age

Adverse events	Age (years)		OR (95% CI)	*P*
	≤45 (*n*=1286)	>45 (*n*=1338)		
Yes	44 (3.4)	75 (5.6)	2.86 (0.88–8.54)	0.038*
No	1242 (96.6)	1263 (94.4)		

*Signifies highly significant *P*<0.001. Data as *n* (%).OR: Odds ratio, CI: Confidence interval

patients primarily involves thrombogenic and vasospastic components, whereas fewer atherosclerotic components are involved. Therefore, decreasing smoking habits may be a more effective strategy for preventing acute coronary syndrome (ACS) in young adults than in older age groups.[19]

In this study, patients aged >45 years had a higher prevalence of HTN, DM, and previous AMI than that in younger patients, consistent with the results reported by Al-Murayeh *et al*.[25] Similarly, Panduranga *et al*. found that the percentage of patients with HTN, DM, and preinfarction angina was significantly higher in patients aged >40 years than in younger patients.[26]

In another study, STEMI was the most common cause of ACS presentation in 128 patients aged <45 years, accounting for 69% of cases, followed by NSTEMI in 20% and unstable angina in 9%.[27] Similarly, Panduranga *et al*. noted a greater predominance of STEMI in younger patients (32%) than in older patients (24%).[26] They also found that the proportion of NSTEMI was similar in both groups (25%), whereas unstable angina was less common in younger patients (43%) than in older patients (51%).[26]

Hokanson and Austin previously reported hypertriglyceridemia as an independent risk factor for CAD.[28] For every 1 mmol/L increase in triglyceride level, the risk of CAD increases by 37% in women and 14% in men. In the present study, we observed significant differences in the levels of total cholesterol and triglycerides between the compared groups, whereas HDL-C and LDL-C levels were similar. In contrast, Kava *et al*. reported a correlation between AMI in young patients and a reduction in the HDL ApoA-l/ApoC-111 ratio, changes in the distribution of the HDL-C subpopulation, and an increase in the oxidation potential of HDL-C.[29]

Table 7: Coronavirus disease-19 severity and vaccination status distribution of patients based on age

	Age (years)		OR (95% CI)	*P*
	<45 (*n*=1286)	>45 (*n*=1338)		
COVID-19 vaccination status				
Unvaccinated	852 (69.8)	893 (66.7)	0.29 (0.11–0.65)	0.002
Fully vaccinated	434 (30.2)	445 (33.3)	0.36 (0.19–0.59)	<0.001*
Severity of COVID-19				
Severe	57 (4.4)	58 (4.3)	0.32 (0.16–0.48)	0.003
Critical	26 (2.0)	29 (2.2)	0.24 (0.08–0.42)	<0.001*

*Signifies highly significant *P*<0.001. Data as *n* (%). Data as mean±SD (25th–75th percentile). OR: Odds ratio, CI: Confidence interval, COVID-19: Coronavirus disease-19, SD: Standard deviation

Table 8: Risk for cardiovascular events in patients based on age and coronavirus disease-19 vaccination status

	Age (years)				OR (95% CI)	*P*
	<45 (*n*=1286)		>45 (*n*=1338)			
	COVID-19 vaccination status					
	Unvaccinated	Fully vaccinated	Unvaccinated	Fully vaccinated		
Gender						
Male	585 (45.5)	261 (20.3)	640 (47.8)	283 (21.2)	0.36 (0.19–0.55)	<0.001*
Female	267 (20.8)	173 (13.4)	253 (18.9)	162 (12.1)	0.44 (0.29–0.64)	0.003
Smoking	489 (38.0)	306 (23.8)	545 (40.7)	312 (23.3)	0.52 (0.34–0.82)	<0.001*
Previous history of AMI	110 (8.5)	102 (8.0)	214 (16.0)	123 (9.2)	0.38 (0.29–0.59)	0.002
STEMI	611 (47.5)	252 (19.6)	595 (44.4)	306 (22.9)	0.39 (0.24–0.71)	<0.001*
NSTEMI	241 (18.7)	182 (14.2)	298 (22.3)	139 (10.4)		
HTN	320 (24.9)	264 (20.5)	562 (42.0)	385 (28.8)	0.40 (0.27–0.83)	<0.001*
Dyslipidemia	136 (10.5)	109 (8.5)	149 (11.6)	127 (8.2)	0.53 (0.33–0.95)	0.004
DM	168 (13.1)	128 (9.9)	227 (17.6)	110 (10.5)	0.06 (0.03–0.24)	<0.001*
BMI	32.5±6.2	30.1±5.3	31.6±5.9	30.9±4.8	-	0.037
Severity of COVID-19						
Severe	34 (2.6)	23 (1.8)	32 (2.4)	26 (1.9)	0.51 (0.36–0.89)	0.043
Critical	15 (1.2)	11 (0.9)	16 (1.2)	13 (1.0)	0.36 (0.23–0.62)	0.021

*Signifies highly significant *P*<0.001. Data as *n* (%). Data as mean±SD (25th–75th percentile). OR: Odds ratio, CI: Confidence interval, AMI: Acute myocardial infarction, STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non-ST-segment elevation myocardial infarction, HTN: Hypertension, DM: Diabetes mellitus, BMI: Body mass index, COVID-19: Coronavirus disease-19, SD: Standard deviation

Table 9: Risk for cardiovascular events in patients based on age and severity of coronavirus disease-19

	Age (years)				OR (95% CI)	*P*
	<45 (*n*=1286)		>45 (*n*=1338)			
	Severity of COVID-19					
	Severe	Critical	Severe	Critical		
Gender						
Male	30 (2.3)	14 (1.1)	30 (2.2)	15 (1.1)	0.33 (0.14–0.48)	<0.001*
Female	27 (2.1)	12 (0.9)	28 (2.1)	14 (1.0)	0.39 (0.24–0.56)	0.002
Smoking	32 (2.5)	13 (1.0)	31 (2.3)	10 (0.8)	0.23 (0.12–0.35)	<0.001*
Previous history of AMI	21 (1.6)	11 (0.9)	23 (1.7)	12 (0.9)	0.31 (0.18–0.47)	0.003
STEMI	26 (2.0)	10 (0.8)	22 (1.6)	11 (0.8)	0.16 (0.08–0.39)	<0.001*
NSTEMI	22 (1.7)	13 (1.0)	25 (1.9)	12 (0.9)		
HTN	30 (2.3)	18 (1.4)	43 (3.2)	14 (1.0)	0.21 (0.10–0.36)	<0.001*
Dyslipidemia	28 (2.2)	15 (1.1)	33 (2.5)	16 (1.2)	0.26 (0.13–0.56)	0.002
DM	16 (1.2)	11 (0.9)	19 (1.4)	14 (1.0)	0.21 (0.10–0.44)	<0.001*
BMI	32.2±6.5	31.9±5.8	33.1±5.9	32.7±6.1	-	0.042

*Signifies highly significant *P*<0.001. Data as *n* (%). Data as mean±SD (25th–75th percentile). OR: Odds ratio, CI: Confidence interval, AMI: Acute myocardial infarction, STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non-ST-segment elevation myocardial infarction, HTN: Hypertension, DM: Diabetes mellitus, BMI: Body mass index, COVID-19: Coronavirus disease-19, SD: Standard deviation

Regarding angiographic variables, we found a predominance of single-vessel disease, involvement of the LAD artery, Type C lesions, use of drug-eluting stents, and reduced left main CAD in patients aged ≤45 years. Similar results were obtained in another study that found a predominance of LAD artery involvement (48.0%), a low percentage of patients with two-vessel (14.9%) and three-vessel disease (3.4%), and an active drug-eluting stent usage of 42.6%.[24] Esteban *et al*. reported a predominance of LAD involvement (43.9%), one-vessel disease (44.7%), and active drug-eluting stent usage (32.5%) in 123 patients aged <40 years who had suffered from AMI.[30] In a comparative study of 2424 patients with AMI treated with interventional procedures, the frequency of LAD artery involvement was significantly higher and that of multivessel disease was significantly lower in the group aged <40 years.[31] Other studies have also reported a higher incidence of single-vessel disease and LAD involvement in this population.[32,33]

The survival rate of patients with single vessel disease is lower in the diabetic population owing to increased complications such as early mortality, cardiogenic shock, myocardial rupture, heart failure, and acute arrhythmias. In addition, the use of drug-eluting stents is higher in this population and has been linked to a higher incidence of heart failure. Therefore, the increased use of drug-eluting stents in young patients with AMI may have contributed to the differences in survival observed in the present study.

Similarly, Al-Murayeh *et al*. reported a higher incidence of AMI and heart failure both in hospital and 1 month after discharge in patients aged >40 years than in younger patients.[25] In addition, Ergelen *et al*. reported that in-hospital mortality was four times higher in older patients than in younger patients in this population.[31] Chen *et al*. reported similar procedural success rates between the two comparison groups in China, and the mortality at 30 day after AMI was significantly lower in the younger group than in the older group.[34]

Patients who have CVD before contracting COVID-19 are at risk of serious outcomes causing death and other sequelae.[35] In addition to potentially causing problems such as blood clots, irregular heartbeat, and myocardial damage, COVID-19 can aggravate previous CVD.[36] In fact, individuals who already have heart failure and are infected with SARS-CoV-2 are more likely to require critical care interventions such as mechanical ventilation, dialysis, and admission to an intensive care unit.[37] Therefore, preexisting heart failure is also a powerful predictor of mortality in hospitalized COVID-19 patients.[38] Furthermore, direct cardiovascular (CV) damage has been associated with COVID-19.[39]

The risk of AMI is significantly increased by COVID-19 both during and after acute infection.[40] Hospitalized adult patients with COVID-19 had a 3–8-fold greater risk of AMI,[40] while the correlation between infection and CVD death varied from 6.7% to 73%.[41,42] Various COVID-19 vaccines are also related to CV complications, including myocarditis,[43,44] and mRNA vaccines may be associated with a higher rate of ACS.[43,45] However, after numerous doses, the risk is minimal, often resulting in mild myocarditis, rapid healing, and no long-term effects.[44]

The present study examined data from hospitals in India and revealed a correlation between full vaccination and a reduction in the risk of AMI following COVID-19, consistent with the results of other studies.[46,47] The strength of our study lies in its inclusion of the largest hospitals in India, characterized by their size and racial diversity. However, this study had some limitations. First, our findings may have been affected by unmeasured confounding variables and the inability to consider vaccines beyond those distributed in India. Second, we did not consider SARS-CoV-2 reinfection in this study, as patients may test positive for varying periods. Lastly, we were unable to distinguish between the different SARS-CoV-2 variants responsible for the infection.

Conclusions

The frequency of STEMI was higher in younger patients with AMI who were primarily smokers and had no history of HTN, DM, or AMI. In contrast, compared to older patients, younger patients with AMI were more likely to have hypercholesterolemia, hypertriglyceridemia, single-vessel disease, Type C lesions, and a history of drug-eluting stent use. However, they were also found to have a higher success rate of interventional procedures and a lower incidence of short-term adverse events.

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Conflicts of interest

There are no conflicts of interest.

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