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Original Article

Type 2 Myocardial Infarction: Patient Characteristics and Clinical Outcomes

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ABSTRACT

Background: Type 2 myocardial infarction (MI) is caused by myocardial ischemia resulting from an imbalance between oxygen supply and demand in the heart. This study aimed to assess the incidence, variations in clinical characteristics, current therapeutic approaches, and overall prognosis in patients with type 2 MI compared to those with type 1 MI.

Methods: This prospective, cross-sectional study involved 350 Egyptian patients presenting with MI. Participants were categorized into type 1 MI (n = 262) and type 2 MI (n = 88) groups based on standard diagnostic criteria. Data regarding demographic characteristics, clinical presentations, management strategies, and outcomes, including in-hospital, 30-day, and 6-month mortality rates; major adverse cardiovascular events (MACE); and hospitalization for heart failure, were collected and analyzed.

Results: Coronary angiography was performed more frequently in patients with type 2 MI than in those with type 1 MI. Primary percutaneous coronary intervention and coronary artery bypass grafting were more commonly performed in type 1 MI patients. A statistically significant difference was observed between type 1 MI and type 2 MI concerning echocardiographic findings and hospital stays. However, there was no significant difference in MACE at 1-month and 6-month follow-ups, except for mortality and stroke, where a statistically significant difference was found.

Conclusions: Type 1 MI is more common than type 2 MI. Nonetheless, type 2 MI is associated with higher mortality rates and longer hospital stays. Patients with type 1 MI typically present with chest pain, while those with type 2 MI predominantly present with dyspnea, syncope, and hemodynamic instability. Cardiac interventions are primarily performed in patients with type 1 MI. ***(Iranian Heart Journal 2024; 25(4): 51-58)***

KEYWORDS: Myocardial infarction, Coronary artery disease, Echocardiography

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Type 2 myocardial infarction (MI) is caused by myocardial ischemia due to an imbalance between oxygen supply and demand in the heart, rather than plaque rupture. It is often associated with medical conditions other than coronary artery disease. Nevertheless, current data do not provide a clear comparison between type 1 and type 2 MI regarding mortality and the prevalence of traditional coronary risk factors. Over the past decade, the incidence of type 2 MI has evolved, approaching that of type 1 MI. Mortality following type 2 MI is higher and primarily results from non-cardiovascular causes in the early stages. The supply-demand mismatch affects long-term survival, underscoring the burden of type 2 MI on healthcare systems and providing insights for designing clinical trials. [1]

MI triggers a robust inflammatory response characterized by leukocyte infiltration into the infarcted heart, potentially exacerbating ischemia-induced damage through cytotoxic effects. Postinfarction ventricular remodeling, which is closely linked to the pathogenesis of heart failure following MI, involves structural, functional, and geometric changes affecting both infarcted and non-infarcted myocardial segments. These changes result in chamber dilation, increased ventricular sphericity, and cardiac dysfunction.

Cardiac remodeling is associated with the progression of heart failure, increased risks of arrhythmias, and poor prognoses for MI survivors. The extent of remodeling following an MI depends on the infarct size and the quality of the cardiac repair. Although experimental studies have challenged the notion that inflammatory signals exacerbate ischemic injury, there is consensus on the critical role of inflammatory pathways in the dilative and fibrotic remodeling of the infarcted heart. These pathways are crucial in the pathophysiology of postinfarction heart failure. [2, 3]

The objective of this study was to assess the incidence, variations in clinical characteristics, current therapeutic approaches, and overall prognosis in patients with type 2 MI compared with those with type 1 acute MI.

METHODS

This prospective, cross-sectional study enrolled 350 Egyptian patients presenting with MI at Ain Shams University Hospital and Misr University for Science and Technology Hospital. Participants were recruited over a 1-year period, from February 2022 through February 2023, and were divided into 2 groups: type 1 MI and type 2 MI, based on standard diagnostic criteria. Patients were followed up to evaluate the prevalence, patient characteristics, in-hospital management, and clinical outcomes, including in-hospital, 30-day, and 6-month mortality rates; major adverse cardiovascular events (MACE); and heart failure hospitalization. Type 1 MI is diagnosed based on the presence of acute atherothrombosis in the artery supplying the infarcted myocardium. Type 2 MI is identified by evidence of oxygen supply-demand imbalance in the heart unrelated to acute atherothrombosis, accompanied by symptoms or indicators of ischemia. [4]

Patients were classified into 2 categories based on the underlying physiopathological mechanism: type 1 MI was diagnosed when the presence of an intracoronary thrombus resulting from atheromatous plaque rupture was indicated, and type 2 MI was diagnosed when oxygen supply or demand imbalance in the heart was attributed to a factor other than probable coronary artery disease. [5]

Oxygen supply reduction was attributed to conditions such as anemia, shock, bradycardia, coronary embolism, and respiratory failure. Oxygen demand increase was associated with circumstances including supraventricular tachyarrhythmia with a

ventricular rate exceeding 150 beats per minute lasting for 20 minutes, hypertensive pulmonary edema necessitating nitrate or diuretic therapy, and arterial hypertension with systolic blood pressure above 160 mm Hg along with concurrent left ventricular hypertrophy detected by echocardiography or ECG.

Inclusion criteria encompassed patients aged 18 years and older presenting with MI based on the fourth universal definition, which includes confirmed myocardial injury (elevated cardiac troponin levels above the upper reference limit with or without symptoms indicative of acute myocardial ischemia), evidence of ischemic heart disease, incidentally discovered impaired left ventricular ejection fraction, or new-onset mitral regurgitation attributable to ischemia. Exclusion criteria included patients with sepsis, shock, anemia, active bleeding, tachyarrhythmia, bradyarrhythmia, respiratory failure, hypertensive crisis, stroke/transient ischemic attack resulting in encephalopathy, coronary embolism, and heart failure leading to elevated cardiac troponin levels and myocardial injury due to coronary oxygen demand-supply mismatch.

Additional exclusion criteria comprised individuals under the age of eighteen, those who experienced cardiac arrest and subsequently recovered, and patients with clinical conditions such as myocarditis, pulmonary embolism, acute or chronic renal failure, and myocardial trauma. These conditions are known to be associated with elevated troponin levels in the absence of symptoms or indicators of myocardial ischemia, a phenomenon referred to as non-ischemic myocardial injury. In non-ischemic myocardial injury, elevated troponin levels may result from inflammatory processes, apoptosis, myocardial strain, cellular injury, or reduced cardiac troponin clearance. Patients residing outside of the referral area were also excluded from the study.

A comprehensive clinical evaluation was performed for all patients, which included detailed history taking and physical examination with particular attention to blood pressure measurement, heart rate assessment, and cardiac examination. Investigations such as surface ECG, routine echocardiography, and routine laboratory tests were conducted for each patient.

Ethical Considerations: All participants provided informed consent before their involvement in the study. The study design and procedures were approved by the University Hospital's ethics committee.

Statistical Analysis: Data collection, tabulation, and analysis were conducted to ensure adequate statistical representation of the outcomes. All results were expressed as mean ± standard deviation and subsequently presented in tabular form.

RESULTS

A statistically significant difference was observed between type 1 MI and type 2 MI regarding age and diabetes, and a highly statistically significant difference was found in terms of sex, smoking status, history of ischemic heart disease, history of congestive heart failure, family history, and hypertension (Table 1). A significantly higher proportion of patients with type 2 MI underwent coronary angiography, while percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG) were more frequently performed in type 1 MI patients ($P < 0.001$) (Table 2).

A highly statistically significant difference was observed between type 1 MI and type 2 MI regarding ECG changes and heart rate (Table 3).

A highly statistically significant difference was observed between type 1 MI and type 2 MI in terms of echocardiographic findings, including ejection fraction, diastolic dysfunction, segmental wall motion abnormalities, and mitral regurgitation (Table 4).

Among patients with type 2 MI, the triggering diseases were hypertensive crisis in 17 (19.3%), bradycardia in 17 (19.3%), tachyarrhythmia in 12 (13.6%), acute bleeding/anemia in 11 (12.5%), sepsis in 10 (11.4%), chronic kidney disease in 10 (11.4%), heart failure in 6 (6.8%), and respiratory failure in 5 (5.7%) (Table 5). A highly statistically significant difference was observed between type 1 MI and type 2 MI concerning hospital stays. No statistically significant difference was found regarding MACE at 1 month and 6 months, except for death and stroke, where a statistically significant difference was noted (Table 6).

Table 1: Comparisons Between Type 1 MI and Type 2 MI Regarding Patient Characteristics

Patient Characteristics		Type 1 MI No. = 262	Type 2 MI No. = 88	Test value	*P* value	Sig.
Sex	female	101 (38.5%)	52 (59.1%)	11.297*	0.001	HS
	male	161 (61.5%)	36 (40.9%)			
Age, y	Mean ± SD	56.94 ± 8.37	54.60 ± 12.22	2.004•	0.046	S
	Range	34 – 68	40 – 72			
Smoking	No	88 (33.6%)	70 (79.5%)	56.184*	<0.001	HS
	Yes	174 (66.4%)	18 (20.5%)			
History of IHD	No	191 (72.9%)	78 (88.6%)	9.170*	0.002	HS
	Yes	71 (27.1%)	10 (11.4%)			
History of CHF	No	228 (87.0%)	52 (59.1%)	32.122*	<0.001	HS
	Yes	34 (13.0%)	36 (40.9%)			
Family history	No	139 (53.1%)	77 (87.5%)	33082*	<0.001	HS
	Yes	123 (46.9%)	11 (12.5%)			
Diabetes	No	134 (51.1%)	57 (64.8%)	4.935*	0.026	S
	Yes	128 (48.9%)	31 (35.2%)			
Hypertension	No	35 (13.4%)	52 (59.1%)	73.760*	<0.001	HS
	Yes	227 (86.6%)	36 (40.9%)			

MI: myocardial infarction, IHD: ischemic heart disease, CHF: congestive heart failure
P > 0.05: nonsignificant; *P* < 0.05: significant; *P* < 0.01: highly significant
* χ^2 test • independent T-test

Table 2: Comparisons Between Type 1 MI and Type 2 MI Regarding Invasive Data

Invasive		Type 1 MI No. = 262	Type 2 MI No. = 88	Test value	***P*** value	Sig.
Cardiac Catheterization	Not done	0 (0.0%)	40 (45.5%)	163.250*	<0.001	HS
	CA	52 (19.8%)	31 (35.2%)			
	PCI	210 (80.2%)	17 (19.3%)			
CABG	No	210 (80.2%)	88 (100.0%)	20.513*	<0.001	HS
	Yes	52 (19.8%)	0 (0.0%)			

MI: myocardial infarction, CA: coronary angiography, PCI: percutaneous coronary intervention, CABG: coronary artery bypass grafting

Table 3: Comparisons Between Type 1 MI and Type 2 MI Regarding ECG Findings

ECG		Type 1 MI No. = 262	Type 2 MI No. = 88	Test value	*P* value	Sig.
ECG Changes	Normal	0 (0.0%)	10 (11.4%)	30.648*	<0.001	HS
	ST elevation	108 (41.2%)	17 (19.3%)	13.765*	<0.001	HS
	ST depression	50 (19.1%)	20 (22.7%)	0.546*	0.460	NS
	Bundle block	35 (13.4%)	18 (20.5%)	2.581*	0.108	NS
	T-wave inversion	34 (13.0%)	10 (11.4%)	0.156*	0.693	NS
	AF	35 (13.4%)	13 (14.8%)	0.111*	0.739	NS
Heart Rate	Normal heart rate	228 (87.0%)	35 (39.8%)	82.274*	<0.001	HS
	Tachycardia	17 (6.5%)	36 (40.9%)			
	Bradycardia	17 (6.5%)	17 (19.3%)			

MI: myocardial Infarction, ST: ST-segment, AF: atrial fibrillation

Table 4: Comparisons Between Type 1 MI and Type 2 MI Regarding ECHO Findings

ECHO Findings		Type 1 MI No. = 262	Type 2 MI No. = 88	Test value	*P* value	Sig.
EF	Normal range: 52%–72%	122 (46.6%)	70 (79.5%)	37.601*	<0.001	HS
	Mildly abnormal:41%–51%	70 (26.7%)	18 (20.5%)			
	Moderately abnormal: 30%–40%	36 (13.7%)	0 (0.0%)			
	Severely abnormal: < 30%	34 (13.0%)	0 (0.0%)			
Diastolic Dysfunction	No	17 (6.5%)	0 (0.0%)	20.978*	<0.001	HS
	Grade I	209 (79.8%)	88 (100.0%)			
	Grade II	36 (13.7%)	0 (0.0%)			
	Grade III	0 (0.0%)	0 (0.0%)			
SWMA	Normal	34 (13.0%)	70 (79.5%)	139.772*	<0.001	HS
	Positive	228 (87.0%)	18 (20.5%)			
MR	Normal	192 (73.3%)	88 (100.0%)	29.389*	<0.001	HS
	Grade I MR	34 (13.0%)	0 (0.0%)			
	Grade II MR	36 (13.7%)	0 (0.0%)			
	Grade III MR	0 (0.0%)	0 (0.0%)			
	Grade IV MR	0 (0.0%)	0 (0.0%)			

MI: myocardial infarction, EF: ejection fraction, ECHO: echocardiogram, DD: diastolic dysfunction, SWMA: segmental wall motion abnormality, MR: mitral regurgitation

Table 5: Distribution of Type MI 2 Regarding the Dx (Triggering) Disease

Dx (Triggering)	Type 2 MI (No. = 88)
HTN crisis	17 (19.3%)
Bradycardia	17 (19.3%)
Tachyarrhythmia	12 (13.6%)
Acute bleeding/anemia	11 (12.5%)
Sepsis	10 (11.4%)
CKD	10 (11.4%)
Heart Failure	6 (6.8%)
Respiratory failure	5 (5.7%)

MI: myocardial infarction, Dx: diagnosis, HTN: hypertension, CKD: chronic kidney disease

Table 6: Comparisons Between Type I MI and Type 2 MI Concerning Hospital Stays and MACE at 1 and 6 Months

		Type 1 MI No. = 262	Type 2 MI No. = 88	Test value	*P* value	Sig.
Hospital Stays, d	Median (IQR)	5 (4 – 8)	12 (10 – 15)	-12.429≠	<0.001	HS
	Range	2 – 10	7 – 15			
MACE at 1 month	No MACE	181 (69.1%)	59 (67.0%)	0.127*	0.722	NS
	Death	13 (5.0%)	9 (10.2%)	3.100*	0.078	NS
	Recurrent MI	15 (5.7%)	5 (5.7%)	0.000*	1.000	NS
	Congestive HF	30 (11.5%)	8 (9.1%)	0.379*	0.538	NS
	Stroke	7 (2.7%)	5 (5.7%)	1.803*	0.179	NS
	Target vessel revascularization	16 (6.1%)	2 (2.3%)	1.985*	0.159	NS
MACE at 6 months	No MACE	197 (75.2%)	63 (71.6%)	0.447*	0.504	NS
	Death	15 (5.7%)	12 (13.6%)	5.791*	0.016	S
	Recurrent MI	16 (6.1%)	2 (2.3%)	1.985*	0.159	NS
	Congestive HF	20 (7.6%)	6 (6.8%)	0.064*	0.800	NS
	Stroke	1 (0.4%)	3 (3.4%)	5.344*	0.021	S
	Target vessel revascularization	13 (5.0%)	2 (2.3%)	1.161*	0.281	NS

MI: myocardial infarction, MACE: major adverse cardiovascular events; HF: heart failure, IQR: interquartile range

DISCUSSION

Our study revealed a statistically significant difference between type 1 MI and type 2 MI vis-à-vis age and diabetes, and a highly statistically significant difference concerning sex, smoking status, history of ischemic heart disease, history of congestive heart failure, family history, and hypertension.

Consistent with our findings, Singh et al [2] reported that patients with type 2 MI and myocardial injury were younger and more likely to be female than those with type 1 MI. Additionally, there was a significant reduction in the prevalence of traditional cardiovascular risk factors, such as diabetes, hyperlipidemia, hypertension, and smoking ($P < 0.001$ for all), among patients with type 2 MI and myocardial injury. Contrary to our study, Wereski et al [7] reported similar prevalence rates of known coronary disease (56% vs 58%), hyperlipidemia (63% vs 67%), cerebrovascular disease (12% vs 13%), and diabetes mellitus (22% vs 21%) in patients with type 1 MI or type 2 MI. This finding was attributed to the presence of multiple cardiovascular disease risk factors in patients admitted with type 2 MI. Nonetheless, patients with type 2 MI were found to be slightly older, more likely to be female, and had a higher likelihood of impaired renal function (44% vs 39%) than those with type 1 MI. In our study, the triggering diseases for type 2 MI included hypertensive crisis in 17 patients (19.3%), bradycardia in 17 (19.3%), tachyarrhythmia in 12 (13.6%), acute bleeding/anemia in 11 (12.5%), sepsis in 10 (11.4%), chronic kidney disease in 10 (11.4%), heart failure in 6 (6.8%), and respiratory failure in 5 (5.7%). Chiming with our study, DeFilippis et al [8] reported that physician panels identified hypertension, arrhythmias, infections, severe anemia, surgery, renal failure, and heart failure as causative factors for type 2 MI in multiple investigations. In contrast to our study, Coscia et al [9] reported different underlying pathophysiological mechanisms among 251 patients with type 2 MI. The most common mechanisms were tachyarrhythmia (53.8%), bradyarrhythmia (4%), hypertension (18.7%), hypotension (1.6%), anemia (4.4%), hypoxemia (3.6%), coronary artery dissection (1.2%), coronary artery spasm (5.6%), coronary embolism (0.8%), multiple triggers (2.8%), and unknown causes (3.6%).

A highly statistically significant difference was observed between type 1 MI and type 2 MI in terms of ECG changes and heart rate. Similar findings were reported by Baron et al, [10] who found a significant association between type 2 MI and higher rates of normal ECG,

sinus tachycardia, and atrial fibrillation. In contrast, type 1 MI was more frequently associated with ST-segment elevation and depression, as well as bundle branch block.

In contrast to our study, Nestelberger et al [11] reported that approximately 51% of patients with type 1 MI and type 2 MI had normal ECG findings. Nevertheless, they also found that ST depression, ST elevation, bundle branch block, and T-wave inversion were more common among patients with type 1 MI, with no significant differences between groups except for ST-segment elevation. A highly statistically significant difference was observed between type 1 MI and type 2 MI in terms of echocardiographic findings, including ejection fraction, diastolic dysfunction, segmental wall motion abnormalities, and mitral regurgitation.

Consistent with our study, Gaggin et al [12] found that left ventricular ejection fraction was significantly lower in patients with type 1 MI (50.4 ± 17.2) than in those with type 2 MI (56.6 ± 15.3) ($P < 0.001$).

Contrary to our study, Lopez-Cuenca et al [13] reported no significant difference in left ventricular ejection fraction between type 1 MI (54 ± 13) and type 2 MI (56 ± 15) (P 0.172). The authors suggested that this similarity could be due to regional wall motion abnormalities in type 2 MI caused by factors such as dissection, spasm, embolization, or supply/demand mismatch in the context of fixed obstructive coronary artery disease, which may limit the utility of echocardiography in distinguishing between some type 2 MI and type 1 MI cases.

In our study, coronary angiography, PCI, and CABG were performed in 23.7%, 64.9%, and 14.9% of patients, respectively. A significantly higher proportion of patients with type 2 MI underwent coronary angiography (35.2% vs 19.8%) and PCI (80.2% vs 19.3%), while CABG was exclusively performed in patients with type 1 MI (19.8% vs 0.0%). Consistent with our study, Coscia et al [9] reported a significant difference in the treatment approach for patients with type 2 MI and type 1 MI. CABG or PCI was performed in 75.2% of patients with type 1 MI and only 3.6% of patients with type 2 MI ($P < 0.001$). A highly statistically significant difference was observed between type 1 MI and type 2 MI in terms of hospital stays. No statistically significant difference was found regarding MACE at 1 month and 6 months, except for death and stroke, where a significant difference was noted.

In agreement with our study, the US Affairs cohort study by DeFilippis et al [8] reported that patients with type 2 MI had a median hospital stay of 10 days, which was twice as long as those with type 1 MI (4 days). During an average 1.8-year follow-up, similar readmission rates were found for type 2 MI (43%) and type 1 MI (42%).

Contrary to our study, Smilowitz et al [14] reported no significant difference in all-cause mortality between patients with type 1 MI and type 2 MI during a median follow-up of 1.82 years (IQR: 0.97–2.14), with 138 deaths (30.1%) occurring in total. The authors suggested that misclassification bias might account for this discrepancy, particularly in the context of type 2 MI. They also noted a reduced use of echocardiography in cases of myocardial injury, which could indicate a missed opportunity to identify new wall motion abnormalities consistent with type 2 MI.

CONCLUSIONS

Type 1 MI is more prevalent than type 2 MI; however, type 2 MI is associated with higher mortality rates and longer hospital stays. Type 2 MI typically affects younger individuals and is more common in females. The clinical presentation differs between the 2 types, with type 1 MI patients predominantly experiencing chest pain, while type 2 MI patients present mainly with dyspnea, syncope, and hemodynamic

instability. The management strategies also differ significantly in that cardiac interventions primarily target type 1 MI patients. All-cause mortality is linked to type 2 MI due to its association with multiple comorbidities, such as hypertension, anemia, sepsis, heart failure, and respiratory failure.

Conflict of Interest

The authors declare that they have no conflicts of interest.

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