



Frequency and Outcome of Myocardial Infarction with Non-Obstructive Coronary Arteries (Minoca) at Tertiary Care Hospital

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ABSTRACT

Background: It is defined as the presence of positive cardiac biomarker with clinical evidence of infarction, absence of ($\geq 50\%$) stenosis in any epicardial coronary arteries on coronary angiography, and lack of any alternative diagnosis for the index presentation. MINOCA tends to affect younger females more frequently. **Objective:** To determine the frequency of myocardial infarction with non-obstructive coronary arteries. To determine the frequency of outcome (cardiogenic shock, ventricular arrhythmias, mechanical complications like papillary muscle rupture in patients having myocardial infarction with non-obstructive coronary arteries (MINOCA). **Material and Methods:** The cross-sectional study enrolled 164 patients ranging from 18–75 years of age, undergoing coronary angiography for myocardial ischemia. MINOCA was identified by absence of $\geq 50\%$ stenosis on angiography without an alternative diagnosis. Statistical analysis was performed using SPSS Version 25 with Shapiro-Wilk test for normality and Chi-square or Fisher's exact test for categorical variables, considering a p-value of ≤ 0.05 as significant. **Results:** In our study, mean age was 60 ± 9.83 years, 59.8% male, 70.7% urban residents. MINOCA was diagnosed in 39% of cases, with Mild luminal irregularities in 7.3% and moderate coronary lesions in 31.7%. Adverse outcomes were notable, with cardiogenic shock observed in 11%, ventricular arrhythmias in 4.3%, mechanical complications in 3.7%, and mortality in 2.4% of patients. Multivariate analysis revealed that age ≤ 50 years, urban residency, and poor glycemic control independently predicted adverse outcomes. **Conclusion:** Our study demonstrates that MINOCA is prevalent in our cohort (39%). Cardiogenic shock occurred in 11% patients, ventricular arrhythmias in 4.3%, mechanical complications (3.7%), and mortality was 2.4%

INTRODUCTION

Acute myocardial infarction (MI) is caused by unstable atherosclerotic plaque with subsequent plaque rupture and thrombus formation, leading to acute coronary obstruction and myocardial ischemia. (1,2). This pathophysiology predominantly manifests as Acute MI, known as "myocardial infarction with obstructive coronary artery disease" (MI-CAD) (3). In recent years, another entity, termed "myocardial infarction with non-obstructive coronary arteries" (MINOCA), has gained recognition. Diagnostic criteria for MINOCA, outlined in guidelines from the US, Europe, and Japan, include Acute MI defined by the fourth universal definition of myocardial infarction (MI) criteria, non-obstructive coronary arteries on angiography (no stenotic lesion $\geq 50\%$ in epicardial coronary arteries), in the absence of alternative diagnoses (e.g., sepsis, pulmonary embolism, myocarditis, and Takotsubo Cardiomyopathy) (4,5).

Unfortunately, MINOCA relates to various conditions that includes coronary microcirculation dysfunction, coronary artery bridging or plaque erosion (6). MINOCA

was first documented more than 75 years ago on the autopsy of a patient with MI, who was found to have myocardial necrosis in the absence of significant coronary artery disease (7,8). Myocardial infarction (MI) in patients without significant coronary artery disease (CAD) has been reported since 1939, when Harry Gross and William Sternberg described its occurrence as "not rare" (9).

For many years after its first clinical description, MINOCA was a loosely defined clinical entity without a standard nomenclature or widespread acceptance. The term MINOCA was coined in 2013 by Dr. John Beltrame (10). Evidence based studies, consensus documents, clinical practice guidelines from multiple cardiovascular societies, and the Fourth Universal Definition of MI have aligned to establish uniform diagnostic criteria for MINOCA to differentiate it from MI with obstructive CAD (MI-CAD) (11). Globally, MINOCA constitutes between 5-25% of patients presenting with an acute myocardial infarction (MI) (11). MINOCA occurs more frequently in whites and young females. Etiology includes coronary artery spasm, spontaneous coronary artery dissection,

plaque disruption or erosion, and microvascular dysfunction (2).

In-hospital mortality associated with MINOCA is ~1% (12). Post-discharge prognosis of patients with MINOCA is worse as compared to patients without MI, but favorable as compared to that of MI-CAD (12,13). Outcomes of patients with MINOCA having nonobstructive coronaries were poor than that of angiographically normal coronary arteries (13). Among individuals age 65 and older, the risk of adverse outcomes is still higher, with up to 12% mortality at 1 year follow-up (14). In another study, a total of 6,812 patients underwent angiography for MI out of which 645 (9.5%) were diagnosed with MINOCA (15).

Although the literature on this subject is growing rapidly, some clinicians still believe that the absence of obstructive CAD excludes MI, posing “a diagnostic and therapeutic dilemma”, and consequently many MINOCA patients are discharged without an appropriate therapeutic strategy, which should be targeted according to the underlying cause. No local study was found aiming to assess frequency of MINOCA. Therefore, this study is planned to determine the frequency of MINOCA in local population. Further we will also determine the frequency of outcomes like cardiogenic shock, ventricular arrhythmias, mechanical complications like papillary muscle rupture, and mortality, among these patients.

MATERIAL AND METHODS

This study was conducted as a prospective observational study at the Department of Cardiology, Liaquat National Hospital and Medical College, Karachi. The study was carried out over a period of six months with the approval of the research synopsis by the Institutional Ethical Review Committee and the College of Physicians and Surgeons of Pakistan. A total of 164 patients were enrolled in the study, with the sample size calculated based on the prevalence of MINOCA 9.5% (15) and a margin of error of 4.5%, using WHO software for sample size calculation at a 95% confidence level. The patients were selected using a non-probability consecutive sampling method.

The patients of both genders aged 18 to 75 years, who had undergone coronary angiography for myocardial infarction were included. Patients with a history of cardiomyopathy, non-ischemic myocardial injury (such as myocarditis, Tako-Tsubo syndrome, sepsis, renal failure, heart failure, and pulmonary embolism), non-cardiac origin of the clinical picture (such as anemia, renal failure, pulmonary embolism, pneumonia, and respiratory failure), or prior coronary artery bypass grafting (CABG) were excluded from the study.

In current study, acute MI was labelled based on the detection of a rise or fall in cardiac troponin (cTn) with at least one value exceeding the 99th percentile upper reference limit, along with clinical evidence of infarction. This clinical evidence included symptoms such as chest pain, shortness of breath, chest heaviness, epigastric pain, and diaphoresis, in addition to any of the following: new ischemic electrocardiographic changes, the development of pathological Q waves, imaging evidence of new loss of viable myocardium or regional wall motion abnormality, or identification of coronary artery disease on angiography.

Coronary artery disease was classified based on angiographic findings. The non-obstructive coronary arteries (NOCA) were defined as any major epicardial vessel with stenosis less than 50%, including normal coronary arteries (no angiographic stenosis), mild luminal irregularities (angiographic disease <30% stenosis), and moderate coronary atherosclerotic lesions (stenosis >30% but <50%). MINOCA was defined as the presence of positive cardiac biomarkers (troponin I) with clinical evidence of infarction, the absence of stenosis \geq 50% in any epicardial coronary arteries on coronary angiography, and the lack of any alternative diagnosis for the index presentation. MINOCA patients were further categorized according to angiographic findings as having normal coronary arteries, mild irregularities, or moderate coronary atherosclerosis.

Co-morbidities such as diabetes mellitus (DM), hypertension, and obesity were also defined in the study. DM was considered present in patients using oral hypoglycemics for at least three months. Hypertension was defined as a patient using anti-hypertensive drugs for the past three months. Obesity was identified based on a body mass index (BMI) greater than 25 kg/m². Smoking was considered positive in patients who smoked 10 or more cigarettes per day for at least one year, including ex-smokers who had not abstained for more than a month. A positive family history of coronary artery disease (CAD) was recorded if any immediate family member had a history of CAD.

Outcomes of interest in this study included cardiogenic shock, ventricular arrhythmias, mechanical complications, and mortality. Cardiogenic shock was defined by the presence of hypotension (systolic blood pressure <90 mmHg for more than 30 minutes or requiring pharmacological or mechanical support to maintain a systolic blood pressure >90 mmHg), along with end-organ hypoperfusion as indicated by altered mental status, cool extremities, lactate >2.0 mmol/L, or urine output <30 mL/h. Ventricular arrhythmias were diagnosed based on ECG findings of broad QRS complex tachycardia. Mechanical complications were considered if patients developed acute mitral regurgitation secondary to papillary muscle rupture, ventricular septal rupture, pseudoaneurysm, or free wall rupture. Mortality was defined as the cessation of the heart's ability to pump blood and cessation of brain activity, resulting in the patient's death.

Data collection was carried out by obtaining baseline clinical characteristics, including age, sex, and socio-demographic data, along with details of diabetes mellitus, hypertension, coronary artery disease history, and smoking history. Blood samples were collected to assess cardiac troponin T (cTnT) levels, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), lipid protein a (Lp-a), alanine aminotransferase (ALT), aspartate aminotransferase (AST), uric acid (UA), serum creatinine (SCr), fasting blood glucose, glycosylated hemoglobin, and N-terminal pro-brain natriuretic peptide (NT-proBNP). Findings from transthoracic echocardiography (TTE) and coronary angiography were also documented. Special attention was given to assessing

coronary artery spasm, spontaneous coronary artery dissection, plaque disruption or erosion, microvascular dysfunction, and outcomes such as cardiogenic shock, ventricular arrhythmias, mechanical complications, and mortality among MINOCA patients. All data were recorded on a predesigned proforma.

The analysis was performed using SPSS Version 25. The normality of the data was assessed using the Shapiro-Wilk test. Descriptive statistics such as mean±standard deviation (SD) were calculated for quantitative variables, including age, height, weight, BMI, cardiac biomarkers, and lipid profiles. For non-normally distributed data, median and interquartile range (IQR) were used. Frequency and percentages were calculated for qualitative variables, including gender, comorbidities, and coronary artery classifications. Stratification was used to control for potential confounding variables, and post-stratification analysis was conducted using the Chi-square test or Fisher's exact test, as appropriate. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

The **Table-1** presents a detailed demographic, clinical, and laboratory profile of the study population, providing insights into their cardiovascular risk factors, biochemical markers, and coronary angiogram findings. Among 164 patients, the mean age was 60 ± 9.83 years with a predominance of older individuals (81.1% >50 years) and males (59.8%). Most patients (70.7%) were urban residents, and a considerable proportion had cardiovascular risk factors—diabetes in 58.5%, hypertension in 62.8%, obesity in 18.3% and 47% were smokers, with 39% reporting a family history of CAD. Biochemical markers revealed a mean troponin level of 3.19 ± 2.11 ng/mL indicating myocardial injury, total cholesterol of 213.32 ± 39.48 mg/dL, triglycerides of 138.96 ± 31.72 mg/dL, and suboptimal glycemic control with a mean HbA1C of $6.96 \pm 0.98\%$. Coronary angiography demonstrated that only 7.3% of patients had mild luminal irregularities while 31.7% exhibited moderate atherosclerotic lesions and 61% had significant stenosis; 39% were diagnosed with MINOCA, highlighting myocardial infarction in the absence of coronary artery obstruction.

The **Figure-1** illustrates the distribution of adverse outcomes among 164 patients, highlighting the occurrence of cardiogenic shock, ventricular arrhythmias, mechanical complications, and mortality. Cardiogenic shock is the most prevalent complication, affecting 18(11%) patients. This significantly higher percentage suggests that cardiogenic shock is a major clinical concern among the observed complications, requiring urgent intervention and revascularization. Ventricular arrhythmias were reported in 7 (4.3%) patients. Although less common than cardiogenic shock, ventricular arrhythmias still pose a notable risk. Similarly, mechanical complications were observed in 6 (3.7%) patients. These complications, though less frequent, may lead to significant morbidity and necessitate careful monitoring and management. Mortality occurred in 4 (2.4%) patients. While this percentage is relatively low as compared to other complications, it

highlights the importance of timely interventions to prevent fatal outcomes.

The **Figure-2** provides an overview of the mechanical obstruction of coronary arteries in patients. The are categorized as mild luminal irregularities (<30% disease), moderate coronary atherosclerotic lesions (30-50% disease), stenosis (>50% disease). Mild luminal irregularities were observed in 12 (7.3%) patients. None of the patients with biochemical evidence of ischemia had normal coronaries. Moderate coronary atherosclerotic lesions were the most frequently occurring condition, affecting 52 (31.7%) patients. Overall, the chart highlights that >50% stenosis was the most common angiographic finding among the patients, followed by moderate lesions.

The comparative analysis in **Table-2** reveals several noteworthy differences between MINOCA and non-MINOCA patients. MINOCA patients were significantly younger, with a mean age of 57.42 ± 10.84 years compared to 62.98 ± 7.56 years in non-MINOCA individuals ($p < 0.001$), and a higher proportion of MINOCA patients were aged ≤ 50 years (28.4% vs. 7.9%, $p < 0.001$). Although there was a trend toward a higher male representation in the MINOCA group (64.8% vs. 53.9%), this difference was not statistically significant ($p = 0.159$). Additionally, MINOCA patients had a lower mean BMI (27.44 ± 2.73 kg/m² vs. 28.42 ± 3.01 kg/m², $p = 0.031$) and were more likely to reside in urban areas (79.5% vs. 60.5%, $p = 0.008$). In terms of comorbidities, diabetes mellitus was significantly less prevalent among MINOCA patients (44.3% vs. 75%, $p < 0.001$), and they also had lower rates of obesity (10.2% vs. 27.6%, $p = 0.004$). Hypertension prevalence was similar across groups (61.4% vs. 64.5%, $p = 0.681$), while smoking and family history of CAD did not differ significantly between the groups. Biochemically, MINOCA patients showed a trend toward lower troponin levels (2.91 ± 1.96 vs. 3.52 ± 2.23 ng/mL, $p = 0.069$) and had significantly lower triglyceride levels (134.26 ± 22.72 vs. 144.42 ± 39.14 mg/dL, $p = 0.049$). Although LDL cholesterol was lower in the MINOCA group (124.79 ± 23.15 vs. 132.01 ± 31.00 mg/dL), the difference was not statistically significant ($p = 0.098$). Furthermore, renal function appears better in MINOCA patients, as indicated by lower serum creatinine levels (1.13 ± 0.24 vs. 1.26 ± 0.41 mg/dL, $p = 0.013$). Importantly, glycemic control was significantly superior in MINOCA patients, reflected by lower mean HbA1C levels (6.77 ± 0.99 vs. $7.19 \pm 0.93\%$, $p = 0.006$), though fasting blood glucose levels did not differ significantly ($p = 0.118$). These findings suggest that MINOCA patients tend to be younger, with a lower metabolic risk profile and better renal and glycemic parameters, factors that may influence their clinical outcomes.

The **Table-3** provides a comprehensive comparison between patients with and without cardiogenic shock and ventricular arrhythmias, highlighting several important clinical differences. Patients with cardiogenic shock were significantly older (mean age 64.38 ± 6.52 years vs. 59.45 ± 10.05 years, $p = 0.009$) and all were above 50 years, compared to 78.8% in the non-shock group ($p = 0.026$). Although gender differences were not statistically significant, a higher proportion of shock patients were male (72.2% vs. 58.2%). Rural residence

was markedly more common in those with cardiogenic shock (66.7% vs. 24.7%, $p < 0.001$), which may reflect disparities in access to care or other socioeconomic factors. In terms of comorbidities, diabetes mellitus (83.3% vs. 55.5%, $p = 0.024$), obesity (44.4% vs. 15.1%, $p = 0.006$), and a family history of CAD (66.7% vs. 35.6%, $p = 0.011$) were significantly more prevalent among patients with shock. Biochemical markers further distinguished the shock group, as evidenced by significantly higher troponin levels (4.34 ± 2.62 vs. 3.05 ± 2.00 ng/mL, $p = 0.049$) and poorer glycemic control indicated by elevated HbA1C ($7.40 \pm 0.85\%$ vs. $6.91 \pm 0.99\%$, $p = 0.046$). In contrast, while the mean age did not differ significantly for patients with ventricular arrhythmias, these individuals had a significantly higher BMI (32.60 ± 4.77 vs. 27.69 ± 2.62 kg/m², $p = 0.035$), a higher prevalence of rural residence (71.4% vs. 27.4%, $p = 0.023$), and notably, all patients with ventricular arrhythmias were smokers compared to 44.6% in the non-arrhythmia group ($p = 0.004$). Other parameters, including lipid profiles, serum creatinine, and fasting blood glucose, showed no significant differences between the respective groups for both adverse outcomes. These detailed comparisons suggest that older age, rural residence, and a greater burden of metabolic and cardiovascular risk factors are strongly associated with cardiogenic shock, while ventricular arrhythmias are notably linked to increased BMI and smoking status, underscoring the need for targeted risk assessment and management strategies in these patients.

The **Table-4** presents a comparative analysis of patients with and without mechanical complications and mortality. Patients with mechanical complications did not differ by gender from those without (66.7% vs. 59.5% male, $p = 1.000$), whereas mortality was significantly associated with female gender (100% of deceased were female, $p = 0.025$). Although the mean age was marginally higher in those with complications (61.66 ± 8.68 vs. 59.93 ± 9.89 years, $p = 0.674$), deceased patients were significantly older (64.50 ± 0.577 vs. 59.88 ± 9.93 years, $p < 0.001$). Both mechanical complications and mortality were strongly linked to higher BMI (31.66 ± 0.51 vs. 27.75 ± 2.85 kg/m², $p < 0.001$ for complications; 32.50 ± 0.57 vs. 27.78 ± 2.84 kg/m², $p = 0.001$ for mortality) and rural residence (100% of complications and deceased patients resided in rural areas compared to 73.4% and 27.5% of their counterparts, $p < 0.001$ and $p = 0.007$, respectively). In addition, mechanical complications were significantly associated with diabetes (100% vs. 57%, $p = 0.042$), obesity (100% vs. 15.2%, $p < 0.001$), higher total cholesterol (246.66 ± 44.12 vs. 212.06 ± 38.88 mg/dL, $p = 0.035$), elevated fasting blood glucose (164.33 ± 6.59 vs. 133.64 ± 23.57 mg/dL, $p = 0.002$), increased serum creatinine (1.63 ± 0.33 vs. 1.17 ± 0.32 mg/dL, $p = 0.001$), and poorer glycemic control (HbA1C $7.93 \pm 0.10\%$ vs. $6.93 \pm 0.98\%$, $p < 0.001$). Similarly, mortality was linked to significantly higher total cholesterol (255.00 ± 28.86 vs. 212.28 ± 39.21 mg/dL, $p = 0.032$) and HbA1C levels ($8.00 \pm 0.00\%$ vs. $6.94 \pm 0.98\%$, $p < 0.001$), while smoking was notably absent among those with mechanical complications (0% vs. 48.7% in the non-complication group, $p = 0.030$).

The **Table-5** summarizes the unadjusted and adjusted odds ratios for various factors associated with clinical outcomes. In the unadjusted analysis, age ≤ 50 years was associated with a significantly higher risk (OR = 4.63, 95% CI: 1.784–12.017, $p = 0.002$), which remained significant even after adjustment (adjusted OR = 3.67, 95% CI: 1.196–11.255, $p = 0.023$). Urban residence also emerged as a significant risk factor, with urban dwellers having an unadjusted OR of 2.54 (95% CI: 1.269–5.070, $p = 0.008$) that increased to an adjusted OR of 2.97 (95% CI: 1.245–7.065, $p = 0.014$). Notably, diabetes mellitus showed a strong inverse association, with an unadjusted OR of 0.265 ($p < 0.001$) that became even more pronounced after adjustment (adjusted OR = 0.056, $p < 0.001$), suggesting that patients without diabetes had a significantly higher likelihood of adverse outcomes. Obesity was protective in the unadjusted model (OR = 0.298, $p = 0.005$) but lost significance after adjustment (OR = 0.412, $p = 0.076$), and while lipid parameters, hypertension, smoking, family history of CAD, troponin, and fasting blood glucose did not show significant associations, HbA1C demonstrated an interesting reversal—from a protective effect in the unadjusted analysis (OR = 0.637, $p = 0.006$) to a significant risk factor in the adjusted model (adjusted OR = 2.79, $p = 0.013$). These findings underscore that younger age, urban residence, and higher HbA1C levels are independently associated with increased risk, whereas diabetes appears to confer a protective effect, highlighting the importance of multivariate adjustments in elucidating these complex relationships.

Table 1
Demographic, Clinical, and Biochemical characteristics of study participants

	n (%)
Gender	
Male	98(59.8)
Female	66(40.2)
Age (years) ↓	60.00±9.83
Age Group	
≤50 years	31(18.9)
>50 years	133(81.1)
Weight(kg) ↓	74.76±6.73
Height(cm) ↓	160.96±6.05
Body Mass Index (kg/m²) ↓	27.9±2.9
Residence	
Urban	116(70.7)
Rural	48(29.3)
Co-Morbids	
Diabetes Mellitus	96(58.5)
Hypertension	103(62.8)
Obesity	30(18.3)
Smoking	77(47)
Family History of CAD	64(39)
Troponin(ng/mL) ↓	3.19±2.11
Total Cholesterol(mg/dL) ↓	213.32±39.48
Triglyceride(mg/dL) ↓	138.96±31.72
LDL (mg/dL) ↓	128.14±27.23
Serum Creatinine(mg/dL) ↓	1.19±0.33
Fasting blood glucose(mg/dL) ↓	134.76±23.87
HbA1C (%) ↓	6.96±0.98
Findings	
Mild luminal irregularities	12(7.3)
Moderate coronary atherosclerotic lesions	52(31.7)
Stenosis	100(61)
MI with non-obstructive coronary arteries (MINOCA)	64(39)

↓ mean± std.

Figure 2
Distribution of coronary conditions in MINOCA patients

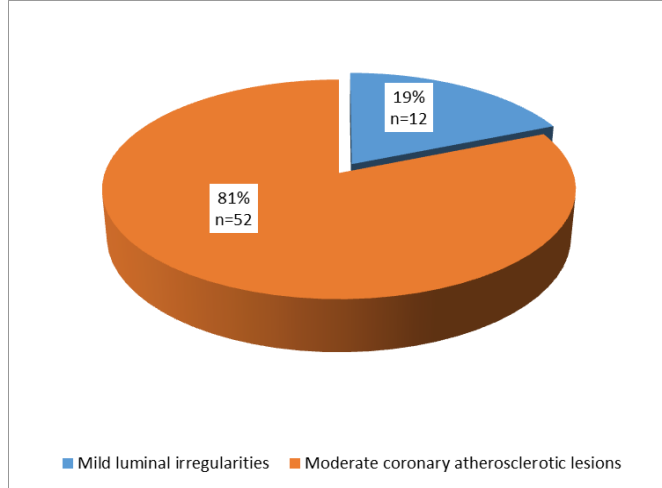


Table 2
Comparison of Clinical and Biochemical Characteristics in Patients With and Without MINOCA

	MINOCA n (%)		p-value
	Yes	No	
Gender			
Male	38(59.4)	60(60)	0.937
Female	26(40.6)	40(40)	
Age (years)↓	56.98±10.98	61.93±8.54	0.003*
Age Group			
≤50 years	20(31.3)	11(11)	0.001*
>50 years	44(68.8)	89(89)	
Body Mass Index(kg/m²) ↓	27.18±1.79	28.36±3.35	0.004*
Residence			
Urban	16(25)	80(80)	<0.001*
Rural	48(75)	20(20)	
Co-Morbid			
Diabetes Mellitus	16(25)	80(80)	<0.001*
Hypertension	42(65.6)	61(61)	0.550
Obesity	4(6.3)	26(26)	0.001*
Smoking	31(48.4)	46(46)	0.760
CAD Family History	20(31.3)	44(44)	0.103
Troponin(ng/mL) ↓	2.79±1.86	3.45±2.22	0.042*
Total Cholesterol(mg/dL) ↓	202.39±32.08	220.33±42.24	0.004*
Triglyceride(mg/dL) ↓	131.01±21.29	144.06±36.07	0.004*
LDL (mg/dL) ↓	120.85±21.29	132.80±27.55	0.006*
Serum Creatinine(mg/dL) ↓	1.07±0.20	1.26±0.38	<0.001*
Fasting blood glucose(mg/dL) ↓	121.95±19.16	142.97±23.04	<0.001*
HbA1C (%) ↓	6.36±0.81	7.35±0.88	<0.001*

Chi-square/fisher exact test was applied. ↓mean± std. Dev; Independent t-test was applied. p-value ≤0.05 were considered as significant. *Significant at 0.05 levels.

Table 3
Multivariate and Univariate Analysis of Predictors of Clinical Outcomes in MINOCA

	Un-Adjusted		Adjusted	
	Odds Ratio (95% CI)	p-value	Odds Ratio (95% CI)	p-value
Gender				
Male	0.974(0.514 -1.847)	0.937		
Female↓	1.000			
Age Group				
≤50 years	3.678(1.620 -8.347)	0.002*	1.860(0.567 -6.104)	0.306

>50 years↓	1.000		1.000	
Residence				
Urban	2.830(1.316 -6.086)	0.008*	3.657(1.237 -10.813)	0.019*
Rural↓	1.000		1.000	
Diabetes Mellitus				
Yes	0.083(0.039 -0.176)	<0.001*	0.021(0.003 -0.156)	<0.001*
No↓	1.000		1.000	
Hypertension				
Yes	1.221(0.635 -2.347)	0.550		
No↓	1.000			
Obesity				
Yes	0.190(0.063 -0.574)	0.003*	0.210(0.044 -1.007)	0.051
No↓	1.000		1.000	
Smoking				
Yes	1.103(0.588 -2.067)	0.760		
No↓	1.000			
CAD Family History				
Yes	0.579(0.299 -1.119)	0.104		
No↓	1.000			
Troponin(ng/mL)	0.853(0.727 -1.002)	0.052		
Total Cholesterol(mg/dL)	0.988(0.979 -0.996)	0.005*	1.009(0.996 -1.023)	0.164
Triglyceride(mg/dL)	0.982(0.969 -0.996)	0.013*	0.980(0.960 -1.000)	0.051
LDL (mg/dL)	0.982(0.970 -0.995)	0.007*	0.997(0.979 -1.016)	0.784
Serum Creatinine(mg/dL)	0.107(0.029 -0.395)	0.001*	0.117(0.017 -0.805)	0.029*
Fasting blood glucose(mg/dL)	0.948(0.928 -0.969)	<0.001*	0.946(0.903 -0.990)	0.946
HbA1C (%)	0.292(0.192 -0.443)	<0.001*	5.449(1.169 -25.413)	0.031*

↓Reference group, CI; Confidence Interval Binary logistic regression was applied. p-value≤0.05 were considered as significant.*Significant at 0.05 levels.

Table 4
Association and comparisons of demographic and clinical parameters with cardiogenic shock and ventricular arrhythmias

	MINOCA n (%)		p-value
	Yes	No	
Cardiogenic Shock			
Yes	3(4.7)	18(18)	0.013*
No	61(95.3)	82(82)	
Ventricular Arrhythmias			
Yes	4(6.3)	7(7)	1.000
No	60(93.8)	93(93)	
Mechanical Complications			
Yes	0(0)	6(6)	0.082
No	64(100)	94(94)	
Mortality			
Yes	1(1.6)	4(4)	0.649
No	63(98.4)	96(96)	

Chi-square/fisher exact test was applied. p-value≤0.05 were considered as significant. *Significant at 0.05 levels.

DISCUSSION

Although the occurrence of an acute myocardial infarction (AMI) without significant coronary artery disease (CAD)

was initially reported almost 80 years ago (16,17), the term MINOCA (myocardial infarction with nonobstructive coronary arteries) has been used only recently to describe these patients (18). Over the past several years, a blossoming body of literature on MINOCA has examined this unique syndrome to guide clinicians caring for such patients (16). MINOCA is a syndrome caused by multiple pathophysiological mechanisms, and due to nonobstructive coronaries, it is often misdiagnosed and not given full attention (19). Various reports show the prevalence of MINOCA with ACS between 1 and 15% (20). To evaluate MINOCA, the European Society of Cardiology published a working paper 2018 that included MINOCA as a type of myocardial infarction (MI) in the Fourth Universal Definition of Myocardial Infarction (21).

The major findings of another study are 1-MINOCA patients represented 1.3% of all subjects; 2-MINOCA patients exhibited distinct characteristics, including younger age, fewer coronary risk factors, a lower incidence of STEMI, lower Killip classification grades, and lower peak CK levels, compared with MI-CAD patients; 3-there was no significant difference in in-hospital all-cause mortality between the MINOCA and MI-CAD groups; 4-however, non-cardiac mortality was notably higher in the MINOCA group; and 5-of particular note, there was a disparity in non-cardiac mortality between elderly and non-elderly patients in the MI-CAD group, but not in the MINOCA group (3). Some reviews have reported a wide range in the prevalence of MINOCA, from 1% to 14%, with an overall calculated incidence of 6% (8). Prior investigations have consistently identified MINOCA patients as typically younger (24,25) and more frequently female (8,20) with lower rates of smoking (24,26) hypertension (26) hyperlipidemia (24-26) and diabetes (25,26) compared with MI-CAD patients.

The gender distribution in a study is in slight favor for women (59.3%). With a median of 74 (60-79) years and average age of 69.8±12.7 years, there were no significant differences in terms of age between the two groups (27). This is consistent with the work published in 2022 by published in 2022 by Lopez-Pais et al. (28). Here, an average age of 64.6±14.9 years or 66.7±13.5 years was described (28). In the VIRGO study, MINOCA was more often found in younger patients; traditional cardiac risk factors than patients with MICAD (29). Contrary to the VIRGO study, the MINOCA patients in the described collective were not significantly younger, which contradicts the assumption that MINOCA is supposedly a disease of younger people.

Troponin is considered an important prognostic marker (30). MINOCA patients have significantly lower concentrations of cardiac necrosis markers as opposed to classical myocardial infarction where the massive release of hs-cTnT (high sensitive cardiac troponin T) occurs as a result of significant damage to the supplied myocardial area due to the immediate total or subtotal occlusion of a coronary artery (30). Moreover, an acute troponin elevation of >5 x URL has a very high positive predictive value for myocardial ischemia compared to troponin elevation ≤3 x URL (31).

Since this is not the case in MINOCA, it can be assumed that there is a lower release of troponin (3). This could

partially explain why MINOCA patients had a lower mortality rate. Although the MINOCA group had no in-hospital deaths, their 30-day mortality rate of 4.2% was significantly lower than the 17.3% observed in the classic myocardial infarction group. However, this mortality rate should still be regarded as substantial and clinically relevant. The study by A study by Bergamaschi et al. (32) reports a mean mortality rate of 8.1% (over a period of 33.7 ± 12.0 months).

Left ventricular ejection fraction at admission was better in the MINOCA group, which could potentially speak for a better long-term prognosis for MINOCA patients. However, it is extremely difficult to make a specific statement about long-term prognosis due to the large heterogeneity of available registries (3). For example, Lopez-Pais et al. (33) describe similar complications such as reinfarction, severe bleeding, stroke, pulmonary edema, or shock in 13.8% of MINOCA patients vs. 17.6% in MICAD (p = 0.335).

Safdar and colleagues (34) demonstrated that in young patients (aged <55 years) presenting with AMI, MINOCA is relatively frequent, occurring in >10% of the population. Although the characteristics of patients with MINOCA and their counterparts with AMI and CAD (AMI-CAD) were different, the mortality rates at 1 month (1.1% versus 0.6%, P=0.43) and 1 year (1.7% versus 2.3%, P=0.68) were not statistically different. Further in the same study it was observed that MINOCA is not an uncommon presentation of AMI. It is more frequent in younger women and usually presents with non-ST segment elevation-myocardial infarction (34). Another study showed that MINOCA is found in roughly 6% of AMI patients (35); however, there is large variability in the reported prevalence, with a range of 3.5% to 15% (36,37), possibly attributable to differences in the studied populations and heterogeneity in its definition. Another study showed that MINOCA is also more common in younger patients and women (35,36).

In another study (16), women with AMI had 5-fold higher odds of having MINOCA than men with AMI, and 1 in 8 women with AMI were found to have MINOCA. It is also noteworthy that in the VIRGO study, all patients with spontaneous coronary artery dissection were categorized as MINOCA.

Elderly patients, often characterized by advanced atherosclerosis, may present with angiographic coronary artery stenosis (≥50%), which could be unrelated to the culprit lesion but still result in a diagnosis of MI-CAD rather than MINOCA. The utilization of intravascular imaging techniques in Japan, such as intravascular ultrasound imaging (IVUS), is notably higher than in other countries (38). This contrasts with usage rates reported in Korea (43-55%) (39) and the US (42.2%) (40). Previous studies have shown varying rates of in-hospital mortality for MINOCA, ranging from approximately 1% to 3% (3), and findings regarding its comparison to MI-CAD have been inconsistent, with some studies reporting lower (24,26), higher (41), or non-significantly different rates (3).

This is a single-center study. The presence of wide standard deviations suggests that while our mean estimates provide a central tendency of expenditure for

MINOCA, there is a substantial variability that needs to be considered when interpreting the results. Other aspects that should be considered as limitations but can be the focus of future studies include: the rate of readmissions/medication compliance /medication rate/quality of life of MINOCA patients.

CONCLUSION

In conclusion, our study demonstrates that myocardial infarction with non-obstructive coronary arteries (MINOCA) is highly prevalent in our cohort, affecting 53.7% of the observed MI cases, and is characterized by a wide spectrum of coronary findings, with moderate

coronary atherosclerotic lesions being most common. Adverse outcomes were notable, with cardiogenic shock occurring in 11% of patients, followed by ventricular arrhythmias (4.3%), mechanical complications (3.7%), and mortality (2.4%). Independent predictors of worse outcomes included younger age (≤ 50 years), urban residence, and poorer glycemic control, underscoring the need for early risk stratification and targeted management strategies.

These findings underscore the urgent need for early risk stratification and individualized management strategies to mitigate complications in MINOCA patients, particularly among those with identifiable high-risk profiles.

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