J Surg Med. 2021;5(1):50-54.
DOI: 10.28982/josam.839523

Research article

Journal of Surgery and Medicine
e-ISSN=2602-2079

Myocardial infarction with non-obstructive coronary artery disease, a retrospective cohort study: Are plaque disruption and other pathophysiological mechanisms the same disease?

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Ethics Committee Approval
University of Health Sciences Gülhane Training and Research Hospital Ethics Committee decision number and date: 19/194, 14/05/2019. All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

Conflict of Interest
No conflict of interest was declared by the authors.

Financial Disclosure
The authors declared that this study has received no financial support.

Published
2021 January 29

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Abstract

Background/Aim: Myocardial infarction with non-obstructive coronary arteries (MINOCA) is an increasingly recognized entity. Recent studies have shown that MINOCA is not a benign syndrome, with younger MINOCA patients having outcomes comparable to their myocardial infarction with obstructive coronary artery disease (MI-CAD) counterparts. In this study, we will describe the demographic, clinical and angiographic characteristics of MINOCA patients in our hospital.
Methods: In this retrospective cohort study, all patients who underwent coronary angiography with the diagnosis of acute coronary syndrome during September 2016-April 2019 were screened and those with MINOCA were detected. We described the demographic, clinical, and angiographic characteristics of MINOCA patients and compared the etiologic and pathophysiological mechanisms.
Results: A total of 3855 patients with acute coronary syndrome were screened and 155 were diagnosed with MINOCA, with a total prevalence of 4.02%. Among them, 48.4% were female and the overall mean age was 55.04 (13.57) years. Plaque disruption was the most common cause of MINOCA (48.4%), which was followed by microvascular dysfunction and slow flow (9.7%). We compared plaque disruption and other causes to find that age (58.31 (13.76) vs 51.89 (12.68) P=0.003), hypertension (37 (48.7%) vs 25 (31.6%) P=0.034), prior coronary artery disease history (16 (21.1%) vs 2 (2.5%) P=0.001) and creatinine clearance (67.35 (IQR: 25.8) vs 74.0 (IQR: 28.58) P=0.009) were higher in patients with plaque disruption than those without.
Conclusions: MINOCA is a diagnosis of exclusion with numerous potential causes. The etiological and pathophysiological mechanisms of plaque disruption are different from other causes of MINOCA and the correct treatment approach determines the prognosis.

Keywords: Microvascular dysfunction, MINOCA, Plaque disruption, SCAD, Vasospasm

How to cite: Asil S, Barış VÖ, Geneş M, Taşkan H, Görmel S, Yıldırım E, Gökoğlan Y, Çelik M, Yüksel UÇ, Kabul HK, Barçın C. Myocardial infarction with non-obstructive coronary artery disease, a retrospective cohort study: Are plaque disruption and other pathophysiological mechanisms the same disease? J Surg Med. 2021;5(1):50-54.

Introduction

Depending on the population examined, around 5%–15% of patients with acute myocardial infarction (MI) present without any significant stenosis (<%50) in their coronary arteries [1-3]. Although this patient group has been known for many years and is mentioned with different names in the literature, it has not received the necessary attention [4, 5]. In 2012, Beltrame suggested that the myocardial infarction with non-obstructed coronary arteries (MINOCA) would be appropriate for identifying this patient group [6]. MINOCA patients have an increased risk of future adverse events comparable to those of MI with obstructive coronary arteries (MI-CAD) patients [7]. A recent meta-analysis reports that the 1-year mortality as high as 4.7% [2, 7]. SWEDEHEART registry data revealed that mortality was 13.4% during a mean follow-up of 4.1 years [8]. All these results show that MINOCA is a quite common disease with high mortality and morbidity in the long term.

In contrast to MI-CAD, the underlying pathophysiology of MINOCA is most likely heterogeneous. Several mechanisms, such as coronary spasm, plaque disruption, a rapidly dissolved thrombus, dissection, microvascular dysfunction, inflammation, and imbalance in myocardial oxygen supply/demand, have been proposed [1-3]. MINOCA patients have a lower prevalence of traditional coronary arteries disease risks factors, such as hypertension (HT), hyperlipidemia (HL), diabetes mellitus (DM), tobacco abuse, and a family history of MI. It is more common in female patients [1-3]. All these factors make diagnosis and treatment of MINOCA challenging in daily clinical practice. The present study aimed to describe the demographic, clinical and angiographic characteristics of MINOCA patients and compare the etiologic mechanism.

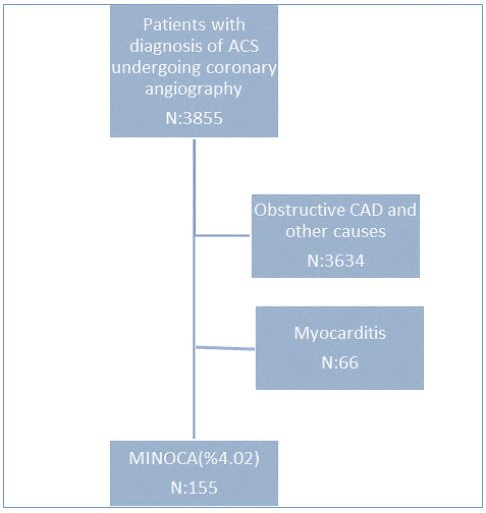
Materials and methods

In this retrospective cohort study, patients who were admitted to the emergency department with chest pain and high cardiac troponin levels between September 2016 and April 2019 and underwent coronary angiography due to acute coronary syndrome were identified from hospital and file records. After excluding patients with obstructive coronary artery disease and other probable causes, MINOCA patients were identified and included in the study. A recent scientific statement from The American Heart Association (AHA) working group has suggested the following diagnostic criteria for MINOCA: (I) Acute myocardial infarction criteria as defined by the “Fourth Universal Definition of Myocardial Infarction [9]” (II) Non-obstructive coronary arteries on angiography, (III) No specific alternate diagnosis for the clinical presentation [3]. Non-coronary diseases, such as pulmonary embolism, myocarditis, Takotsuba cardiomyopathy, which cause cardiac enzyme elevation and chest pain, were excluded from the definition of MINOCA and the study [3]. Patients over 18 years of age with elevated cardiac biomarkers and non-obstructive coronary arteries (<50%) in coronary angiography were included in the study, while patients with other causes of cardiac enzyme elevation like pulmonary embolism, cardiomyopathy, severe renal impairment, aortic dissection, myocarditis and Takotsuba cardiomyopathy, those with sepsis, stroke, multi-organ failure, malignancy or any clinical condition associated with poor prognosis were excluded. All procedures were performed in accordance with the Declaration of Helsinki and the study was approved by the local ethics committee (University of Health Sciences Gülhane Training and Research Hospital Ethics Committee decision number and date: 19 / 194, 14 / 05 / 2019).

All participants’ medical histories, routine laboratory tests and cardiac biomarkers, echocardiography, and electrocardiography (ECG) results were obtained from the hospital database. In our hospital, cardiac magnetic resonance imaging (MRI) is not performed in all patients to exclude patients with myocarditis in the diagnostic algorithm of MINOCA. However, myocarditis is clarified by cardiac MRI in patients who are at elevated risk clinically, electrocardiographically and echocardiographically for the diagnosis of myocarditis.

A total of 3855 acute coronary syndrome patients were investigated. Coronary angiography images were evaluated by at least two specialist cardiologists at our center and patients without occlusive coronary artery disease were identified. The degree of coronary artery stenosis was determined by averaging at least 2 invasive cardiologists' visual evaluation decisions and the quantitative angiography (QCA) measurements. The study flow chart is shown in Figure 1. The pathophysiological mechanisms of MINOCA were investigated from the coronary angiography images of patients. Intravascular ultrasound (IVUS) and optical coherence tomography (OCT) are not routinely used in our center. In the literature, vulnerable plaque morphology was defined angiographically in the studies performed before IVUS and OCT. Based on those studies, we determined that irregular borders or intraluminal lucency, haziness, slowing of the flow rate in the lesion area are characteristic of vulnerable plaque [10, 11]. Routine provocation test is not performed for the detection of epicardial coronary vasospasm, which is diagnosed according to the clinical and angiographic images of the patient.

Figure 1: Study flow chart (ACS: Acute coronary syndrome, CAD: Coronary artery disease, MINOCA: Myocardial infarction with non-obstructive coronary artery disease)



Statistical analysis

Statistical analyses were performed using Statistical Package for Social Sciences (SPSS) statistical software (version 20.0; IBM SPSS Inc., Chicago, IL, USA). Continuous variables were evaluated for normal distribution using the Kolmogorov-Smirnov test. Normally distributed continuous variables were expressed as mean (standard deviation, SD), whereas non-

normally distributed continuous variables were expressed in median (IQR: Inter Quartile Range). Continuous variables were analyzed with sample t-test or Mann-Whitney U test. Categorical variables were expressed as percentages when appropriate and they were compared with chi-square and Fisher exact tests. A *P*-value <0.05 was considered statistically significant.

Results

A total of 3855 acute coronary syndrome patients were evaluated retrospectively and 155 (4.02%) were diagnosed with MINOCA. Myocarditis was also considered in MINOCA in consensus reports published during the period when MINOCA was first described [1, 2, 4]. In our study, when we included myocarditis in our analysis, the total number of patients diagnosed with MINOCA increased to 221 and the frequency increased to 5.73%. However, a recent study suggests that it is not appropriate to include myocarditis in MINOCA, which is why our entire analysis comprises 155 patients [3].

The mean age of our study population was 55.04 (13.57) years, and 48.4% were female. The incidence of HT and DM were 40% and 21.3%, respectively. The median ejection fraction and troponin values of the MINOCA patients were 60 (IQR: 10) and 403 (IQR: 1252), respectively. ST elevation was observed in 5 (3.2%) patients on admission ECG. Eighteen (9.9%) patients had a history of coronary revascularization, all of which had plaque disruption as MINOCA etiology.

There was a total of 5 (3.2%) patients with mechanical prosthesis valves, among which 2 had mitral metallic valves, and 3 had aortic metallic valves. Their coronary angiography results were normal or coherent with slow flow phenomena. They were considered high-risk patients for coronary embolism even though the plaques could not be demonstrated with IVUS and OCT. Three (1.94%) patients were diagnosed with SCAD. The diagnosis and classification of these patients were made according to angiographic images. While two complied with type 2B SCAD class according to the Saw classification, one patient complied with the Type 3 SCAD class and all the patients were medically treated [12, 13].

In-hospital mortality was seen in 1 patient, who had coronary vasospasm and did not respond to medical and invasive treatment, dying at the 6th hour of hospitalization. Details of the demographics and clinical characteristics of the study population are presented in Table 1.

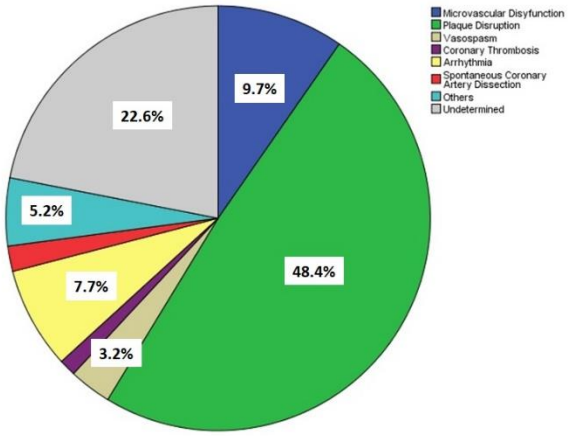
Table 1: Baseline characteristics of the study the population

	All patients (n = 155)
Age, mean (SD)	55.04 (13.57)
Sex (Female) (%)	48.4%
Diabetes Mellitus (%)	21.3%
Hypertension (%)	40%
Cerebrovascular Accident (%)	2.6%
Tobacco Use (%)	9%
Peripheral Vascular Disease (%)	0%
Ejection Fraction (%) Median (IQR)	60 (10)
Creatinine clearance	
MDRD (mL / min / 1.73m2) Median (IQR)	72.6 (28.5)
Wight Blood Cell (x10^3 cells UL) Median (IQR)	9.0 (4.7)
C-Reactive protein (mg / L) Median (IQR)	6.3 (12.15)
Hs Troponin I (pg / ml) Median (IQR)	403 (1252)
Duration of Hospitalization (days) Median (IQR)	1 (1)

MDRD: Modification of Diet in Renal Disease

In our study, based on angiography images, clinical features and laboratory results, plaque disruption was the most common cause of MINOCA (48.4%). The second most common cause was microvascular dysfunction and slow flow (9.7%), followed by arrhythmia (7.7%) The pathophysiology causing cardiac biomarker elevation in 22.4% of patients was not fully understood and classified as undetermined (Figure 2). We decided to compare patients with plaque disruption and other causes. Age (58.31 (13.76) vs 51.89 (12.68) *P*=0.003), HT (37 (48.7%) vs 25 (31.6%) *P*=0.034), prior coronary artery disease history (16 (21.1%) vs 2 (2.5%) *P*=0.001) and creatinine clearance 69.35 (IQR: 25.8) vs 74.0 (IQR: 28.58) *P*=0.009) were higher in patients with plaque disruption than those without. All these parameters were analyzed by multiple logistic regression, and a correlation was found between prior coronary artery disease history and plaque disruption (*P*<0.008, Beta: -2.09, Wald: 6.99).

Figure 2: The causes of MINOCA



DM was insignificantly more prevalent in the group with plaque disruption. Inflammatory markers and ejection fraction were similar between the two groups but Hs troponin I and duration of hospitalization were insignificantly higher in patients with plaque disruption than those without (Table 2).

Table 2: Comparison of plaque disruption and other causes

	Plaque Disruption (n:76)	Others (n:79)	*P*-value
Age, mean (SD)	58.31 (13.76)	51.89 (12.68)	0.003
Female gender, n (%)	34 (44.7%)	41 (51.9%)	0.37
Diabetes Mellitus, n (%)	21 (27.6%)	12 (15.2%)	0.08
Hypertension, n (%)	37 (48.7%)	25 (31.6%)	0.034
Cerebrovascular Accident, n (%)	2 (2.6%)	2 (2.5%)	0.96
Prior Coronary artery disease (%)	16 (21.1%)	2 (2.5%)	0.001
Ejection Fraction (%)			
Median (IQR)	60 (10)	60 (10)	0.37
Creatinine clearance			
MDRD (mL / min / 1.73m2)			
Median (IQR)	69.35 (25.80)	74.0 (28.58)	0.009
Wight Blood Cell (x10^3 cells UL)			
Median (IQR)	9.26 (5.24)	8.90 (3.10)	0.53
C-Reactive protein (mg / L)			
Median (IQR)	9.34 (30.03)	6 (9.51)	0.32
Hs Troponin I (pg / ml)			
Median (IQR)	366 (1281)	208 (686)	0.10
Duration of Hospitalization (days)			
Median (IQR)	2 (1.75)	1 (1)	0.16

Among patients with plaque disruption, 59.6% had only plaques and wall irregularities in their coronary arteries, 6.7% had 30% stenosis and 33.7% had 30-50% stenosis (Figure 3). There were no differences between demographic and clinical features in this sub-group (Table 3).

The coronary angiograms of the patients revealed that 56.1% had plaques and less than 50% stenosis in their coronary arteries. Coronary angiography was normal in 29% of the patients. The diagnosis of normal coronary arteries is used for completely normal coronary arteries without plaque, wall irregularities, slow flow, vasospasm, dissection, and thrombus.

Slow flow was observed in 7.7% of patients, while vasospasm was present in 3.2% (Figure 4). Eighteen patients (11.6%) had a coronary stent or coronary artery bypass grafts, but no occlusive lesions.

Table 3: Comparison of plaque disruption subtypes

	Minimal plaque	30% plaque	30-50% plaque	*P*-value
Age				
Median (IQR)	25 (26)	33	55.5 (19.25)	0.53
Ejection Fraction (%)				
Median (IQR)	60 (5)	40	60 (10)	0.34
Creatinine clearance				
MDRD (mL /min / 1.73m2)				
Median (IQR)	74 (27.95)	89	63.8 (27.3)	0.84
Wight Blood Cell (x10^3 cells UL)				0.22
Median (IQR)	9.0 (4.48)	9.24 (3.87)	9.26 (6.23)	
C-Reactive protein (mg / L)				
Median (IQR)	5.51 (18.64)	33.5	9.4 (35)	0.02
Hs Troponin I (pg / ml)				
Median (IQR)	479 (1302)	1884	667 (1093)	0.37
Duration of Hospitalization (days)				
Median (IQR)	2 (1)	1.5	1 (2)	0.98

Figure 3: Distribution of subtypes of plaque disruption

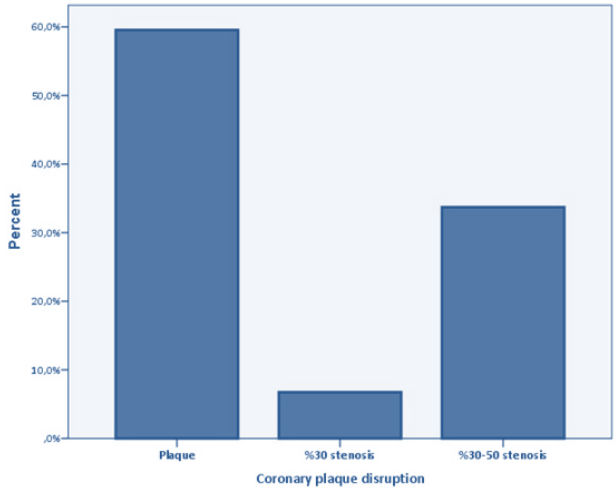
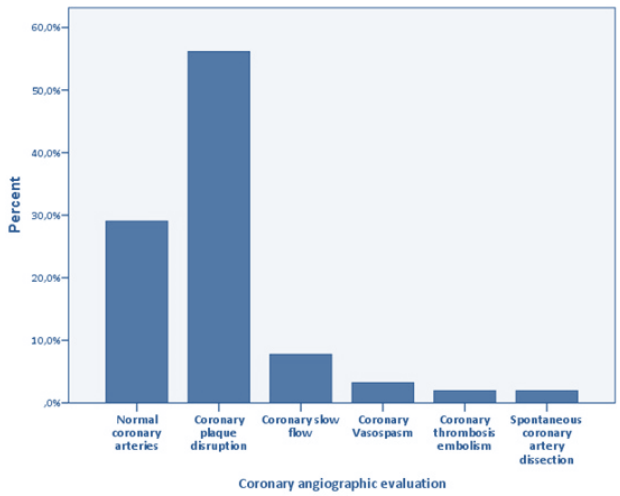


Figure 4: Coronary angiography evaluation results



Discussion

To the best of our knowledge, the present study is the first one to identify basal demographic characteristics of the Turkish MINOCA cohort. We showed that MINOCA patients represent a sizable proportion of MI patients referred for invasive assessment in Turkey and it is prevalence is about 4.02%. The frequency of MINOCA was 5-15% in recent reports, which include both coronary and non-coronary pathologies [2,3,8]. The main reason for the decrease in the frequency of MINOCA is that the disease was clearly defined, and myocarditis and other non-coronary causes were removed from the diagnostic algorithm in the 2019 AHA report [3].

In line with previous reports, we found MINOCA patients to be younger than obstructive coronary artery disease patients. In the current MI-CAD trials, the mean age was 61-62 years [14, 15]. Although female predominance of MINOCA is reported in the literature, the rates were almost equal among males and females in our study [16]. In the study by Jędrychowska et al. [16], no difference was found between the sexes in terms of clinical course and prognosis.

Our study also showed that MINOCA patients had a lower risk profile than other MI-CAD trials [14,15]. However, as we classify our patients into those with plaque disruption and other causes of MINOCA, the risk factors for cardiovascular disease in the plaque disruption group were similar to those of other MI-CAD studies [14,15]. These results showed us that MINOCA due to plaque disruption has similar pathophysiology and risk factors as MI-CAD, and similar medical treatment protocols should be implemented to improve long-term outcomes [3]. In a meta-analysis of MINOCA studies, Pasupathy et al. showed that there was no significant difference in hypertension, diabetes mellitus, tobacco use between MINOCA and MI-CAD patients [2,14,15].

Similar to the literature, the most common cause of MINOCA was plaque disruption in our trial. However, plaque disruption was decided by two invasive cardiologists with the evaluation of ECG, echocardiography, and angiographic images. To clarify the diagnosis and pathophysiology of MINOCA, intracoronary imaging should be performed, but routine uses of IVUS and OCT are not available in our country [17]. If IVUS or OCT could be performed in patients with normal coronary angiography, which were classified as "undetermined", plaque disruption could be detected in many. Opolski et al. [17] showed plaque disruption (24%), coronary thrombus (18%) and eroded plaque (11%) in 53% of MINOCA patients in their study utilizing OCT. Reynolds et al. [18] showed that plaque disruption was present in 38% of their patients in their study using IVUS. OCT provides higher spatial resolution than IVUS, allowing more detailed and complete visualization of plaque pathology [19].

Other rare causes of MINOCA are microvascular dysfunction, slow flow phenomenon, coronary vasospasm, coronary embolism, thrombosis, and arrhythmia attacks. Although spontaneous episodes may be fortuitously documented for diagnosis of coronary vasospasm, provocative spasm testing is often required to establish the diagnosis. In studies using the provocation test for the diagnosis of vasospasm, the frequency increases to 46% [20]. Another rare but significant cause of MINOCA is coronary embolism and thrombosis. A systematic review which examines inherited hypercoagulability state in MINOCA patients showed up to 10% genetic mutation [2]. Therefore, all patients with thrombophilia suspected for clinical and coronary angiographic features should undergo hematological and genetic research.

Considering its clinical findings, cardiac MRI was performed in every patient suspected of myocarditis, and those diagnosed with it were excluded from the study. We cannot

perform cardiac MRI for every MINOCA patient due to logistic limitations in our hospital and country. In a small-scale study of 21 patients, Gościniak et al. [21] performed cardiac MRI in all patients diagnosed with MINOCA to reveal that 38% had myocarditis. In our study, patients strongly suspected of myocarditis were excluded from the study based on cardiac MRI, a procedure all patients did not undergo. In line with these results, performing cardiac MRI could be a major factor affecting the results.

Limitations

Our study has many limitations. First, this was a small, single-center, and retrospective study. The most important limitation is the visual assessment of coronary angiographic images and the lack of IVUS and OCT use. We did not compare our MINOCA data with MI-CAD patients and did not routinely use provocative tests for the diagnosis of vasospasm. Also, hereditary thrombophilia was not evaluated among all patients, but a suspected group. Because of these limitations, there is a margin of error of the data obtained in our study, but we think it is important that it reflects actual Turkish data.

Conclusion

MINOCA constitutes an important proportion of myocardial infarction patients. The pathophysiology of the disease has been better understood with intracoronary imaging (IVUS and OCT) studies. Considering this information in the literature, the diagnosis and treatment continue to be updated. The etiological and pathophysiological mechanism of plaque deterioration are different from other causes of MINOCA and the correct treatment approach determines the prognosis. However, there are many shortcomings in our country. In our study, we wanted to shed light on these points and our hospital cohort of MINOCA.

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