

Myocardial Infarction with Non-Obstructive Coronary Arteries: Clinical Profile, Diagnostic Investigation, and Outcomes in a Tertiary Center Cohort

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Abstract

Background: Myocardial infarction with non-obstructive coronary arteries (MINOCA) represents a heterogeneous clinical syndrome associated with diverse pathophysiological mechanisms and a non-negligible prognosis. Despite conceptual advances, diagnostic uncertainties and significant variations in etiological investigation and clinical management persist in real-world practice, particularly in centers with limited access to advanced imaging modalities.

Objectives: To describe the clinical profile, diagnostic strategies employed, and clinical outcomes of patients with MINOCA followed at a Brazilian tertiary center.

Methods: Observational, retrospective study including consecutive patients diagnosed with MINOCA, defined according to the Fourth Universal Definition of Myocardial Infarction and European guidelines. Clinical, angiographic, and complementary diagnostic data, as well as the treatment instituted and clinical outcomes during follow-up, were collected.

Results: A total of 196 patients were included, with a mean age of 50.6 ± 11.8 years and a slight female predominance (52.6%). Systemic arterial hypertension (60.2%), dyslipidemia (45.9%), and current or former smoking (48.0%) were frequent. Cardiac magnetic resonance imaging was performed in 42.3% of cases, whereas intracoronary imaging methods were rarely used (2.6%). In 40.8% of patients, a definitive etiology could not be established. During follow-up ($n = 183$), mortality was 3.8%, reinfarction occurred in 6.6%, and new cardiovascular hospitalizations occurred in 16.9%.

Conclusions: Patients with MINOCA present an intermediate-risk clinical profile and a relevant incidence of cardiovascular events during follow-up. The high proportion of cases without a defined etiological diagnosis reflects limitations in investigation and reinforces the need for more structured diagnostic strategies and individualized clinical management.

Keywords: MINOCA; Coronary Artery Disease; Myocardial Infarction.

Introduction

Acute myocardial infarction (AMI) without significant obstructive coronary artery disease, known as myocardial infarction with non-obstructive coronary arteries (MINOCA), accounts for approximately 5% to 10% of myocardial infarction (MI) cases undergoing coronary angiography.¹⁻³ For many years, this condition was considered an atypical form of MI or one with a more favorable prognosis. However, evidence accumulated over the past decades has demonstrated that patients with MINOCA carry a relevant clinical risk, with

significant rates of mortality, reinfarction, and cardiovascular hospitalizations.⁴⁻⁶

According to the Fourth Universal Definition of Myocardial Infarction, the diagnosis of MINOCA requires fulfillment of the clinical and laboratory criteria for MI, combined with the absence of coronary stenosis $\geq 50\%$ in major epicardial arteries and the exclusion of alternative diagnoses that could account for the elevation of myocardial necrosis biomarkers.⁷ It is, therefore, a syndromic and exclusionary diagnosis encompassing heterogeneous pathophysiological mechanisms, such as non-obstructive plaque rupture or erosion, coronary thromboembolism, spontaneous coronary artery dissection, epicardial vasospasm, microvascular dysfunction, myocarditis, and Takotsubo cardiomyopathy.⁸⁻¹⁰

Observational studies and population-based registries have demonstrated that patients with MINOCA may present a distinct clinical profile from that observed in MI with obstructive coronary disease, with a relatively higher proportion of women, younger age, and a greater prevalence of non-atherosclerotic mechanisms.¹¹ However,

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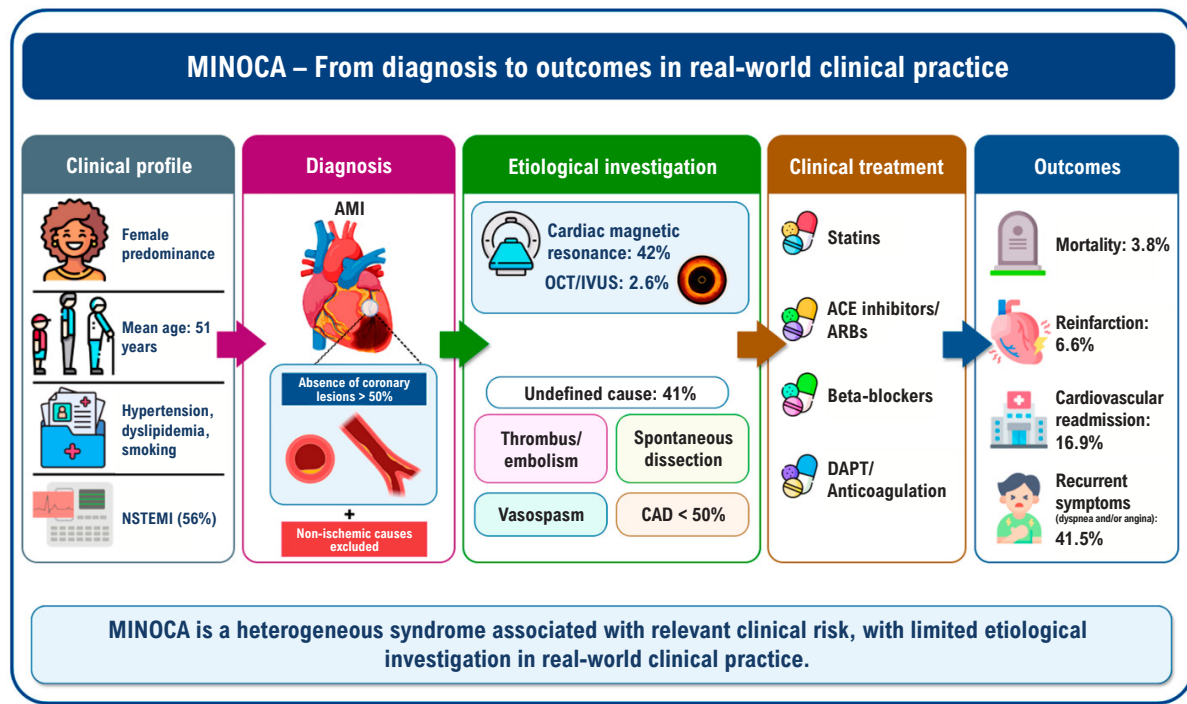
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Central Illustration: Myocardial Infarction with Non-Obstructive Coronary Arteries: Clinical Profile, Diagnostic Investigation, and Outcomes in a Tertiary Center Cohort

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Clinical profile, diagnostic investigation, and clinical outcomes of the evaluated patients. ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; CAD: coronary artery disease; DAPT: dual antiplatelet therapy; IVUS: intravascular ultrasound; MINOCA: myocardial infarction with non-obstructive coronary arteries; NSTEMI: non-ST-elevation myocardial infarction; OCT: optical coherence tomography; AMI: acute myocardial infarction.

traditional cardiovascular risk factors, such as systemic arterial hypertension, dyslipidemia, and smoking, remain prevalent.¹²⁻¹⁴ Furthermore, contemporary data indicate that the prognosis of MINOCA is not as benign as once believed, with rates of major cardiovascular events comparable to those observed in obstructive MI.^{5,14}

Despite conceptual advances, the diagnostic approach to MINOCA remains heterogeneous, with marked difficulty in establishing and adhering to clinical protocols in real-world practice. The systematic use of advanced imaging methods, such as cardiac magnetic resonance imaging (CMR) and intracoronary imaging by optical coherence tomography (OCT) or intravascular ultrasound (IVUS), has been shown to significantly increase the rate of etiological elucidation.¹⁵⁻¹⁹ However, structural and logistical limitations still restrict their routine application, particularly in developing countries.

In this context, the present study aims to describe the clinical characteristics, cardiovascular risk factors, diagnostic strategies employed, and clinical outcomes of a contemporary cohort of patients with MINOCA followed at a Brazilian tertiary center.

Methods

Study design

This was an observational, retrospective study evaluating consecutive patients diagnosed with MINOCA at a tertiary center specializing in cardiology. Adult patients who fulfilled the universal criteria for MI and presented with the absence of coronary stenosis $\geq 50\%$ on coronary angiography performed during the index hospitalization were included.

The sample size was defined by convenience, including all consecutive patients diagnosed with MINOCA identified during the study period.

Diagnostic criteria

The diagnosis of MINOCA followed the recommendations of the European Society of Cardiology and the Fourth Universal Definition of Myocardial Infarction.^{7,20} Inclusion criteria comprised: biochemical evidence of MI; electrocardiographic changes compatible with myocardial ischemia; angiographic stenosis < 50% in all major epicardial coronary arteries; and exclusion of non-ischemic causes of troponin elevation.

Original Article

When clinically indicated, non-ischemic etiologies were excluded by means of CMR, evaluating patterns of edema and late gadolinium enhancement; echocardiography; pulmonary computed tomography angiography to rule out pulmonary embolism; and clinical and laboratory assessment for sepsis.

Patients with inadequate angiographic documentation, confirmed non-ischemic etiology (or insufficient data to exclude it), or prior MI unrelated to the index event were excluded to prevent misclassification.

Data collection

Data were obtained through systematic review of institutional electronic medical records, including hospitalization records, complementary examination reports, procedure reports, and outpatient consultation records. Demographic data, cardiovascular risk factors, clinical history, characteristics of the index event presentation, angiographic findings, complementary diagnostic methods used, treatment instituted, and clinical outcomes during outpatient follow-up were collected. The present analysis included consecutive patients enrolled from March 2023 to January 2025, representing the data cutoff for the cohort reported here.

The evaluated outcomes included all-cause mortality, new MI, stroke, need for coronary revascularization, and new hospitalization for cardiovascular causes. Some follow-up data were not available for all patients due to non-attendance at follow-up consultations and/or the absence of new examinations during the follow-up period. Consequently, outcome analyses were performed based on the number of patients with available data for each variable.

Statistical analysis

Descriptive analysis of the data was performed. Categorical variables were described as absolute frequencies and

percentages. The normality of continuous variables was assessed using the Shapiro–Wilk test, adopting a significance level of $p > 0.05$ to characterize normal distribution. Variables with normal distribution were described as mean $\pm$ standard deviation, while those with non-normal distribution were expressed as median and interquartile range (IQR).

Statistical analyses were performed using R software.

This analysis was part of a study approved by the Institutional Research Ethics Committee (CAAE: 5.102.212/2024).

Results

A total of 196 patients diagnosed with MINOCA were included. The population had a mean age of 50.6 ± 11.8 years, with a wide age range (22 to 80 years), and a slight female predominance (52.6%). A high prevalence of traditional cardiovascular risk factors was observed, most notably systemic arterial hypertension (60.2%), dyslipidemia (45.9%), and current or former smoking (48.0%), while diabetes mellitus was present in 21.4% of patients. A history of MI was identified in 19.4% of the sample, characterizing a population with an intermediate-to-high cardiovascular risk profile (Table 1).

Regarding the clinical presentation of the index event, the majority of patients presented with non-ST-elevation MI (56.6%), although a substantial proportion had ST-elevation MI (43.4%). Among patients with an identified culprit artery ($n = 104$), the left anterior descending artery was the coronary artery most frequently related to the ischemic event (60.6%). Episodes of aborted sudden cardiac death were documented in 5.1% of cases. The mean initial left ventricular ejection fraction was $48.6\% \pm 14.0\%$, indicating that a relevant proportion of patients already presented left ventricular dysfunction during the acute phase of the event (Table 2).

Despite the diagnostic investigation performed, 40.8% of cases remained without a clear etiological definition. Among

Table 1 – Demographic and clinical characteristics of the study population ($n = 196$)

Variable	n (%)
Age (years)	50.6 ± 11.8
Female sex	103 (52.6)
Family history of premature CAD	22 (11.2)
Prior MI	38 (19.4)
Diabetes mellitus	42 (21.4)
Dyslipidemia	90 (45.9)
Systemic arterial hypertension	118 (60.2)
Chronic kidney disease (GFR < 60)	10 (5.1)
Obesity	32 (16.3)
Atrial fibrillation/flutter	13 (6.6)
Current smoking	35 (17.9)
Former smoking	59 (30.1)

CAD: coronary artery disease; GFR: glomerular filtration rate; MI: myocardial infarction.

Table 2 – Clinical presentation of MI (n = 196)

Variable	n (%)
NSTEMI	111 (56.6)
STEMI	85 (43.4)
Aborted sudden cardiac death	10 (5.1)
Left ventricular ejection fraction (%)	48.6 ± 14.0
Coronary territory related to the event (n = 104)	
Left anterior descending artery	63 (60.6)
Right coronary artery	29 (27.9)
Left circumflex artery	11 (10.5)
Left main coronary artery	1 (1.0)

NSTEMI: non-ST-elevation myocardial infarction; STEMI: ST-elevation myocardial infarction.

patients with an identified presumed etiology, non-obstructive coronary artery disease, coronary thromboembolism, spontaneous coronary artery dissection, and epicardial vasospasm were the most prominent findings (Table 3). CMR was the most frequently used complementary method and was performed in 42.3% of patients; however, it was generally performed late, with a median of 180 days after the event (IQR 22–722 days). In contrast, the use of intracoronary imaging methods, such as OCT or IVUS, was quite limited (2.6%). Other complementary tests included myocardial perfusion scintigraphy (25.5%) and coronary computed tomography angiography (5.1%), reflecting the heterogeneity of the diagnostic approach in real-world clinical practice (Table 4).

Regarding the treatment instituted after the diagnosis of MINOCA, a predominance of pharmacological therapy based on beta-blockers combined with angiotensin-converting enzyme inhibitors or angiotensin receptor blockers was observed, prescribed in 77.6% of patients. Dual antiplatelet therapy was used in 42.9% of cases, while oral anticoagulation was indicated in 16.8%, reflecting the diversity of pathophysiological mechanisms involved. Revascularization strategies were infrequent: during hospitalization, only 1.5% of patients underwent percutaneous coronary intervention, and no patient was referred for coronary artery bypass grafting (Table 5).

During clinical follow-up, available for 183 patients with a median of 39.4 months (IQR 17.4–72.1 months), all-cause mortality was 3.8%. The incidence of new MI, stroke, and new hospitalization for cardiovascular causes was 6.6%, 4.9%, and 16.9%, respectively, representing a relevant rate of adverse cardiovascular events throughout follow-up. Only 1 patient (0.5%) required late coronary revascularization (Table 6).

From a symptomatic standpoint, 41.5% of patients (76/183) experienced some degree of symptoms during follow-up (dyspnea to some degree in 37.7% and angina in 14.2%, with overlap between the groups). The majority of symptomatic patients were in mild functional classes: with respect to dyspnea, New York Heart Association classes I and II predominated, and regarding angina, most patients reported

low-intensity symptoms (Canadian Cardiovascular Society I–II). These data reinforce that, although mortality is low, a significant proportion of patients remain symptomatic during follow-up, highlighting the functional morbidity associated with MINOCA. The final left ventricular ejection fraction remained stable relative to the initial assessment (48.9% ± 13.5%), with no evidence of significant functional deterioration in the overall population (Table 6).

Discussion

The findings of the present study reinforce the multifactorial nature of MINOCA and the need for an integrated diagnostic approach that encompasses both structural and functional mechanisms, as summarized in the Central Illustration. Patients with MINOCA generally present a more favorable prognosis compared to those with MI associated with obstructive coronary disease; however, this condition remains associated with lower survival relative to age- and sex-matched healthy individuals. Furthermore, the absence of significant coronary stenoses does not necessarily imply a benign course, since in MINOCA, myocardial injury frequently results from functional or structural mechanisms not identifiable by conventional coronary angiography.^{1–3,5,11} This finding helps explain why patients with MINOCA present relevant event rates during follow-up, as described in different populations.^{5,6,13,20}

The clinical profile observed in our sample, with a higher proportion of women and younger patients and a lower burden of traditional atherosclerotic risk factors, is consistent with that described in prior studies.^{1,11–14} This pattern may be partially explained by pathophysiological differences between sexes, including a higher prevalence of microvascular dysfunction, coronary vasospasm, and spontaneous coronary artery dissection in women, as well as distinct hormonal and vascular response factors. The lower prevalence of obstructive atherosclerosis suggests that functional mechanisms play a central role in this population, which helps explain less typical clinical presentations and greater initial diagnostic difficulty.^{6,13}

The clinical heterogeneity observed in our cohort directly reflects the diversity of mechanisms involved in MINOCA.

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Table 3 – Frequency of causes of AMI in the sample (n = 196)

Cause of MI	n (%)
No defined diagnosis	80 (40.8)
Non-obstructive coronary artery disease < 50%	31 (15.8)
Coronary thromboembolism	26 (13.3)
Spontaneous coronary artery dissection	24 (12.2)
Coronary vasospasm	13 (6.6)
Coronary aneurysm/ectasia	12 (6.1)
Myocardial bridging	10 (5.1)

Table 4 – Diagnostic methods used during MINOCA investigation (n = 196)

Variable	n (%)
Cardiac magnetic resonance imaging	83 (42.3)
Time to performance – median (IQR), days	180 (22–722)
Myocardial perfusion scintigraphy	50 (25.5)
With ischemia	14/50 (28.0)
Thrombophilia workup	35 (17.9)
Positive thrombophilia	8/35 (22.9)
Coronary computed tomography angiography	10 (5.1)
OCT or IVUS	5 (2.6)

IQR: interquartile range; IVUS: intravascular ultrasound; MINOCA: myocardial infarction with non-obstructive coronary arteries; OCT: optical coherence tomography.

Table 5 – Treatment instituted after the diagnosis of MINOCA (n = 196)

Variable	n (%)
Beta-blocker + ACE inhibitor/ARB	152 (77.6)
Oral anticoagulation	33 (16.8)
Dual antiplatelet therapy	84 (42.9)
Percutaneous coronary intervention	3 (1.5)
Coronary artery bypass grafting	0 (0)

ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; MINOCA: myocardial infarction with non-obstructive coronary arteries.

Microvascular dysfunction, coronary vasospasm, subtle epicardial abnormalities, and spontaneous coronary artery dissection are frequently described causes and, in many cases, may coexist.^{8–11} In clinical practice, microvascular dysfunction is often diagnosed empirically, since specific methods for evaluating the coronary microcirculation, such as invasive coronary flow reserve measurements or pharmacological testing, are not widely available in most centers. Accordingly, a relevant proportion of patients classified as MINOCA without a defined etiological

diagnosis may, in fact, have microvascular dysfunction or vasospasm as the underlying mechanism, which helps explain the persistence of symptoms and recurrence of ischemic events even in the absence of significant coronary obstructions.^{11,18}

In this context, coronary vasospasm represents a frequently underdiagnosed mechanism. Recent evidence from the PROMISE trial¹⁶ demonstrated that a subset of patients initially classified as MINOCA based on coronary stenoses < 50% had their etiological diagnosis redefined

Table 6 – Clinical outcomes during follow-up (n = 183)

Variable		n (%)
Recurrent symptoms	No dyspnea or angina	107 (58.5)
	Dyspnea and/or angina	76 (41.5)
Current dyspnea	Asymptomatic	114 (62.3)
	Class I	19 (10.4)
	Class II	39 (21.3)
	Class III	9 (4.9)
	Class IV	2 (1.1)
Current angina	Asymptomatic	157 (85.8)
	Class I	6 (3.3)
	Class II	18 (9.8)
	Class III	1 (0.5)
	Class IV	1 (0.5)
Death	No	176 (96.2)
	Yes	7 (3.8)
New MI	No	171 (93.4)
	Yes	12 (6.6)
Stroke	No	174 (95.1)
	Yes	9 (4.9)
New hospitalization for cardiovascular causes	No	152 (83.1)
	Yes	31 (16.9)
Revascularization	No	182 (99.5)
	Yes	1 (0.5)
Final left ventricular ejection fraction (%)		48.9 ± 13.5

Dyspnea classified according to the New York Heart Association (NYHA). Angina classified according to the Canadian Cardiovascular Society (CCS).

after a systematized approach, which included vasoreactivity testing and stratified treatment according to the identified mechanism. These findings clearly illustrate how the absence of functional evaluation may lead to an initial classification of MINOCA in patients whose predominant mechanism is functional, particularly vasospasm, reinforcing the limitations of isolated angiographic assessment and the importance of more targeted diagnostic strategies when available.^{17,18} Spontaneous coronary artery dissection constitutes another relevant mechanism, particularly in younger women, and may go unrecognized on conventional angiography when clinical suspicion is not high, as the images are often subtle or may be misinterpreted as atherosclerotic plaque.^{9,10}

Given these limitations, the role of advanced cardiovascular imaging methods becomes central in the investigation of MINOCA. In our cohort, CMR contributed to the exclusion of alternative diagnoses and characterization of myocardial injury; however, its performance was frequently delayed (median of 180 days), which may have

reduced sensitivity for detecting acute edema and late gadolinium enhancement, thereby underestimating the rate of etiological elucidation. Studies have shown that CMR performed within the first 2 weeks following the event yields a higher diagnostic output,¹⁵ reinforcing that the results presented here reflect the limitations of real-world clinical practice. Furthermore, the combined use of OCT and CMR, as described in observational studies, enables the identification of non-obstructive plaque rupture or erosion in a significant proportion of patients initially classified as MINOCA.¹⁷ Although this strategy was not applied systematically across our entire population, these data may account for why some patients experience recurrent events despite the absence of significant stenoses.

Regarding clinical outcomes, the 3.8% mortality observed over a median follow-up of 39.4 months was accompanied by a high incidence of non-fatal cardiovascular events, including reinfarction (6.6%) and new cardiovascular hospitalization (16.9%), consistent with prior studies describing MINOCA as a condition of intermediate risk

between MI with obstructive coronary disease and healthy individuals.^{5,6,13,20} This substantial number of events may be explained by the combination of persistent underlying mechanisms, the presence of comorbidities, and, in some cases, lower utilization of secondary prevention therapies. Indeed, observational studies demonstrate that patients with MINOCA frequently receive fewer evidence-based therapies compared to those with MI and obstructive coronary disease, possibly due to diagnostic uncertainty and etiological heterogeneity.⁴ Evidence from meta-analyses suggests that the use of statins, renin–angiotensin system inhibitors, and beta-blockers is associated with a reduction in adverse events even in the absence of significant coronary obstructions;^{19,21} however, there is no robust evidence supporting uniform pharmacological regimens with respect to hard outcomes, largely because this is not a single disease but rather a clinical syndrome, reinforcing the importance of an individualized therapeutic approach.^{22,23}

Overall, the findings of the present study must be interpreted in light of the interaction between multiple pathophysiological mechanisms, the diagnostic limitations of conventional angiography, and the challenges in therapeutic management of MINOCA. The integration of careful clinical assessment, judicious use of imaging methods, and secondary prevention strategies tends to reduce diagnostic uncertainty and contribute to more consistent clinical management of this condition in daily practice.

Limitations

This study has limitations inherent to its observational, retrospective, and single-center design. Outpatient follow-up based on medical record review is subject to loss-to-follow-up bias, as patients who did not return for consultations or were seen at other institutions did not have their outcomes captured. The diagnostic investigation did not follow a standardized protocol, reflecting real-world clinical practice, which may have contributed to the high proportion of cases without a defined etiology. Furthermore, the delayed performance of examinations such as CMR may have reduced diagnostic accuracy in a portion of the cohort.

Conclusion

In a contemporary cohort of patients with MINOCA followed at a Brazilian tertiary center, a clinical profile characterized by relatively young age, a slight female predominance, and a high prevalence of traditional cardiovascular risk factors was observed. Despite low, yet not negligible, mortality during follow-up, the occurrence of non-fatal cardiovascular outcomes was relevant, including

reinfarction and hospitalization for cardiovascular causes. The high proportion of cases without a defined etiology underscores the need for more structured diagnostic strategies and the rational incorporation of advanced imaging methods, with a view to better etiological characterization and individualization of clinical management.

Author Contributions

Conception and design of the research: Vital K, Valente BP, Pavanello R, Feres F; acquisition of data: Vital K, Valente BP, Blaas BN, Arruda BFT; analysis and interpretation of the data: Vital K, Blaas BN, Arruda BFT, Rossi R, Del Monaco MI, Pavanello R; statistical analysis: Vital K, Arruda BFT, Rossi R; obtaining financing: Vital K; writing of the manuscript: Vital K, Blaas BN, Arruda BFT, Rossi R, Pavanello R, Feres F; critical revision of the manuscript for intellectual content: Vital K, Valente BP, Rossi R, Del Monaco MI, Pavanello R, Feres F.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Instituto Dante Pazzanese de Cardiologia under the protocol number 7.904.853. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

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