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ACUTE MYOCARDIAL INFARCTION AND NON-OBSTRUCTIVE CORONARY ARTERIES: A CASE REPORT

Infarto agudo do miocárdio e artérias coronárias não obstrutivas: um relato de caso

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ABSTRACT

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a rare event that is difficult to diagnose. Given the rarity of this condition, we report the case of a 61-year-old patient who was treated in the intensive care unit of the Padre Bento Hospital Complex in Guarulhos, Brazil, who developed a rare case of MINOCA syndrome during hospitalization for subarachnoid hemorrhage. Coronary angiography did not show significant coronary artery obstruction. The patient showed clinical improvement and was discharged after 48 hours, with recommendations for outpatient cardiology follow-up and cardiac magnetic resonance imaging.

Keywords: acute myocardial infarction, non-ST elevation myocardial infarction, MINOCA, coronary angiography.

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INTRODUCTION

Myocardial infarction with nonobstructive coronary arteries (MINOCA) is a syndrome resulting from a set of conditions that affect the structure of the coronary microcirculation, culminating in acute myocardial infarction (AMI), but without evidence of significant obstructive coronary artery disease on angiography[1].

Given the complexity associated with MINOCA syndrome, the challenges for its correct and early diagnosis are significant. Much of this complexity arises from the wide range of possible etiologies for the syndrome, which include coronary spasm, microvascular dysfunction, coronary embolism, and inflammatory conditions. Furthermore, there are still no treatment guidelines for MINOCA. As a result, many patients

do not receive an accurate diagnosis nor appropriate and timely treatment[2].

Thus, considering the uncertainties and the knowledge gap surrounding this subject, this study presents a clinical case of MINOCA and discusses essential aspects of its diagnosis and treatment. This aims to increase awareness, particularly among emergency physicians, allowing early recognition and more effective treatment of MINOCA.

CLINICAL CASE PRESENTATION

The case involves a 61-year-old female patient, N.S.S., of mixed race, from Guarulhos, State of São Paulo, Brazil, with a history of smoking, systemic arterial hypertension, type II diabetes mellitus, and prior coronary artery angioplasty with stent placement. The patient was admitted to the intensive care unit at the Padre Bento Hospital Complex in Guarulhos on April 24, 2019, due to subarachnoid hemorrhage (Fisher grade II) with associated abnormalities within the brain parenchyma. The patient was hemodynamically stable, conscious, and oriented. On May 13, 2019, cerebral angiography showed irregular intracranial artery contours but no significant stenosis or aneurysms.

The next day, in the morning, the patient experienced severe left-sided chest pain radiating to the back and left arm, accompanied by sweating and uncontrollable vomiting. This presentation was identified as type B precordial anginal pain. She was given 300 mg of acetylsalicylic acid and underwent a 12-lead electrocardiogram that showed ST-segment elevation in AVR and ST-segment depression in V6, prompting the initiation of the high-risk coronary syndrome protocol.

Cardiac markers of myocardial necrosis were slightly elevated six hours after symptom onset, with creatine kinase MB (CK-MB) increasing from 21 U/L to 33 U/L (normal < 25 U/L) and troponin rising from 1.25 mg/dL to 1.55 mg/dL (normal < 0.1 mg/dL), indicating AMI.

The patient's pain subsided with morphine and nitroglycerin. On the same day, coronary angiography revealed a dominant right coronary circulation pattern, irregular coronary arteries, and a patent stent in the anterior interventricular artery but no significant stenosis or obstruction.

After 48 hours of clinical stabilization, troponin levels normalized, and the patient was discharged from the ICU with referrals for outpatient follow-up care with cardiology and cardiac magnetic resonance imaging (MRI).

DISCUSSION

MINOCA accounts for approximately 5-6% of AMI cases[3-6]. Patients' demographic and clinical characteristics differ from those with AMI due to obstructive causes. MINOCA patients tend to be younger (average age 58) compared to those with obstructive AMI (average age 61)[7-10] Furthermore, women with a diagnosis of AMI are twice as likely to experience MINOCA compared to men, and higher incidences are observed among Black and Hispanic populations in the U.S5,11. However, despite these epidemiological differences, it is not possible to differentiate between AMI caused by MINOCA and obstructive mechanisms based solely on clinical or epidemiological features, making coronary angiography essential for this differential diagnosis[5].

Diagnostic criteria for MINOCA are[5]:

1. Classic AMI criteria (elevated cardiac markers of myocardial necrosis, suggestive clinical presentation, ECG changes such as ST-segment elevation or new left bundle branch block, myocardial changes on cardiac perfusion scan, or new ventricular wall motion abnormalities).
2. Absence of ≥50% coronary artery obstruction on angiography[9];
3. Exclusion of other clinically evident causes that could result in the observed acute clinical presentation.

It is important to emphasize that MINOCA is a working diagnosis with multiple underlying etiologies that require further work-up to be identified[5,12]. This process involves thoroughly reviewing the patient's history and physical examination, looking for factors suggesting conditions such as viral infections, drug use, increased thrombotic risk, or significant psychological stress (which could indicate Takotsubo cardiomyopathy). However, if a specific etiology is established (e.g., recent cocaine use causing artery coronary spasm), MINOCA should not be the primary diagnosis[3].

However, if the cause remains unclear, further diagnostic evaluation is critical. The initial step involves a detailed analysis of coronary angiography, which may uncover alternative causes of AMI other than ≥ 50% obstruction, such as coronary dissection or plaque instability. The latter, despite having an atherothrombotic mechanism, might not obstruct the main coronary arteries but instead potentially lead to distal embolization of thrombi resulting from plaque erosion or rupture[3]. Techniques such as optical coherence tomography or intracoronary ultrasound can help identify these conditions[3], while cardiac MRI is highly valuable for MINOCA evaluation, especially when myocarditis or structural abnormalities are suspected[3,13]. When coronary spasm is on the differential, a spasm provocation test can be a useful diagnostic adjunct, while echocardiography has lower specificity in this context[5]. **Table 1** outlines the potential causes of MINOCA and the recommended work-up evaluation.

In addition to cardiac causes, further investigation may be indicated to rule out non-cardiac causes of elevated cardiac markers of myocardial necrosis, such as chronic kidney disease and pulmonary thromboembolism, depending on clinical suspicion[5]. For instance, if a patient presents with unilateral lower limb edema, a workup for pulmonary embolism may be indicated, including tests such as D-dimer and chest CT with pulmonary angiography. In cases where thrombophilia is suspected, additional tests for specific conditions, such as Factor V Leiden, may be indicated.

While it is crucial to exclude non-cardiac causes of false-positive results for AMI, the absence of coronary obstruction on angiography should not be interpreted as the absence of myocardial injury. Instead, a thorough evaluation to determine the specific etiology of MINOCA is essential to avoid overlooking a serious cardiovascular event. Furthermore, accurate diagnosis is critical for guiding targeted treatment, preventing recurrence, and reducing mortality. For instance, if coronary spasm is identified, treatment with calcium channel blockers should be initiated, as this approach is linked to better outcomes[5,13].

The case of patient N.S.S. illustrates the typical epidemiological profile for patients at increased risk for MINOCA, meeting all clinical diagnostic criteria. Although she received AMI treatment according to standard guidelines, the institution where she underwent coronary angiography did not adhere to the recommended diagnostic algorithm for MINOCA following confirmation of the absence of coronary obstruction[14].

The MINOCA syndrome remains relatively under-recognized, even among specialists. Although the syndrome is increasingly described in the literature, this limited awareness often impedes diagnostic evaluation and patient follow-up. As a result, many patients continue to receive treatment based on traditional AMI protocols, which may not be optimal for MINOCA. To improve patient outcomes, it is crucial to enhance physician awareness of MINOCA, particularly among those working in emergency settings.

In the case presented, a complete etiological investigation was not possible due to limited resources, reflecting a common challenge in many healthcare facilities in Brazil resulting from insufficient resources. This case underscores the need to establish efficient referral and counter-referral workflows to facilitate the proper investigation and management of MINOCA.

CONFLICT OF INTEREST

The authors attest they have no conflict of interest to declare.

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TABLE 1 - Differential diagnosis of MINOCA etiologies and diagnostic work-up

POSSIBLE ETIOLOGIES OF MINOCA		RECOMMENDED DIAGNOSTIC TESTS
Myocardial causes	Cardiomyopathies (Takotsubo, hypertrophic, dilated)	Cadiac magnetic resonance imaging, ventriculography
	Miocarditis	C-reactive protein, erythrocyte sedimentation rate, cadiac magnetic resonance imaging, and myocardial biopsy
	Trauma	History and physical exam
	Acute myocardial infarction induced by tachyarrhythmia	Monitorization
Coronary causes	Coronary artery dissection	Echocardiogram and coronary computed tomography angiography
	Coronary spasm induced by sympathomimetics	Toxicology tests
	Epicardial coronary artery spasm	Intracoronary acetylcholine provocation testing
	Coronary microvascular spasm	Intracoronary acetylcholine provocation testing
	Coronary microvascular disease	Coronary flow reserve calculation
	Coronary slow flow phenomenon	Angiography with TIMI frame count
	Thrombosis secondary to plaque instability (erosion or rupture)	Optical coherence tomography and intravascular ultrasound
	Coronary emboli	Echocardiogram

Adapted from Pasupathy *et al.* 2016[10] and Tamis-Holland *et al.* 2019[12].

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