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The editorial team of the Arquivos Brasileiros de Cardiologia journal hereby announces the formal publication of a Partial-Retraction for the article:

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As plagiarism of the figure has been confirmed.

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[PARTIAL-RETRACTION] MINOCA Phenotypes – A Challenge for Patient-Specific Management

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Abstract

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a puzzling clinical phenomenon with an unclear prognosis, characterized by evidence of myocardial infarction (MI) with normal or near-normal coronary arteries on angiography¹. Currently, there are no guidelines for management, and many patients are discharged without a determined etiology, often meaning that optimal treatment gets postponed.

We report three MINOCA case studies with main pathophysiological cardiac causes, particularly epicardial, microvascular, and non-ischemic, prompting differential management. The patients presented with acute chest pain, troponin raise, and no angiographically significant coronary disease.

In this study, we analyzed the etiology, clinical diagnosis, and treatment of MINOCA concerning the relevant literature.

MINOCA is considered to be a dynamic working diagnosis, including coronary, myocardial, and non-coronary disorders. Prospective studies and registries are needed to improve patient care and outcome.

Introduction

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a puzzling clinical phenomenon with an unclear prognosis characterized by evidence of myocardial infarction (MI) with normal or near-normal coronary arteries on angiography.¹ Currently, there are no guidelines for management, and many patients are discharged without a determined etiology, often meaning that optimal treatment gets postponed. We report three MINOCA case studies with main pathophysiological cardiac causes, particularly epicardial, microvascular, and non-ischemic, prompting differential management.

Case 1

A 63 years old white woman presented with acute chest pain induced by unexpected emotional stress.² Her medical

Keywords

Phenotype; MINOCA; Diagnosis Diferential; Dilatation Pathologic; Coronary Artery; Microvascular Spasm

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history included mild hypertension. During admission, an electrocardiogram (ECG) showed sinus tachycardia with ST elevation of 5 mm in V2-V6, pathological Q wave, and ST elevation of 2 mm in II, III, and avF. Laboratory results included an elevated troponin T level of 886.3 pg/mL (normal range 12.7-24.9 ng/ml) and NT-proBNP 1434 pg/ml (normal < 125 pg/mL). A transthoracic echocardiogram (TTE) revealed an ejection fraction (EF) of 45 % with hyperdynamic basal function and a dilated, akinetic apex lateral wall. According to the result of emergent coronary angiography, there was no coronary stenosis \geq 50% in any potential infarct-related artery. However, left ventriculography revealed apical ballooning dilatation with akinesis [REDACTED]. The diagnosis of Takotsubo syndrome was suspected based on results of apical motion abnormalities of LV, preceded by a stressful, emotional trigger, absence of culprit atherosclerotic coronary artery disease, new ECG abnormalities, positive troponin test and significantly elevated NT –pro-BNP.

On the 12th day, TTE showed no change in the apical ballooning and akinesis. The level of troponin T was decreased in dynamics. The patient was discharged from the hospital on metoprolol and ramipril therapy.

In 3 weeks, the apical wall-motion abnormalities had resolved, and the LVEF had returned to normal at 59 %. Newly apparent hypertrophy of the LV myocardium at the apex was consistent with apical hypertrophic cardiomyopathy (HCM). A contrast agent was administered; no apical pouches or thrombi were found. The maximal LV wall thickness of apex was 17 mm, and the interventricular septum was 12 mm at end-diastole. The ECG showed repolarization changes and giant, inverted T waves in the anterolateral leads. The patient was counseled about the diagnosis of apical HCM. She was asymptomatic at her 6 and 12-month follow-up examinations, which included TTE.

Case 2

A 31-year-old male without prior cardiac history presented with crushing substernal chest pain at rest and palpitation. His cardiac risk factors included tobacco abuse and a family history of myocardial infarction in his father at 40. During the past 5 years, he has been in bodybuilding and used high doses of anabolic androgenic steroids (AAS). During admission, initial ECG showed pathological Q waves in II, III, AVF, 2 mm ST segment elevation in II, III, AVF, and V4–6 leads without reciprocal ST segment depression in I, AVL (Fig.2A). There was biochemical evidence of myocardial damage: troponin T 1952 pg/mL, NT-proBNP 260,8 pg/ml. TTE revealed hypokinesis of the posterior and lateral wall with normal EF. Based on these data, he was managed as STEMI. However, the results of coronary angiography revealed no significant lesion of coronary arteries (Figure 2B); however provocative intracoronary test with acetylcholine for suspected vasomotor dysfunction was positive.

A diffuse spasm pattern was found. Diagnostic work-up of the patient's current symptoms included a cardiovascular magnetic resonance. No pathological changes were found. He was discharged on calcium channel blockers and nitrates. Caution was given regarding the usage of AAS. At his 6 and 12-month follow-up examination, he was asymptomatic.

Case 3

A 45-year-old overweight hypertensive male presented to the emergency department with crushing chest pain at rest and palpitation 1 hour after the onset of symptoms. ECG demonstrated ST-segment elevation in I, AVL, and V2-V6 leads at admission. Bedside transthoracic echocardiography showed hypokinesis of the anterior-lateral walls of the left ventricle. The patient was transferred for emergency coronary angiography

due to severe chest pain and elevated high-sensitive troponin T-level at 585.0 ng/ml. There was no coronary stenosis $\geq 50\%$. However, there were multiple ectasias in the left coronary artery, particularly a small sac-like aneurysm in 11 segments and a spindle-shaped aneurysm in 13 segments; a large-sized fusiform aneurysm with contrast stasis in the 6-7 segments of the left anterior descending artery; ectasia in 1 segment of the right coronary artery were revealed (Figure 3). The patient underwent coronary artery bypass grafting of left descending and circumflex coronary arteries in 1 month after ACS.

Discussion

Since MINOCA involves several pathophysiological mechanisms and various clinical presentations, the type of

RETRACTED FIGURE

Figure 1 – Left ventriculography revealed apical ballooning dilatation with akinesis and TTE-apical hypertrophy.

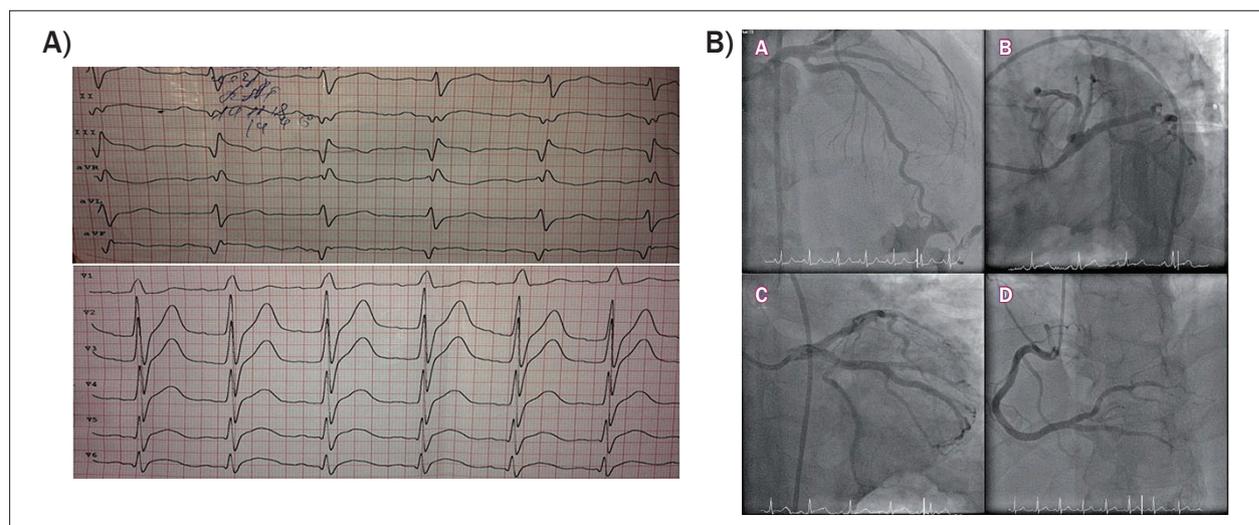


Figure 2 – A) Initial ECG. B) Coronary angiography with no significant lesion of coronary arteries.

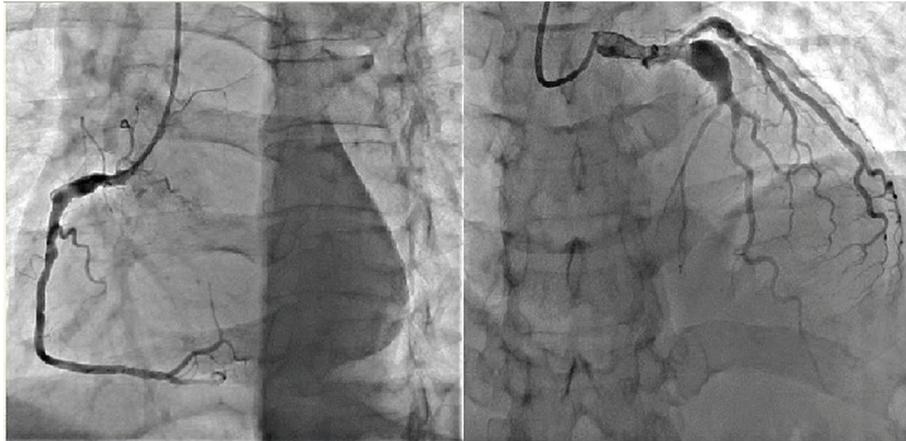


Figure 3 – Coronarography with multiple coronary ectasia..

management varies depending on the underlying cause.³ The differential diagnosis includes myocarditis, coronary microvascular disease, pulmonary embolism, myocardial diseases such as Takotsubo, and an imbalance between oxygen supply and demand of myocardium (Type 2 MI).⁴ Despite having a contemporary position statement from the ESC and the AHA, great variability exists in how patients with suspected MINOCA are evaluated. Currently, the consensus now excludes myocarditis and Takotsubo syndrome from the final diagnosis of MINOCA.⁵⁻⁷

We reported 3 clinical cases of patients with acute chest pain, troponin raise, and absence of angiographically significant coronary disease. Although elevated troponin levels reflect cardiomyocyte injury with the release of this intracellular protein into the blood, the process is not disease-specific and can result from either ischaemic or non-ischaemic mechanisms.⁶

In the first case, apical HCM was masked by MINOCA as it fulfilled the following criteria: rise in cardiac troponin, symptoms of myocardial ischemia: new ischemic ECG changes, new regional wall motion abnormality on TTE; no coronary stenosis $\geq 50\%$ in any potential infarct-related artery. There were no specific alternate diagnoses for the clinical presentation: sepsis, pulmonary embolism, and myocarditis. However, MINOCA is an initial working diagnosis, and proper cardiac imaging is crucial. Apical akinesis and dilation in the absence of obstructive coronary artery disease were considered signs of stress-induced (Takotsubo) cardiomyopathy, whereas apical hypertrophy was found during follow-up on TTE was an apical-variant of hypertrophic cardiomyopathy. CMR can identify the underlying cause in as many as 87% of patients with MINOCA.⁸ In the sub-endocardium, late gadolinium enhancement may indicate an ischemic cause, while sub-epicardial localization may prove cardiomyopathies or myocarditis, and the absence of relevant late gadolinium enhancement with edema and associated specific wall motion abnormalities are a hallmark of Takotsubo syndrome.^{8,9}

Beta-blocker therapy in such patients can be useful to achieve adrenergic blockade, and other conventional heart failure therapies might be applied.¹

In the second case, the possible pathogenesis of AAS-related infarction includes coronary artery spasm and/or temporary thrombosis. Current abuse of AAS should be avoided. Calcium antagonists are central in managing coronary artery spasms and are strongly recommended as first-line drugs.¹⁰ As opposed to beta-blockers as they prosper spasm by leaving alpha-mediated vasoconstriction unopposed by beta-mediated vasodilation.¹¹

In the third case, in patients with ACS due to coronary artery ectasia, the emphasis is restoring flow. The percutaneous coronary intervention of an aneurysmal/ectatic culprit vessel had lower procedural success and a higher incidence of no-reflow and distal embolization.¹² Surgical resection is considered the first-line therapy for CAE involving the left main coronary artery, multiple or giant (>20 mm, or $> 4\times$ reference vessel diameter) aneurysms.^{13,14}

Conclusion

MINOCA is considered to be a dynamic working diagnosis, including coronary, myocardial, and non-coronary disorders. Prospective studies and registries are needed to improve patient care and outcome.

Author Contributions

Conception and design of the research; Acquisition of data; Statistical analysis; Analysis and interpretation of the data; Writing of the manuscript; Critical revision of the manuscript for important intellectual content: Lypovetska S.

Potential conflict of interest

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Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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