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Case Report

A Case Report of MINOCA/INOCA with Cardiac Damage and Dysfunction. How to Treat?

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Abstract

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a syndrome with clinical evidence of myocardial infarction, elevation of myocardial necrosis enzyme and with normal anatomy or absence of stenosis >50% in any epicardial coronary artery at coronary angiography. Conversely in ischemia with non-obstructive coronary artery disease (INOCA) they do not present elevation of myocardial necrosis enzyme. Almost 70 % of patients who undergoing coronarography for symptoms and/or signs of myocardial ischemia present epicardical coronary arteries without significative obstruction [1]. We report the case of a 53-year-old woman with history of repeated episodes of chest pain over the years both under stress and at rest. The features of this case allowed a diagnosis to be made only when myocardial damage was evident. The diagnosis and management of patients with MINOCA/INOCA is a challenge.

Abbreviations: AMI = Acute myocardial infarction; CFR = Coronary flow reserve; INOCA = Ischemia with non-obstructive coronary artery disease; MINOCA = Myocardical infarction with non-obstructive coronary arteries; OCT = Optical coherence tomography; IVUS = Intravascular ultrasound; FFR = Fractional flow reserve measurement CMR = Cardiac magnetic resonance; CAD = Coronary artery disease; MACE = Major adverse cardiovascular events; bAPV= Baseline average peak velocity

Introduction

The term MINOCA denotes all those cases of myocardial infarction with an angiographic imagine of epicardial coronary arteries free of angiographically significative lesions. To date, the percentage of MINOCA in the AMI population is estimated to be between 3% and 15% [2]. This subclass was introduced in 2018 by the European Society of Cardiology with diagnostic criteria based on the fourth universal definition of AMI.

1. elevation of cardiac biomarkers in patients with symptoms and/or signs of myocardial ischemia
2. absence of stenosis >50% in any epicardial coronary artery at coronary angiography
3. lack of any alternative diagnosis

The pathophysiological mechanisms identified, to date, are thought to be the following: rupture of a coronary plaque, coronary vasospasm, microvascular dysfunction, spontaneous coronary artery dissection, embolism or coronary thrombosis. Undoubtedly, coronarography remains crucial for the study of epicardial arteries and the detection of absence of stenosis >50%. For microvascular dysfunction is important consider other ratings. The main cause seems to be the impaired coronary flow reserve (CFR), which is

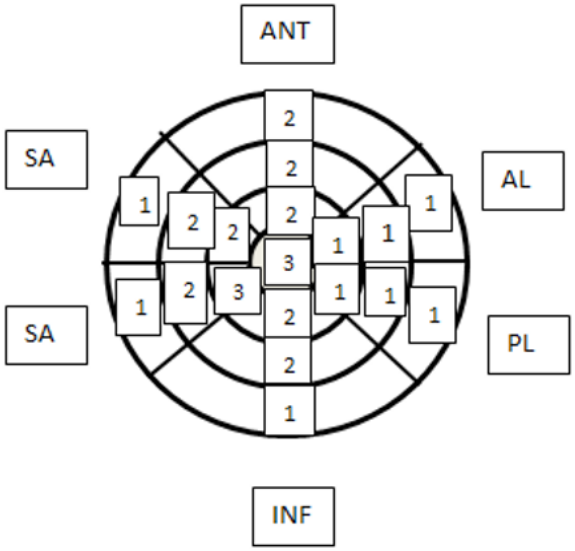
associated with angina and adverse cardiovascular events. CFR is a ratio of hyperemic to baseline average peak velocity (bAPV). Diagnosis currently makes use of various tools.

Intravascular studies such as optical coherence tomography (OCT), intravascular ultrasound (IVUS) are also helpful in this regard, as they allow a better study of the plaque, its composition and its hemodynamic effect. There are also additional imaging techniques such as cardio CT and cardiac magnetic resonance (CMR) that are essential for the differential diagnosis with pathologies that could potentially have similar clinical and biohumoral characteristics but different pathogenetic mechanisms, such as acute myocarditis, some cardiomyopathies and Tako-tsubo syndrome.

In addition to MINOCA there are also cases of ischemia with non-obstructive coronary artery disease (INOCA). INOCA is a condition similar to MINOCA but without the elevation of myocardionecrosis enzymes [3].

Case Description

A 53-year-old woman with a family history of coronary artery disease (CAD) comes to our attention; her father had died of complications related to AMI at the age of 60; she has dystothyroidism and previous carcinoma of the left breast (2011). In June 2023, the patient went for a visit to her cardiologist due to repeated episodes of chest pain, both under stress and at rest, which had reappeared in recent months as similar episodes had already occurred in previous years (period between January 2015 and February 2019) followed by cardiology checks in the absence of clinical or electrocardiographic abnormalities suggestive of an ongoing or previous ischemic event. An ergometric test had also been performed with negative results. During this outpatient check-up, no altered laboratory parameters were revealed, but the electrocardiogram showed a regular normofrequent sinus rhythm with negative antero-lateral T waves. (Image 1). Later the echocardiographic examination showed a moderate global contractile deficit with reduced regional of kinetics in the mid apical infero-postero-lateral region (see diagram).



Basale	Medio-ventricolare	Apicale
1. Anteriore	7. Anteriore	13. Anteriore
2. antero-settale	8. antero-settale	14. settale
3. infero-settale	9. infero-settale	15. inferiore
4. inferiore	10. inferiore	16. laterale
5. infero-laterale	11. infero-laterale	17. Apice
6. antero-laterale	12. antero-laterale	

Diagram: Image of wall motion.

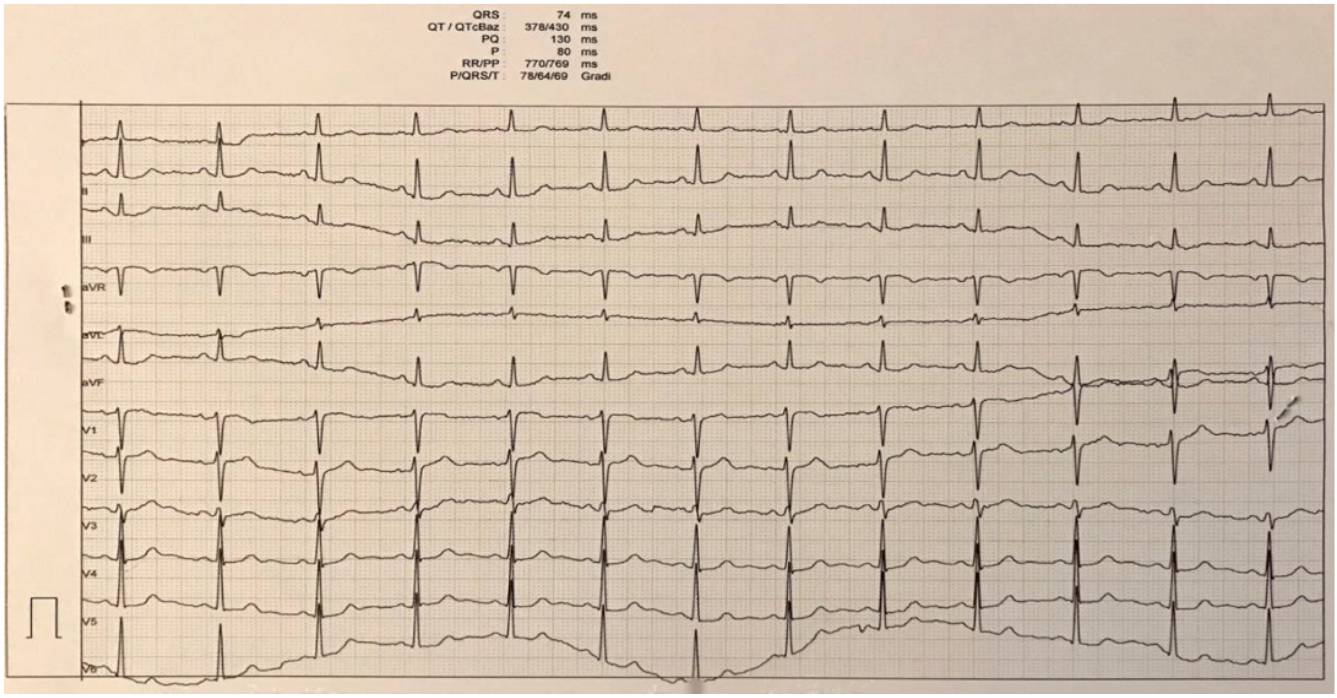
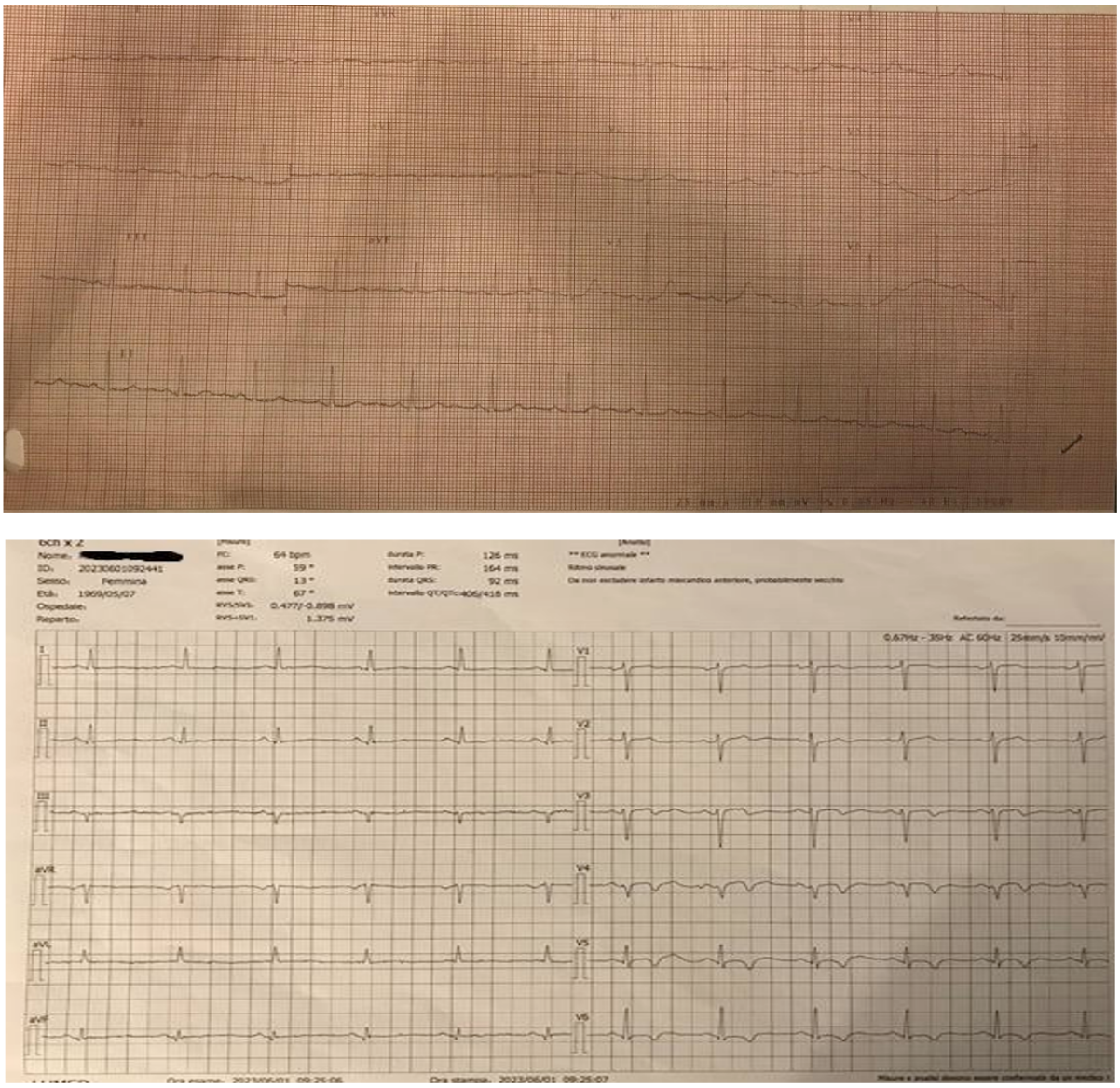


Image 1: The electrocardiogram showed a regular normofrequent sinus rhythm with negative antero-lateral T waves.

The decision was made to refer the patient to the cath lab for severe angina (image 2,3). The result of the angiographic examination was negative for significant atheromatous lesions susceptible to revascularisation (image 4). The diagnostic course was completed with CMR in order to discriminate the underlying cause of the cardiac damage. From this evaluation in the pre-contrast phase, the presence of areas of signal hyperintensity involving the apical septal and inferior segments of the left ventricle (compatible with adipose metaplasia typical of infarction) was reported on cine SSFP and T1-weighted FSE images (image 5). Late post-contrast images, on the other hand, showed the presence of an extensive area of enhancement with subendocardial/transmural distribution, compatible with ischemic necrosis, involving the middle segments of the inferior septal and inferior walls as well as the entire apex of the left ventricle. Compatible with the final diagnosis of an area of previous ischemic necrosis involving the middle segments of the inferior wall and infero-septal as well as the apex in toto appearing aneurysmal with associated mild reduction in global systolic function. A good control of symptoms was achieved thanks to the use of Ranolazine titrated at a dosage of 500 mg twice a day (EuroQol score)



Images 2 and 3: The decision was made to refer the patient to the cath lab for severe angina.

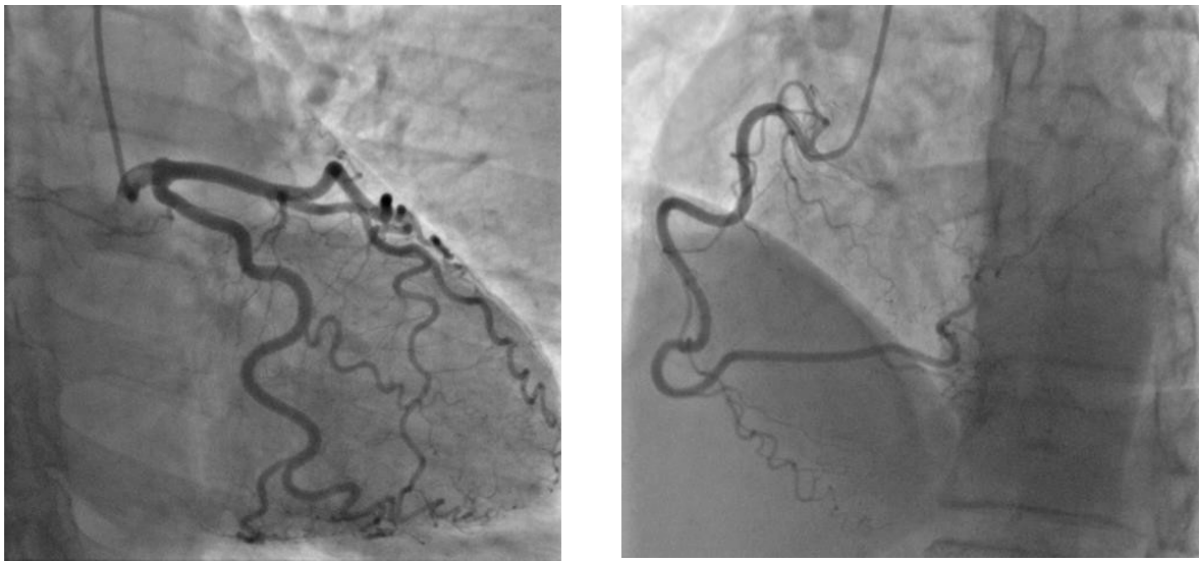


Image 4: The result of the angiographic examination was negative for significant atheromatous lesions susceptible to revascularisation.

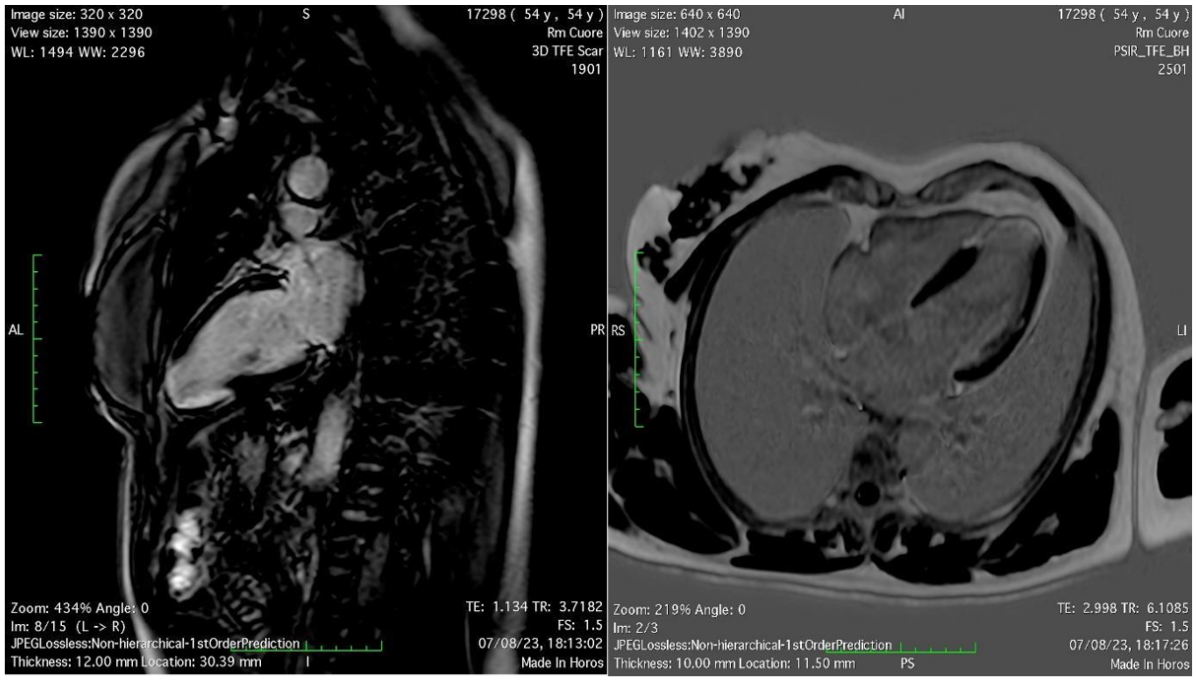


Image 5: The pre-contrast phase, the presence of areas of signal hyperintensity involving the apical septal and inferior segments of the left ventricle was reported on cine SSFP and T1-weighted FSE images.

Discussion

As the clinical case shows, diagnosing MINOCA/ INOCA is very complex and often delayed. MINOCA is not a benign condition, the literature seems to show that there is a higher incidence in groups of patients who tend to be younger than myocardial infarction patients with obstructive CAD, as well as a worse prognosis. For example, in a 30-day follow-up of a sample of 14,045 patients, a higher mortality rate was found than in cases of AMI-CAD (4.48 and 3.46%, respectively) [4].

Other studies also report worse prognoses for MINOCA than for those with obstructive MI-CAD [5,6]. However, there are also different opinions, such as studies reporting similar prognoses for the two groups [4].

Undoubtedly, this pathology still presents many grey areas from a diagnostic and prognostic as well as therapeutic point of view. The search is currently on for 'target' risk factors that allow this category of patients to be identified and stratified so as to plan a diagnostic protocol that allows these patients to be correctly identified and treated at an early pre- event stage. Probably the current pathway used, which is well understood and established in the classic form of presentation of coronary syndrome with epicardial coronary artery involvement, has shortcomings in cases labelled as MINOCA. The biggest problem is that the mode of presentation of MINOCA can occur with different scenarios that cannot always be framed as ischemic pictures because they have not the clinical, instrumental and laboratory criteria that would justify invasive examinations and\or level II imaging. In the present case, the patient started a backward pathway from the evidence of an outcome (not always so evident at a trans-thoracic echocardiographic evaluation), up to a diagnosis with advanced imaging labelling the patient as "ischemic" and only afterwards putting in place a polypharmacological therapy aiming at treatment goals that are often not required for "non CAD" patients. Identifying the target patient early is crucial to avoid irreversible damage [7].

Coronarography remains critical to document stenoses <50% and to study the plaque. A non-critical lesion may be a true dynamic entity subject to changes and/or alterations that may trigger an ischemic cascade leading to an equivalent event or symptomatology (MINOCA/INOCA).

Thus, patients with MINOCA could benefit from intravascular techniques that allow the nature of the plaque to be studied, such as IVUS, which in MINOCA seems to have detected plaque rupture in up to 40% of cases [8], and OCT, which revealed the underlying lesion in about half of the cases [8,9].

Intravascular imaging also makes it possible to highlight coronary dissections that are not recognized on angiography. For atherosclerotic plaques without the criteria of instability, it would also be possible to perform a vasoreactivity test with acetylcholine or ergotamine and to evaluate microvascular dysfunction.

Another grey area is to draw a well-defined course of treatment and follow-up for these patients. Studies such as the one done in Sweden with a large national registry of AMI patients showed that patients discharged on statins and renin-angiotensin system inhibitors had a 23% to 18 % lower risk for any MACE during the mean follow-up period of 4 years [10].A Korean study, on the other hand, found that the lack of statins and renin-angiotensin system inhibitors at discharge of MINOCA patients increased the 2-year risk of death from all causes by a factor of two [6].

Moreover, one must also consider presentation pictures in which neither enzyme elevation nor acute electrocardiographic alterations (INOCA) are found, patients who, among other things, tend to be young and in the absence of particular risk factors that might alarm the physician.

Unfortunately, in our clinical case the reiterating and non-disabling chest pain, the non-evidence of myocardial necrosis enzyme elevation, and the absence of significant electrocardiographic changes trace, have contributed to the difficult diagnosis not before consolidation of the myocardial damage. The tool that eventually allowed diagnosis was cardiac MRI because through the late gadolinium enhancement (LGE) it identified and localized the damaged areas characterizing the tissue phenotype. Unfortunately, all this occurred when tissue damage was already present. Even though for most patients with MINOCA/ INOCA it is not always possible to quantify the area of necrosis because the area affected during the acute event is so narrow as to cause an enzymatic rise but not to determine the detection of the area with MRI (the sequences in use in most laboratories do not allow the LGE to be detected below 0.2 g of infarcted myocardial mass). Therefore, even this method has its limitations.

Undoubtedly, the identification of patients at risk is very difficult at the moment and it is therefore complex to think of acting preventively. Much attention is being paid to a recent evolution of combined coronary CT with dynamic CT perfusion evaluation in patients with stable angina pectoris at intermediate risk and in cases of acute chest pain with negative troponins in the absence of clear electrocardiographic alterations or with doubtful or suboptimal ergometric testing. This examination provides both anatomical and functional information on the morphology of the plaque, the degree of stenosis as well as myocardial blood flow under conditions of fatigue (stress phase). An improvement in symptom-free time during physical activity was achieved thanks to the introduction of Ranolazine titrated to a dosage of 500 mg

twice a day in therapy as reported an improvement in EuroQol score after 4 weeks [13].

Conclusion

Intercepting at an early or early stage the patient with INOCA\ MINOCA so as to prevent or limit the extension of an acute event remains one of the ultimate challenges for those dealing with the ischaemic patient. Several tools are now available to us with increasingly sophisticated and accurate methods that are often not considered or taken into account due to the complex and subtle clinical presentation of this syndrome that does not always seem to justify them. Among the patients, especially women with INOCA, angina relates, a result supported by the concomitant greater use of anti-anginal drugs [11]. These results suggest that high bAPV contributes to impaired CFR and may represent a specific pathophysiologic contributor to CMD and may be a treatment target in INOCA [12].

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