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Troponin-Positive Non-Obstructive Coronary Arteries –TpNoca

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Introduction

The term MINOCA denotes all those cases of myocardial infarction with an angiographic immagine of epicardial coronary arteries free of angiographically significant lesions. To date, the percentage of MINOCA in the AMI population is estimated to be between 3% and 15% (1). 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 infarct clinic; 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. Diagnosis currently makes use of various tools, primarily coronarography without which the state of the epicardial coronary arteries could not be ascertained. Intravascular studies such as optical coherence tomography (OCT), intravascular ultrasound (IVUS) and fractional flow reserve measurement (FFR) are also helpful in this regard, as they allow a better study of the plaque, its composition and its haemodynamic 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 presentation pictures but different pathogenetic mechanisms, such as acute myocarditis, some cardiomyopathies and Tako-tsubo syndrome.

Keywords: Coronary Syndrome; Troponin-Positive; Coronary Arteries; MINOCA

Abbreviations: AMI = Acute myocardial infarction; 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

Case Description

A 53-year-old woman with a family history of Coronary artery disease (CAD); 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 ischaemic 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. (Figure 1). Later the echocardiographic examination showed a moderate global contractile deficit with regionality of chinese in the mid apical infero-postero-lateral region (see diagram).

Cinesi regionale							
		setto		P. Inf.	P. P.Lat	P.Lat.	P.Ant.
	bas	1	1	1	1	1	1
	med	2	2	2	2	1	1
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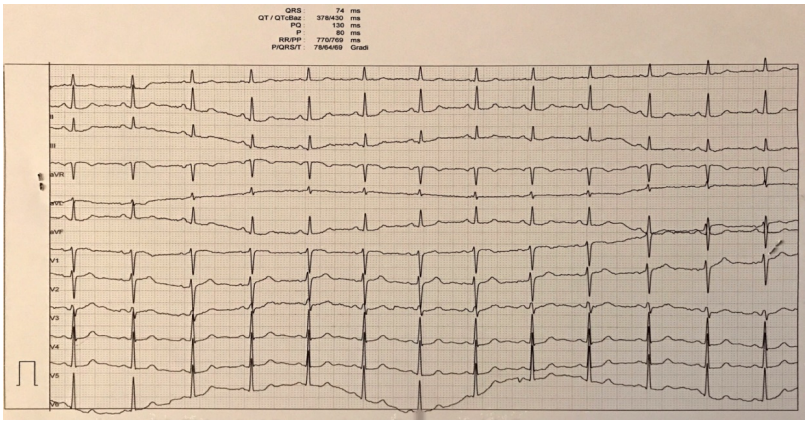

Figure 1

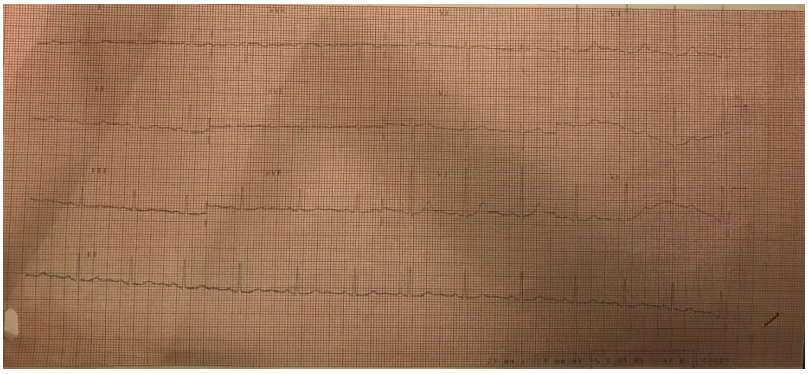

Figure 2

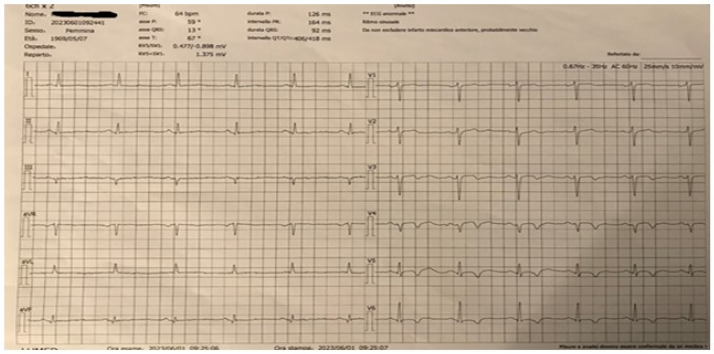

Figure 3

The decision was made to refer the patient to the haemodynamics laboratory for severe angina. The result of the angiographic examination was negative for significant atheromatous lesions susceptible to revascularisation (Figure 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 (Figure 5). Late post-contrast images, on the other hand, showed the presence of an extensive area of enhancement with subendocardial/transmural distribution, compatible with ischaemic necrosis, involving the middle segments of the inferior septal and inferior septal walls as well as the entire apex of the left ventricle. Compatible with the final diagnosis of an area of previous ischaemic 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.

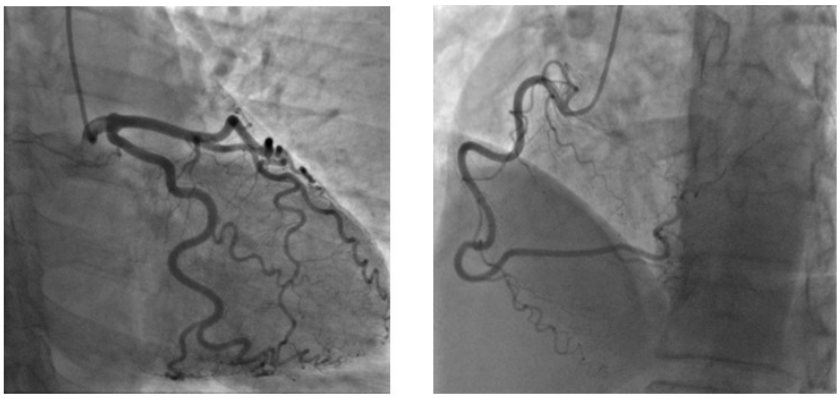

Figure 4

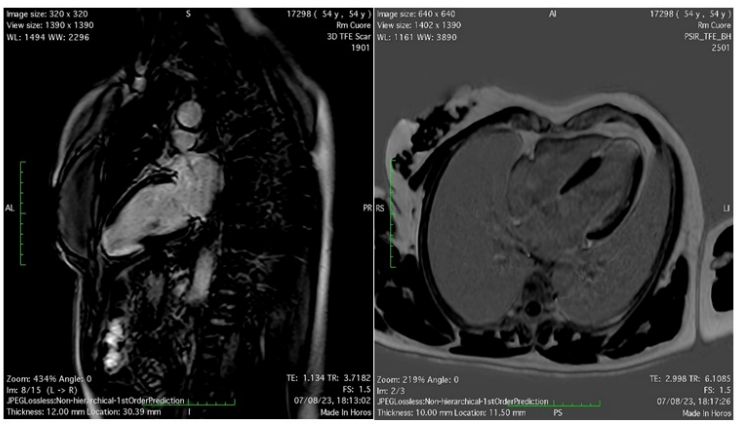

Figure 5

Discussion

As the clinical case shows, diagnosing MINOCA is very complex and often delayed. Unfortunately, it is not a benign condition; on the contrary, 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) [2]. Other studies also report worse prognoses for MINOCA than for those with obstructive MI-CAD [3, 4]. However, there are also different opinions, such as studies reporting similar prognoses for the two groups [5, 6].

Undoubtedly, this pathology still presents many grey areas from a diagnostic and prognostic as well as therapeutic point of view. This often results in late diagnosis. 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 are not always indicated as ischaemic pictures as they are not characterised by all those 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 "ischaemic" and only afterwards putting in place a polypharmacological therapy aiming at treatment goals that are often not required for "non CAD" patients.

Thus, for patients in the first case, the course will be one in which coronarography is imperative; indeed, this examination can be used to intervene promptly in cases of haemodynamically significant stenosis. Patients with MINOCA will remain untreated or risk late treatment. Identifying the target patient can be useful to use various accurate diagnostic tools such as the study of the plaque even if it is not critical or just moderate, considering this lesion, not as a static lesion but as a real dynamic entity subject to variation and/or alterations that can trigger an ischaemic cascade leading to an event or equivalent symptomatology (INOCA) . For example, there are intravascular techniques that make it possible to study the nature of the plaque, such as IVUS, which in MINOCA appears to have detected plaque rupture in up to 40% of cases [7], and OCT, which has revealed the underlying culprit lesion in about half of the cases [8].

Intravascular imaging also makes it possible to highlight coronary dissections that are not recognised on angiography. For atherosclerotic plaques without the criteria of instability, it would also be possible to perform a vasoreactivity test with acetylcholine or ergotamine or to consider stress TC in a well-defined category of patients. 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 [9]. 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 [10]. Unfortunately, studies are few and often retrospective with many limitations, so further work is needed to achieve more meaningful data. 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.

To this group belongs the patient of our clinical case in which a reiterating and non-disabling chest pain not followed by enzymatic withdrawal and/or significant electrocardiographic changes (INOCA) trace probably contributed to the difficult interception and not before the consolidation of the myocardial damage. The tool that eventually allowed diagnosis, and often allows it, was cardiac MRI because through the late gadolinium enhancement (LGE) it identified and localised the damaged areas characterising the

tissue phenotype. Unfortunately, all this occurred when tissue damage was already present, even though for most patients with MINOCA it is often 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 corobary 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).

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.

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