

The fundamental role of optical coherence tomography in MINOCA: a case of STEMI and plaque ulcer in the left anterior descending artery

El rol fundamental de la tomografía de coherencia óptica en el MINOCA: un caso de SCACEST y úlcera de placa en la arteria descendente anterior

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ABSTRACT

Myocardial infarction with non-obstructive coronary arteries (MINOCA) accounts for between 1% and 15% of all acute coronary syndrome cases. Its diagnosis poses a challenge and intravascular imaging plays a crucial role in determining its etiology. We describe the case of a patient with anterior ST-elevation acute coronary syndrome (STEACS) Killip and Kimball III without angiographically significant coronary stenosis on coronary angiography. Optical coherence tomography showed evidence of ulcerated atherosclerotic plaque in the proximal third of the left anterior descending artery.

Keywords: acute coronary syndrome, MINOCA, optical coherence tomography.

RESUMEN

El infarto de miocardio sin obstrucciones coronarias significativas (MINOCA) constituye entre el 1 al 15% de los casos de síndromes coronarios agudos. Su diagnóstico representa un desafío en el cual las imágenes intravasculares juegan un rol crucial para determinar su etiología. Se describe el caso de una paciente con síndrome coronario agudo con elevación del segmento ST anterior Killip y Kimball III sin estenosis coronarias angiográficamente significativas en la coronariografía, a quien se le realizó una tomografía de coherencia óptica con evidencia de placa aterosclerótica ulcerada en el tercio proximal de la arteria descendente anterior.

Palabras clave: síndrome coronario agudo, MINOCA, tomografía de coherencia óptica.

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INTRODUCTION

Myocardial infarction with non-obstructive coronary arteries (MINOCA) accounts for between 5% and 15% of all cases of acute coronary syndrome (ACS), depending on the studied population¹. The initial conception that MINOCA is a benign condition has been revised in recent years. It is now clear that these patients have lower survival rates and are at a higher risk of adverse events compared to healthy individuals of the same age and sex². This is a heterogeneous phenomenon, since it can derive from diverse factors, including atherosclerotic causes, such as coronary plaque disruption (rupture, erosion, ulceration, or calcified nodules), and non-atherosclerotic causes, such as spontaneous dissection, vasospasm, embolism, and microvascular dysfunction¹.

The etiological diagnosis of MINOCA can be challenging, but it is fundamental, as treatment will depend on the underlying cause. Consequently, thorough patient assessment is crucial in these cases. Current consensus recommends a multimodal imaging approach to better understand the involved pathophysiology^{1, 2}. Intracoronary imaging plays a key role in the precise identification of MINOCA causes. For this reason, when possible, optical coherence tomography (OCT) or intravascular ultrasound (IVUS) are recommended, especially when plaque disruption is suspected. OCT offers ten times greater resolution than IVUS regard-

ing internal wall structures and tissue characteristics, making it the preferred choice in these scenarios^{2, 3}.

CLINICAL CASE

We present the case of an 82-year-old hypertensive woman with no known cardiovascular history and a diagnosis of multiple myeloma relapsed after multiple lines of therapy, who was undergoing chemotherapy treatment with daratumumab, monthly dexamethasone, and pomalidomide. She was admitted at the Emergency Department with 24-hour progressive dyspnea up to functional class IV, without other associated symptoms.

Upon admission, the patient was hypertensive, saturating 89% on room air, tachycardic, and tachypneic. For this reason, airway protection was ensured by orotracheal intubation.

Admission laboratory tests showed normocytic normochromic anemia (hemoglobin 10 g/dL, similar to previous values), preserved renal function, and elevated high-sensitivity troponin (1486 pg/mL; normal value up to 14 pg/mL). The admission electrocardiogram showed sinus rhythm at 100 bpm with ST-segment elevation in the anterior leads (**Figure 1**). The echocardiogram revealed new moderate to severe left ventricular systolic dysfunction with anteroseptal akinesia from base to apex, anterior medial, and apical anterior segments. Interpreting the presentation as an anterior ST-elevation ACS Killip and Kimball III, the patient received a loading dose of aspirin and clopidogrel, and emergency cardiac catheterization with possible angioplasty was requested.

The study revealed coronary arteries with wall irregularities, without angiographically significant stenosis, and

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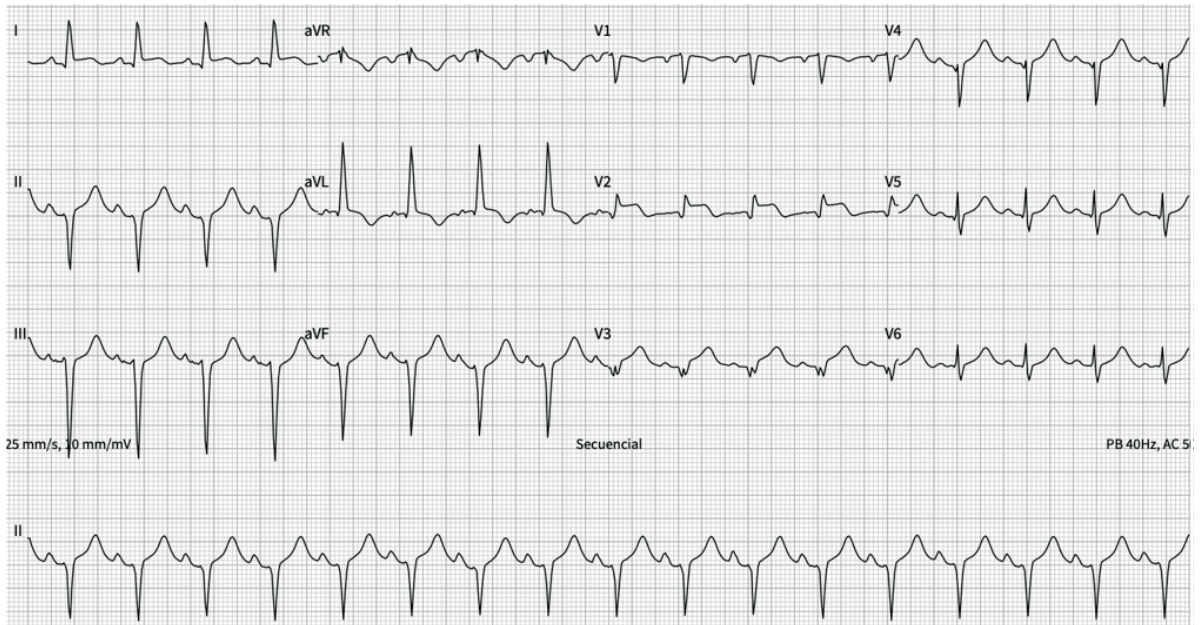


Figure 1. Admission ECG. Sinus rhythm at 100 bpm; left axis deviation. Narrow QRS with ST-segment elevation in leads V2-V4 and I, V2-V4 y DI.

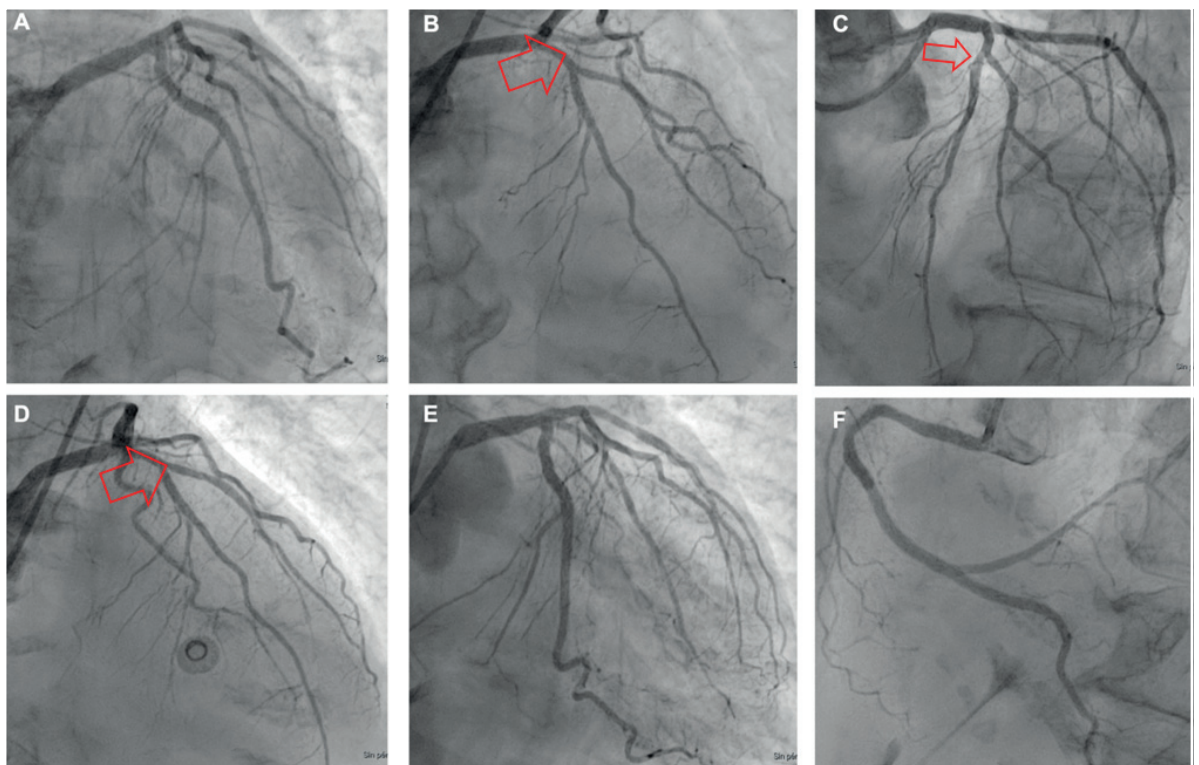


Figure 2. Emergency diagnostic cardiac catheterization. Left coronary artery: A) Right caudal projection. B) Strict cranial projection. C) Left cranial projection. D) Right cranial projection. E) Strict caudal projection. There are wall irregularities without angiographically significant stenosis. There is a 20-30% stenosis in the proximal third of the left anterior descending artery (red arrow), with TIMI III flow. Right coronary artery: F) Left oblique projection. There are wall irregularities without angiographically significant stenosis.

TIMI III flow (**Figure 2**). Given this situation and having interpreted the clinical setting as MINOCA, the next step was intracoronary imaging. In this case, OCT was chosen due to its higher definition and ability to visualize the different layers of the arterial wall and characterize atherosclerotic plaque. There was atherosclerotic plaque in the proximal third of the left anterior descending artery, immediately proximal to the origin of the first large diago-

nal branch, a hypointense and homogeneous image with a thin fibrous cap and discontinuity, compatible with ulcerated lipid plaque (**Figure 3A**). Additionally, a few millimeters distally, there was a crescent-shaped hyperintense image in the medium layer, compatible with intramural hematoma (**Figure 3B**). It should be noted that there was no endoluminal material compromising the vessel lumen (**Figures 3A and B**).

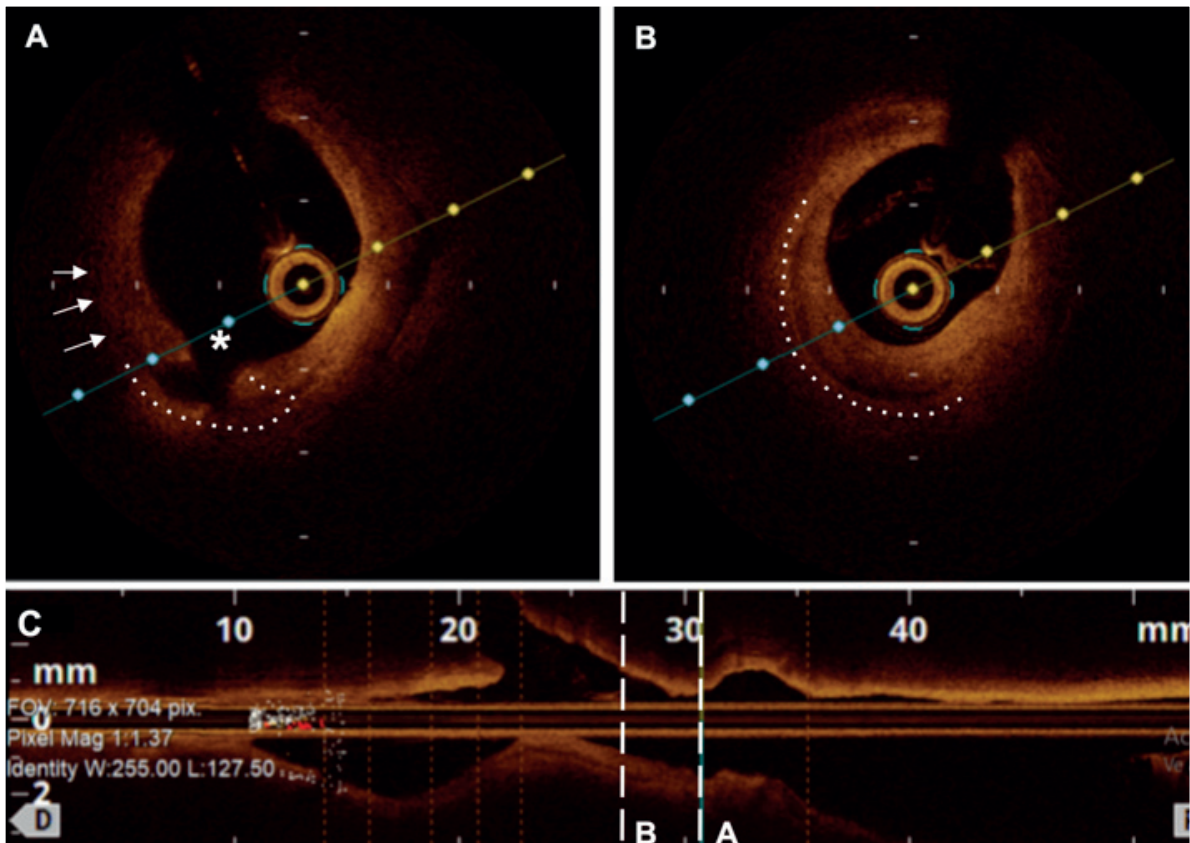


Figure 3. Optical coherence tomography images: cross-sectional (A, B) and longitudinal (3A) cuts of the left anterior descending artery. A) Hypointense and homogeneous image (arrows) with a thin fibrous cap with discontinuity (asterisk) compatible with ulcerated plaque (dotted line). B) Crescent-shaped hypointense image in the medium layer compatible with intramural hematoma (dotted line).

Since the patient remained hemodynamically stable, without the need for vasoactive drugs, with TIMI III flow in the left anterior descending artery and an acceptable intraluminal area, a conservative strategy was chosen and the procedure was concluded. The patient remained in the coronary unit and received medical treatment with dual antiplatelet therapy with aspirin and clopidogrel. She had a favorable evolution and was discharged, continuing outpatient cardiology follow-up without events at 6 months.

DISCUSSION

This case is particularly interesting for multiple reasons. First, our patient presented with ST-elevation acute coronary syndrome (STEACS) Killip and Kimball III, demonstrating that MINOCA can have a significant clinical impact with more severe implications. There is an underrepresentation of MINOCA patients in the context of ST-elevation ACS, as it more frequently presents as a non-ST elevation acute coronary syndrome (8-10% vs. 2.8-4.4%)⁴. Determining its true incidence is key, as these patients may require specific studies and treatments. Moreover, MINOCA is not a benign condition: it has a mortality rate of 3.6% at 30 days and 4.5% at one year in patients who experience STEACS⁵. Second, the case exemplifies the fundamental role of intravascular imaging in identifying the underlying cause of MINOCA. OCT allowed us to confirm atherosclerotic plaque disruption (plaque ulceration). This can trigger thrombi formation, causing infarction by distal embolization, superimposed coronary spasm, or even transient complete thrombosis with spontaneous thrombolysis¹. It is also important

to emphasize that OCT timing is crucial in the assessment of MINOCA, as it yields high diagnostic value when performed simultaneously with the initial angiography^{3,6}. This imaging-based approach allows for the immediate identification of potential culprit lesions not usually visible by angiography alone, as illustrated in our clinical case³. Reynolds *et al.* reported that, in women with MINOCA who underwent OCT immediately after cardiac catheterization, culprit or probable lesions were identified in 46% of participants, highlighting the usefulness of this technique in the acute phase⁶. Furthermore, integrating OCT with other cardiovascular imaging modalities, such as cardiac magnetic resonance imaging (CMR), further improves diagnostic accuracy. It has been demonstrated that combining OCT with CMR can identify the underlying cause of MINOCA in a significant proportion of patients, thus guiding subsequent management and secondary prevention strategies⁷. For these reasons, the literature generally supports the use of OCT during initial angiographic evaluation in patients with MINOCA to rapidly identify underlying pathologies and guide clinical and therapeutic decision-making.

OCT overcomes some limitations of cardiac catheterization by providing real-time, 360° cross-sectional visualization, which is crucial for the identification of the various differential diagnoses of MINOCA, which can include atherosclerotic and non-atherosclerotic causes^{1,8}. The former, such as rupture, erosion, ulceration, and calcified nodules, account for 40% of all MINOCA cases. In plaque rupture, there is discontinuity of the fibrous cap, while erosion is characterized by thrombus without signs of rupture. The calcified nodule appears as a low-signal area protruding into the lu-

men. Regarding non-atherosclerotic causes, OCT can detect spontaneous coronary artery dissection, characterized by separation of the coronary wall layers due to intramural hemorrhage, with or without intimal tear. The aforementioned tool is also useful to distinguish thromboembolic mechanisms causing MINOCA⁸. In our case, the high resolution of OCT allowed us to clearly diagnose lipid plaque ulceration associated with intramural hematoma (**Figure 3**). Finally, since evidence on MINOCA treatment is limited, it is important to report the therapeutic strategy adopted in our case. While this is a controversial topic, position papers suggest that MINOCA patients with plaque disruption could benefit from antiplatelet therapy with aspirin, since the pathophysiology resembles that of type 1 myocardial infarction. Additionally, the inclusion of a second antiplatelet agent, such as clopidogrel in our case, is considered reasonable¹. On the other hand, angioplasty is not our usual strategy in patients with MINOCA and plaque disruption, as evidence supporting this practice is scarce¹. Indeed, a small study that assessed MINOCA patients treated with dual antiplatelet therapy only reported an acceptable revascularization rate of 5.7% at one year after the event⁹. Moreover, whi-

le the incidence of angioplasty-related complications is currently low, the risk of adverse events such as stent thrombosis, restenosis, or neoatherosclerosis cannot be ignored¹.

CONCLUSION

This case reflects the importance of thorough diagnostic assessment in patients with MINOCA to elucidate the underlying etiology. In particular, the use of intracoronary imaging techniques, such as OCT or IVUS, is essential not only to reach an accurate diagnosis but also to guide therapeutic decisions that are fundamental to improving patient prognosis. Specifically, this intravascular-imaging-focused diagnostic approach allows for a more precise evaluation and better identification of underlying causes, which can modify the therapeutic strategy.

Our case demonstrates how intravascular imaging can transform the diagnostic approach in patients with MINOCA, highlighting the need to use these technologies in specific clinical situations where they can make a significant difference. More frequent use of these tools may be crucial to optimize treatment and improve long-term outcomes.

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