Case Report

A Young Patient with Chest Pain: Going Beyond the Obvious

Paciente Jovem com Dor Torácica: Além do Óbvio

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Abstract

We describe the case of a young patient with a history of pulmonary embolism and a first-trimester spontaneous abortion, who presented with sudden-onset chest pain and dyspnea. After a systematic retrospective review of the patient's clinical history and work-up, she was diagnosed with myocardial infarction with nonobstructive coronary arteries secondary to coronary thrombosis. Background: Myocardial infarction with nonobstructive coronary arteries (MINOCA) is diagnostically and therapeutically challenging for clinicians, as a large spectrum of causes may result in this clinical condition, multiple entities can mimic it, and there are relatively few evidence-based recommendations for its assessment. Discussion: Accurate diagnosis of the underlying etiology of MINOCA requires a systematic and dynamic evaluation of the clinical history and work-up of each patient, as well as the use of additional diagnostic tools that allow more in-depth characterization of the cardiac anatomy and coronary function, such as cardiac magnetic resonance imaging, coronary vascular imaging (intravascular ultrasound, optical coherence tomography), and coronary functional assessments. The prognosis of MINOCA highly depends on its underlying cause, and therefore, an accurate diagnosis of its etiology is necessary.

Introduction

Myocardial infarction with nonobstructive coronary arteries (MINOCA) is diagnostically and therapeutically challenging for clinicians, as a large spectrum of causes may result in this clinical condition, multiple entities can mimic it, and there are relatively few evidence-based recommendations for its assessment.[1,2] Current evidence suggests that a diagnosis of MINOCA should only be considered after confirming the presence of acute coronary syndrome (ACS), as defined by the "Fourth Universal Definition of Myocardial Infarction,"[3] and after ruling out the coexistence of any other etiology that may cause myocardial injury (such as sepsis, pulmonary embolism [PE], trauma, etc.) and the presence of obstructive coronary arteries on coronary angiography.[1] Once the diagnosis of MINOCA is established, further investigations are necessary to clarify the underlying mechanism, so that appropriate therapeutic actions can be implemented.[4]

In order to achieve an accurate diagnosis of the underlying etiology, a systematic and dynamic evaluation of the patients' clinical history, as well as the use of additional diagnostic tools to better characterize the cardiac anatomy and coronary function, such as cardiac magnetic resonance imaging (cMRI), coronary vascular imaging (intravascular ultrasound, optical coherence tomography), and coronary functional assessments. Herein, we present the case of a young female patient with a previous history of PE who presented with chest pain, in whom, an underlying hypercoagulability syndrome led to a subsequent thrombotic event leading to a MINOCA. In all studies, a high degree of clinical suspicion and a thorough interpretation were necessary in order to reach an accurate diagnosis.

Case presentation

A 19-year-old woman presented to the emergency department with sudden-onset of intense oppressive substernal chest pain that radiated to the back and neck, and was accompanied by diffuse epigastric discomfort and severe shortness of breath. Physical examination revealed blood pressure of 132/89 mmHg, heart rate of 107 beats/min, respiratory rate of 30 breaths/minute, and oxygen saturation of 93% at room air. Her heart sounds were tachycardic but regular. Cardiac examination was otherwise unremarkable, without murmurs, gallops, or rubs. No other abnormal findings on examination were observed. Three months earlier, after a spontaneous first-trimester abortion, she presented with dyspnea and severe chest pain, accompanied by intense dizziness and palpitations. On that occasion, she was found to have multiple PE. She was discharged with enoxaparin therapy, but her compliance was suboptimal.

The initial differential diagnosis of this young patient with previous history of PE and poor adherence to anticoagulation therapy, who presented with new-onset chest pain, dyspnea, and tachycardia, was directed toward a possible recurrence or worsening of the recent PE. However, the signs and symptoms in the current presentation were different from those in the prior episode, leading the team to pursue work-up of other differential diagnoses; these included ACS, in the context of coronary artery dissection or coronary vasospasm (due to her young age, coronary atherosclerotic disease was unlikely), aortic dissection, and an inflammatory condition, such as pericarditis or myocarditis.

Keywords

Chest pain, Pulmonary embolism, Antiphospholipid syndrome.

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Baseline laboratory tests revealed normal renal and hepatic function, normal complete blood count, and normal electrolytes. Computed tomography angiography showed no evidence of aortic dissection. Pulmonary circulation evaluation revealed partial resolution of the previous PE, with no new acute findings.

Electrocardiogram showed sinus rhythm, with ST-segment depression in the anterolateral wall leads and ST-segment elevation in aVR (Figure 1). Her high-sensitivity troponin I concentrations were 4.2 ng/mL on presentation, and 3.4 ng/mL 4 hours later (normal < 0.026 ng/mL). A bedside transthoracic echocardiogram (TTE) revealed mildly reduced systolic function with a left ventricular ejection fraction (LVEF) of 45% (Video 1). Anterior wall hypokinesia with mild pericardial effusion was observed. The right ventricle had normal morphology and function. Despite her young age, ACS was considered to be the most likely diagnosis.

Initial coronary angiography showed no angiographic stenosis (normal coronary arteries). A working diagnosis of MINOCA was established. Considering the current recommendations for work-up of MINOCA, further investigation to exclude clinically elusive nonischemic mechanisms of myocardial injury was considered.[1] cMRI showed hypokinesia in the mid-segment of the anterolateral wall (Video 2), as well as a perfusion defect at the anterolateral papillary muscle and in the mid-segment of the anterolateral wall (Figures 2A and B; Video 3). Subendocardial late gadolinium enhancement (LGE) was observed at the basal

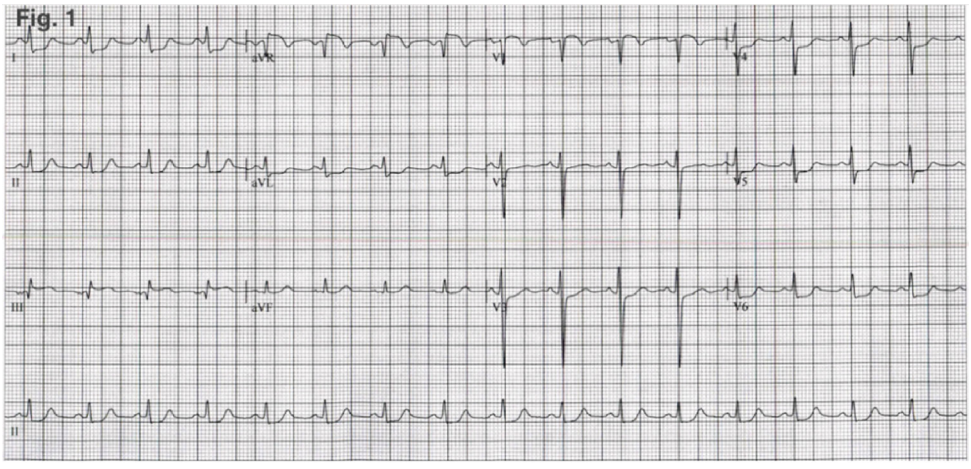
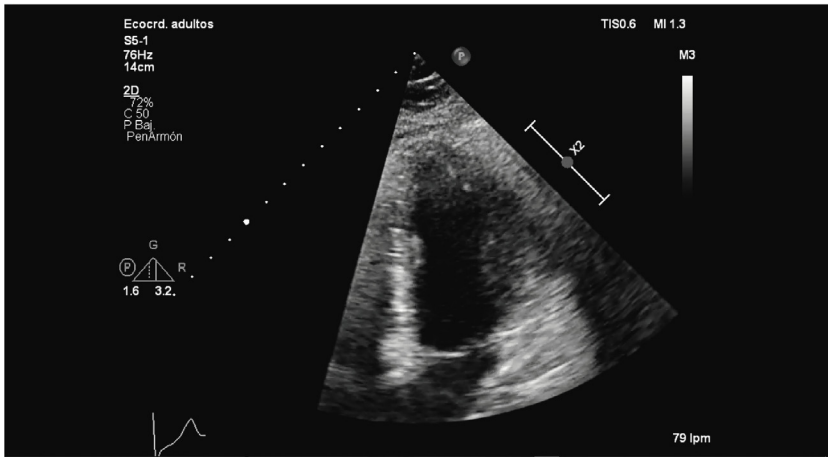
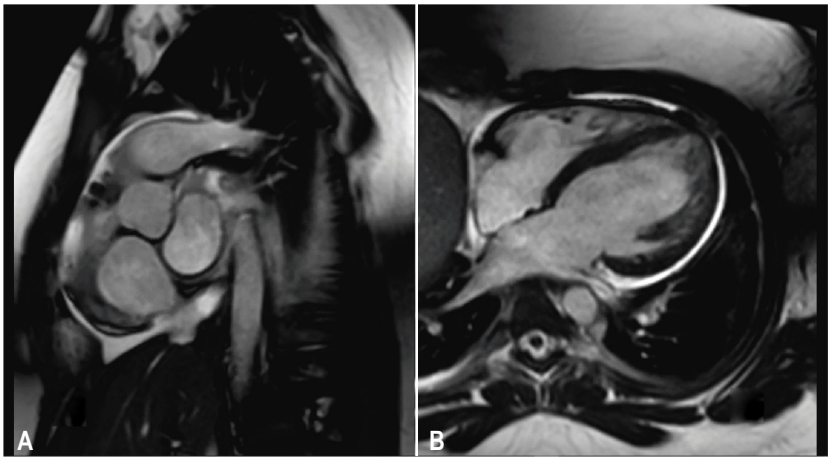


Figure 1 – *A 12-lead electrocardiogram showing ST -segment depression in the anterolateral wall leads and ST-segment elevation in aVR .*



Video 1 – *Transthoracic echocardiogram (TTE): apical four-chamber view. TTE showing mildly reduced systolic function (left ventricular ejection fraction of 45%), with anterolateral wall hypokinesia and mild pericardial effusion. The right ventricle had normal morphology and function, with a preserved ejection fraction.*



Video 2 – *Cardiac magnetic resonance: cine sequences. Short-axis (A) and horizontal long axis view (B) showing hypokinesia of the anterolateral mid-segment.*

segment of the anterior wall (Figure 2C), as well as edema in the T2-STIR sequence (Figure 2D). Together, these results confirmed the presence of myocardial infarction.

An exhaustive review of coronary angiography images suggested that the cause of the MINOCA was a possible re-canalized thrombus with microvascular obstruction of the small diagonal branch in the left anterior descending artery. Initial anticoagulation with intravenous unfractionated heparin was decided, and she was subsequently transitioned and discharged with enoxaparin. Considering her past medical history (spontaneous abortion and family history of autoimmune disease), thrombophilia work-up was performed, which confirmed the diagnosis of antiphospholipid syndrome.

A TTE 4 weeks after the initial presentation showed normalization of the previously depressed LVEF and resolution of the regional wall motion abnormalities. No long-term clinical sequelae were evident during her last visit to our outpatient clinic. Compliance with anticoagulation therapy was emphatically addressed. Due to the diagnosis of antiphospholipid syndrome, permanent anticoagulation was decided, with follow-up plans at her local anticoagulation clinic.

Discussion

We present the case of a young female patient with previous history of PE who presented with chest pain, in whom, an underlying pathophysiology of hypercoagulability led to a subsequent thrombotic event and MINOCA. Careful work-up for hypercoagulability led to the final diagnosis of antiphospholipid syndrome. A high degree of clinical suspicion and a thorough interpretation of all studies were necessary to reach an accurate diagnosis. Her past medical history of PE and first-trimester spontaneous abortion, in addition to the thrombotic obstruction of a coronary artery were just the manifestations of the hypercoagulable state caused by antiphospholipid syndrome.

MINOCA is diagnostically and therapeutically challenging for clinicians.[1,2] The optimal evaluation for patients with an established diagnosis of MINOCA should be focused on exploring and determining its specific cause. A systematic evaluation of the individual's clinical history and a dynamic work-up is necessary to determine the underlying etiology of MINOCA, in order to provide the most appropriate management for each causal mechanism.

As illustrated in this case report, cMRI is a key diagnostic tool for the assessment of patients presenting with MINOCA, owing to its safety and ability to characterize the myocardium with high definition and low inter-observer variability.[2] In this way, cMRI is useful to confirm the diagnosis of ACS, and to rule out entities such as myocarditis, takotsubo cardiomyopathy, and other cardiomyopathies.

The causes of MINOCA can be divided into atherosclerotic and non-atherosclerotic. Non-atherosclerotic causes of MINOCA include epicardial coronary vasospasm, coronary dissection, coronary microvascular dysfunction, supply/demand mismatch, and coronary thrombosis/embolism. As illustrated by the current case, coronary thrombosis may result in MINOCA if transient complete thrombosis with spontaneous thrombolysis results in nonobstructive angiographic disease, or if the thrombus involves the downstream microcirculation[1]. Hypercoagulable states, such as antiphospholipid syndrome, significantly increase the risk of venous and arterial thrombosis and embolisms. However, coronary thrombosis is not a common initial presentation. Venous thromboembolisms are more common, and within arterial thrombosis, strokes are significantly more prevalent than ACS.[5] Nonetheless, in patients with coronary embolism, approximately 7.5% were later found to have antiphospholipid syndrome.[5]

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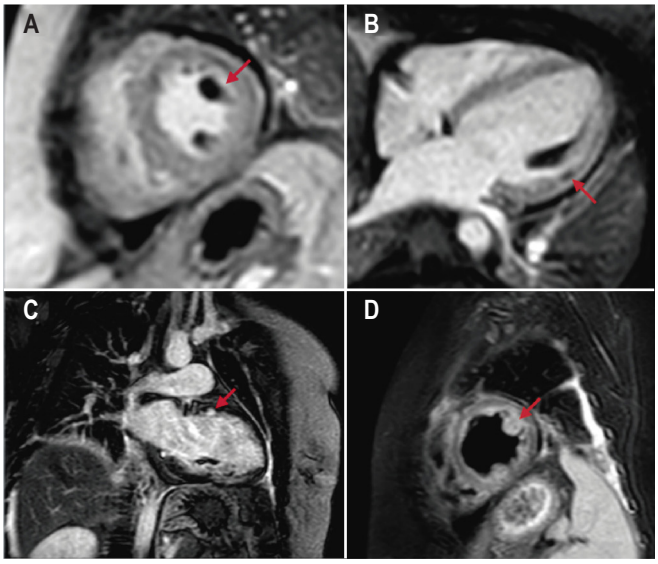
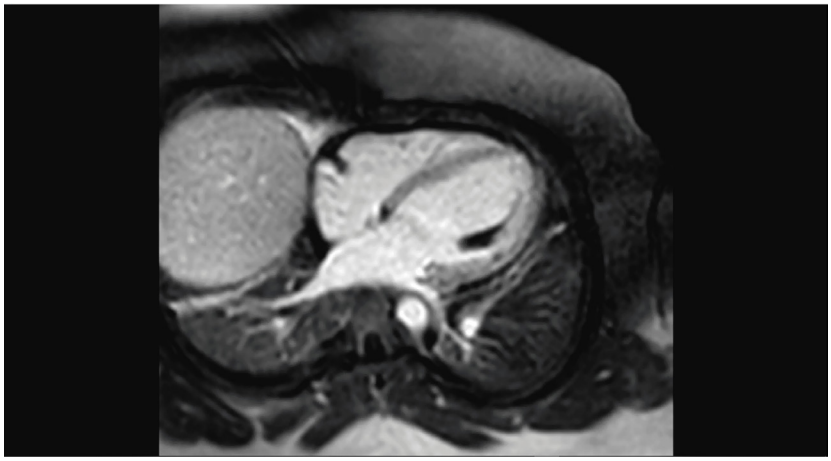


Figure 2 – *Cardiac magnetic resonance imaging. Perfusion sequences showing a perfusion defect of the anterolateral (red-arrow) and posteromedial (blue-arrow) papillary muscles (A), as well as in the mid-segment of the anterolateral wall (red-arrow on B). Subendocardial Late Gadolinium Enhancement (LGE) at the basal segment of the anterior wall (red-arrow on C). STIR-T2 sequence showing myocardial edema in the anterolateral papillary muscle (red-arrow on D).*



Video 3 – *Cardiac magnetic resonance imaging: perfusion sequence. Perfusion defect of the anterolateral and posteromedial papillary muscles, as well as in the mid-segment of the anterolateral wall.*

Consent

The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

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Authors' contributions

Research creation and design: Bernal SG; Vasquez-Rodriguez JF; Isaza D; Calixto CA; Jaimes C.: Data acquisition: Bernal SG; Martínez A; Vasquez-Rodriguez JF; Isaza D; Calixto CA; Jaimes C.: Data analysis and interpretation: Bernal SG; Martínez A; Isaza D; Calixto CA; Jaimes C.: Writing: Bernal SG; Isaza N; Isaza D; Jaimes C.: Critical revision for important intellectual content: Bernal SG; Martínez A; Vasquez-Rodriguez JF; Isaza N; Isaza D; Jaimes C

Conflict of interest

The authors have declared that they have no conflict of interest.

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