

MINOCA Resulting from Coronary Spasm Confirmed Angiographically without Ergonovine Stimulation: A Case Report

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Abstract

Introduction: Myocardial infarction with nonobstructive coronary arteries disease (MINOCA) encompasses a heterogeneous group of disorders. Multimodality imaging is crucial to figure out the underlying mechanism. Coronary artery spasm is a common cause of MINOCA and might be difficult to identify. **Case Presentation:** A 43-year-old male with no cardiovascular risk factor presented with prolonged acute chest pain at 5 am that resolved spontaneously. On admission, he was asymptomatic and clinical findings were unremarkable. The electrocardiogram showed an anterolateral negative T wave. The troponin was positive. Coronary angiography (CA) revealed a moderate lesion of the proximal left anterior descending (LAD) coronary artery. He was treated with dual antiplatelet therapy, statin, and beta-blocker with a favourable hospital course. One week later, he presented chest pain recurrences with anterior ST-segment elevation. CA, without intra-radial nitrate, owing to a vasospasm suspicion, showed a sub-occlusion of the proximal LAD which resolved after intracoronary nitrates. Chest pain and ST-segment elevation then disappeared. He remained asymptomatic and was discharged on CCB (calcium channel blocker), long-acting nitrates dual antiplatelet and statin. Cardiac magnetic resonance (CMR) demonstrated an ejection fraction (EF) of 45% with anterior ischemia. **Conclusion:** Our case is about a common cause of MINOCA, namely coronary spasm, diagnosed by coronary angiogram without provocative test.

Keywords

MINOCA, Coronary Spasm, Case Report

Learning Objectives

- MINOCA is not exceptional and may be due to coronary artery vasospasm.
- A provocative test by ergonovine is necessary to confirm the diagnosis of coronary spasm most of the time, even though risky in the acute setting.
- Multimodality imaging including cardiac magnetic resonance and intracoronary imaging is necessary to elucidate the underlying mechanism of MINOCA.

1. Introduction

Myocardial infarction with nonobstructive coronary arteries disease (MINOCA) is a growing subject of interest in the field of cardiology. Tremendous efforts have been made to elucidate the definition and potential causes. Identifying the latter might be very challenging and require multimodality imaging including a coronary angiogram, cardiac magnetic resonance (CMR) and intracoronary imaging. Coronary artery spasm, known classically as Prinzmetal angina, is one of the main causes of MINOCA. The former's diagnosis remains difficult due to the broad spectrum of clinical presentations ranging from transient angina to cardiac arrest. An invasive approach with an ergonovine test may be necessary to confirm the diagnosis even though not routinely performed, unless for typical clinical presentation.

We report a case of a young male patient presenting a MINOCA with coronary spasm as an underlying mechanism.

2. Case Presentation

A 43-year-old male presented with acute chest pain, radiating in both arms and the back at 5 am, lasting for around 30 min and resolving spontaneously. He described a similar episode one week earlier.

He had smoking as a single risk factor for cardiovascular disease with no past medical history. He denied alcohol and recreational drug consumption. His past medical history is unremarkable. He reported smoking cessation for more than 2 years. Despite this acute and severe chest pain, he delayed consultation until 13:00 am in a general hospital. The 12-lead electrocardiogram (ECG), performed (while the patient was asymptomatic) revealed a sinus rhythm with a deep negative T wave in the anterior and lateral leads (**Figure 1(a)**). Initial troponin was 15XN prompting the Cardiologist to administer loading doses of Aspirin 300 mg and Clopidogrel 600 mg orally along with low molecular weight heparin (LWMH) and Bisoprolol 2.5 mg orally. The patient was then transferred to our centre, which is a tertiary one with a catheterization laboratory.

He was admitted to our Coronary Care Unit (CCU), he was asymptomatic and afebrile; his blood pressure was 106/69 mmHg, pulse 55/min, Saturation 98% (room air), and his physical examination was unremarkable. The ECG was similar to the previous one (**Figure 1(b)**).

Repeated troponin at arrival and 12 hours later were 38N and 24N respectively.



Figure 1. ECG. The first ECG showed sinus rhythm with a deep negative T wave in the anterior and lateral leads (a). ECG was performed upon his arrival in the CCU (b) and in post-critical during the second hospital admission (c) with both similar findings as the first one. ECG, performed per-critical during the second hospital admission (d) revealed ST segment elevation in leads V2, V3 and V4 with positive T wave in leads V5, 6, and D1 (see red arrows). ECG was performed post intracoronary nitrate administration in the catheterization laboratory (e) with the resolution of ST-segment elevation in leads V2, 3 and 4; negative T waves in anterior and lateral leads.

He was kept on dual antiplatelet therapy (Aspirin 100 mg + Clopidogrel 75 mg), LWMH, bisoprolol and atorvastatin 80 mg.

The echocardiogram demonstrated hypokinesia of the anterior segment with preserved ejection fraction and excluded pericardial effusion, significant valve disease and mechanical complications.

The coronary angiography (CA) performed 48 h later by transradial access revealed a coronary artery tree free of significant stenosis except a moderate (<50%) atherosclerotic plaque on the proximal segment of the left anterior descending (LAD) coronary artery (**Figure 2(a)**).

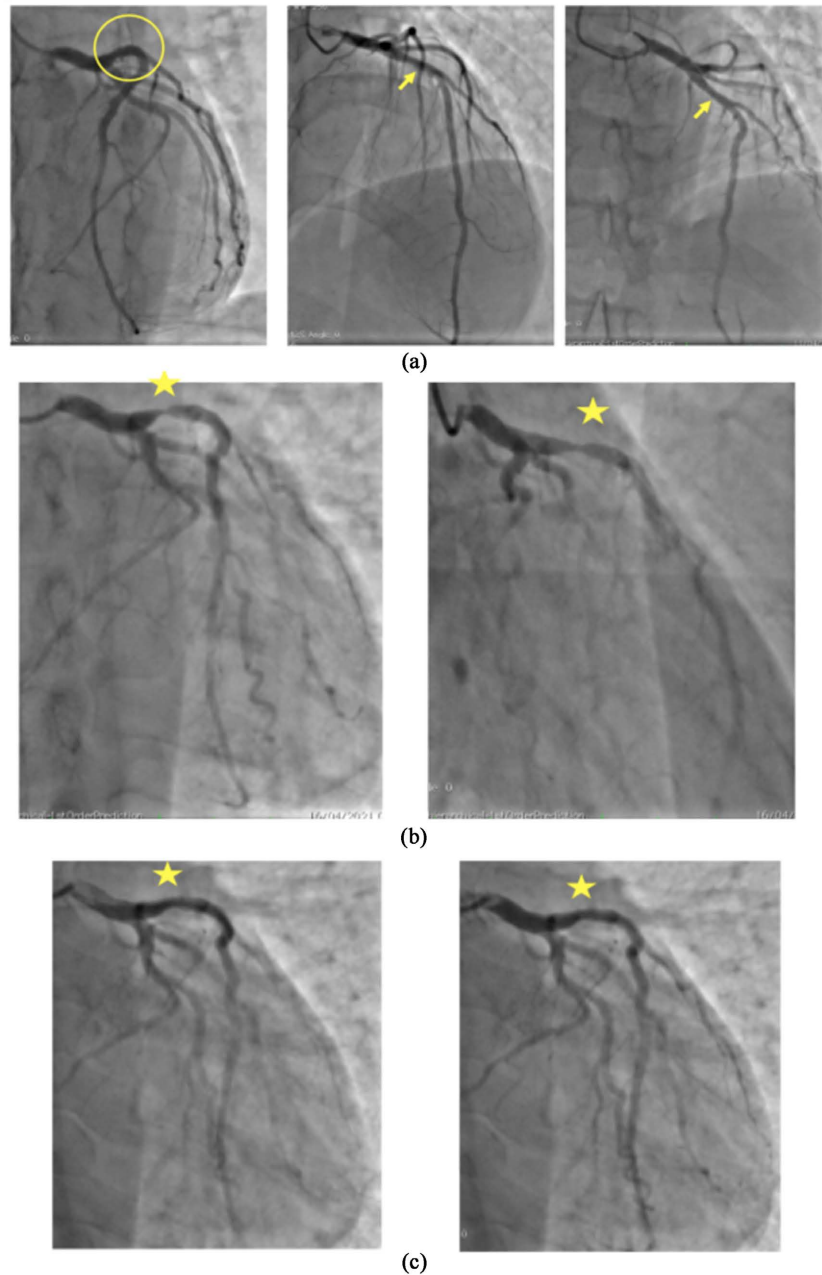


Figure 2. Coronary angiogram (CA). Initial CA (a) showing moderate (<50%) atherosclerotic plaque on the proximal segment of the left anterior descending (LAD) coronary artery (see yellow circle and arrows). The other coronary arteries were normal. Repeated invasive coronary angiogram during chest pain recurrence (b) with subtotal focal occlusion of the proximal segment of the LAD, on the site of the atherosclerotic plaque (see yellow star). Repeated invasive coronary angiogram during chest pain recurrence (c) with a resolution of subtotal occlusion of the proximal LAD (see yellow star) after intracoronary nitrate administration.

The patient was kept on the same treatment with a favourable hospital course. He was discharged with the same medications part from LMWH and CMR was planned.

One week later, the patient is awakened around 5 am by acute constrictive chest pain lasting for 30 min, associated with diaphoresis, nausea and vomiting. He consulted directly with our emergency department. However, chest pain resolved spontaneously meanwhile and ECG recording (post-critical) revealed similar findings to the first ones (**Figure 1(c)**). While waiting for the troponin result, he complained about ongoing chest pain and the ECG, performed per-critical, showed ST segment elevation in leads V2, V3 and V4 with positive T wave in leads V5, 6, and D1 (**Figure 1(d)**).

We decided to repeat CA by transradial route, but without intra-radial nitrate or calcium channel blocker (CCB) administration, owing to a coronary spasm suspicion. The patient was still in pain. The CA revealed a subtotal focal occlusion of the proximal segment of the LAD, on the site of the atherosclerotic plaque; the other coronary arteries were diseased-free (**Figure 2(b)**). The stenosis resolved after 2mg of intracoronary isosorbide dinitrate administration with a residual plaque of less than 50% (**Figure 2(c)**). The chest pain and ST-segment elevation disappeared (**Figure 1(e)**). The patient was admitted to the CCU for surveillance and treated with CCB and long-acting nitrates; the beta-blocker was removed. He was asymptomatic without chest pain recurrence. The cardiac magnetic resonance (CMR) performed, a few days later, demonstrated a left ejection fraction of 45% with anterior ischemia without late gadolinium enhancement (**Figure 3**). The patient remained free from chest pain recurrences 2

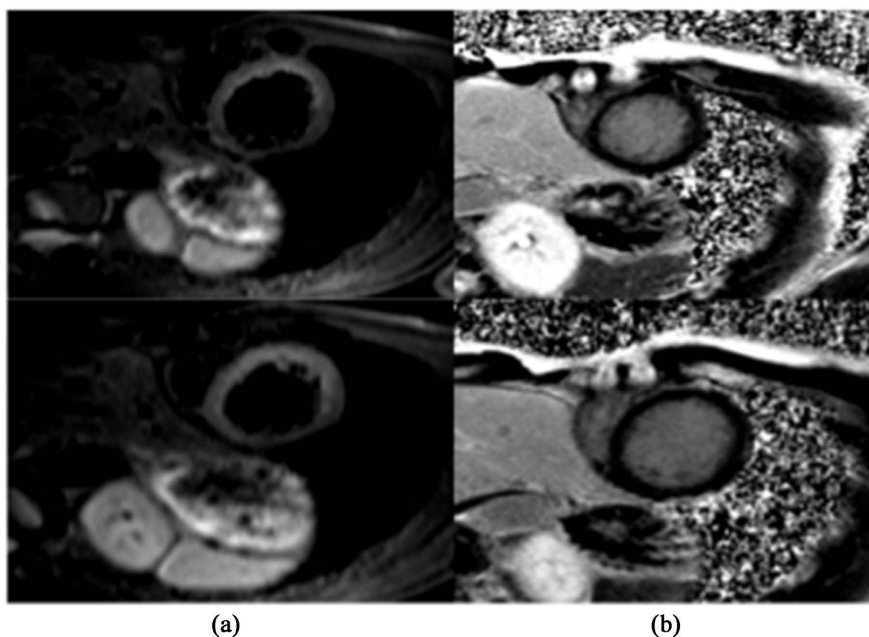


Figure 3. Cardiac magnetic resonance (CMR). (a) STIR technique: ischemia (subendocardial oedema) of the anterior territory; (b) PSIR technique: absence of late gadolinium enhancement.

months later and completed cardiac rehabilitation. He was seen on consultation 6 months later and was symptom-free.

3. Discussion

Our patient presented an established anterior myocardial infarction (MI) diagnosis according to the 4th Universal definition of MI [1] without obstructive coronary artery disease explaining the MI and without an objective alternative diagnosis. That clinical scenario is the current definition of myocardial infarction with nonobstructive coronary arteries disease (MINOCA) according to the European Society of Cardiology (ESC) guidelines in conjunction with the American Heart Association (AHA) [2] [3]. MINOCA is known to be more frequent in young women, and commonly not associated with a traditional risk factor of cardiovascular disease [4]. It represents around 9% of myocardial infarction cases [5].

Until recently, MINOCA was a very conflictual entity due to heterogeneous causes of myocardial injury with or without proven ischemia [6]. Figuring out the underlying cause can be very challenging and require a systematical approach with tapered imaging utilization, especially coronary angiography, intracoronary imaging and CMR. A recent study revealed the high value of multimodality imaging with CMR and optical coherence tomography (OCT) for detecting potential mechanisms. A cause was isolated in 84.5% of women with a diagnosis of MINOCA, 75.5% of which were ischemic and 24.5% of which were non-ischemic, alternate diagnoses to myocardial infarction [7]. A plaque rupture was found with the aid of intravascular ultrasound (IVUS) in approximately one-third of patients with MINOCA diagnosis [8].

During the first hospitalization of our patient, we considered a plaque rupture or erosion of the proximal LAD complicated by thrombus formation with distal embolization as a potential MI cause. Intracoronary imaging, either by OCT or IVUS, would have been of great help to confirm that hypothesis. It was not performed due to a lack of availability. Early atheroma is a plausible and legitimate underlying mechanism for our patient, who had smoking as a single cardiovascular risk factor. However, during his second presentation, a deep investigation regarding the clinical history favoured a coronary artery spasm. The latter is typically undetectable by coronary angiogram without provocation [3]. By chance, we witnessed chest pain recurrences allowing us to confirm angiographically the diagnosis without performing the Ergonovine test, which might be risky in the acute phase. Coronary artery spasm is a frequent aetiology and may reach 46% of patients with MINOCA [3]. It is more common in female and Japanese patients [9]. It may be spontaneous or triggered by smoking or drug abuse such as cocaine, amphetamines marijuana and alcohol consumption, thus explaining MI occurrence in a young patient with few or without the cardiovascular risk factor. Apart from smoking, our patient denied other recreational substance consumption.

The other potential causes for this young patient were coronary embolization (CE) and thrombophilia. CE may require a transesophageal echocardiogram to exclude intracardiac thrombus or patent foramen ovale (PFO) with or without interatrial septal aneurysm.

He was managed as STEMI-like taking into consideration the vasospasm with a treatment comprising dual antiplatelet therapy, calcium channel blocker, long-acting nitrate and short one if needed, angiotensin-converting enzyme (ACE) inhibitor and atorvastatin. He was asymptomatic at 4 months follow-up and completed cardiac rehabilitation successfully.

4. Conclusion

Coronary artery spasm is a common cause of MINOCA. Its diagnosis may be challenging owing to the transient nature of the broad clinical presentations justifying frequently provocative tests by ergonovine even though not always necessary. Multimodality imaging, especially cardiac magnetic resonance and intracoronary imaging, is fundamental to elucidate the underlying mechanism.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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