

Typical ST-segment elevation myocardial infarction with normal coronary arteries: a case report

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Abstract

Myocardial infarction with nonobstructive coronary arteries (MINOCA) is a potentially multipathogenic syndrome that affects a subgroup of patients who present with acute myocardial infarction yet have no significant coronary artery disease on angiography. We herein describe a 71-year-old man with typical angina who showed inferior ST-segment elevation on electrocardiography and an increased troponin-I level. Emergency coronary angiography showed no angiographic stenosis. Cardiac magnetic resonance imaging (CMR) and myocardial contrast echocardiography (MCE) with two-dimensional speckle tracking imaging (2D-STI) were performed after coronary angiography. Good consistency was observed between the CMR findings and MCE with 2D-STI findings in identifying the potential causes of MINOCA. We explored an imaging method that is potentially more effective and accurate than CMR, namely MCE combined with 2D-STI, to identify myocardial abnormalities when angiography reveals no obstruction. This application of MCE with 2D-STI may optimize timely treatment. MINOCA has various causes, and the patient in this case was discharged with aspirin, verapamil, and atorvastatin on the presumption that the infarct had arisen from either plaque disruption or coronary spasm. In this study, we analyzed the etiology, clinical diagnosis, and treatment of MINOCA with reference to the relevant literature.

Keywords

Myocardial infarction, MINOCA, echocardiography, coronary angiography, cardiac magnetic resonance imaging, ST-segment elevation

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Introduction

Myocardial infarction with nonobstructive coronary arteries (MINOCA) is characterized by clinical evidence of acute myocardial infarction (AMI) with normal or near normal coronary arteries on angiography (stenosis severity of $<50\%$ in main epicardial coronary arteries).¹ MINOCA is not uncommon in patients with AMI. A recent systematic review showed that the prevalence of MINOCA ranges from 1% to 14% with an overall prevalence of 6% (95% confidence interval, 5%–7%).² Therefore, it is important to actively use a variety of diagnostic techniques to determine the multiple potential causes of MINOCA, many of which require different treatments. The prognosis is not benign and is likely to be heterogeneous depending on the underlying cause of the infarction.

Case report

A 71-year-old man with a medical history of arterial hypertension presented to the emergency room because of mid-sternal chest pain episodes that lasted from 10 to 20 minutes and had started 2 hours previously.

He reported no radiating pain. On further questioning, the patient stated that he had experienced no chest pain prior to presentation and had no specific disease related to the present chest pain or family genetic history. On arrival, the patient's heart rate was 70 beats/minute and his blood pressure was 100/68 mmHg. Cardiovascular examination findings were normal with an undisplaced apex beat, normal heart sounds, no murmurs or pericardial rub, and normal breath sounds on auscultation. An electrocardiogram was obtained during an episode of chest pain, and ST-segment elevation was recorded in precordial leads II, III, and AVF (Figure 1). The patient's serum cardiac troponin-I level was elevated at 3.78 ng/mL (upper limit of normal, 0.4 ng/mL). A complete blood count, coagulation profile, natriuretic peptide level, and comprehensive metabolic profile were within normal limits. Notably, his high-sensitivity C-reactive protein level, sedimentation rate, and autoimmune panel were negative. The patient was suspected to have an evolving ST-segment elevation myocardial infarction and was immediately admitted for monitoring and treatment with sublingual nitrate and loading doses of aspirin, ticagrelor, and

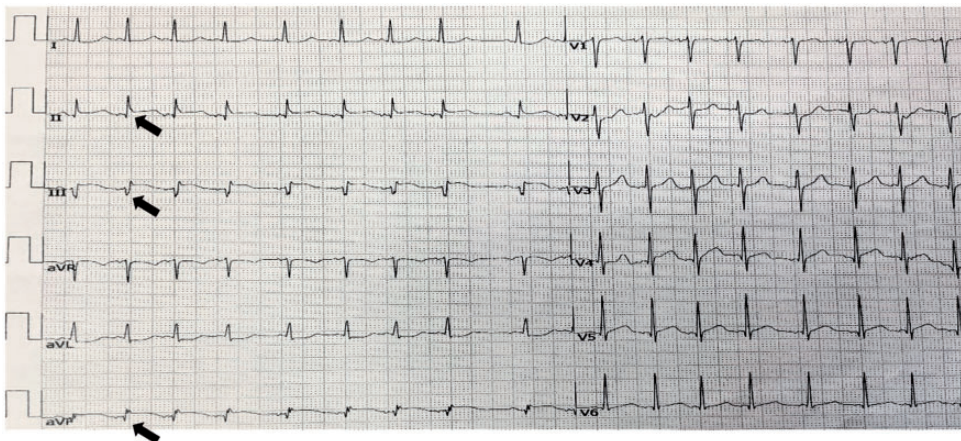


Figure 1. During the chest pain episode, the electrocardiogram showed an ST-segment elevation in precordial leads II, III, and AVF (black arrow).

atorvastatin. Within 1 hour of arrival to the emergency department, he was transferred on an emergency basis to the cardiac catheterization laboratory of our heart attack center. Coronary angiography (CAG) revealed no abnormalities (Figure 2). The troponin-I level rose from 3.78 ng/L on arrival to 15.2 ng/L at 12 hours after hospitalization, and on day 2, an electrocardiogram showed negative T waves in the same precordial leads. His antinuclear antibody spectrum and antiphospholipid antibody were negative.

Angiography revealed no findings that could explain the cause of the acute coronary syndrome; therefore, further examinations were performed. Thoracic enhanced computed tomography with vascular three-dimensional imaging, two-dimensional transthoracic echocardiography (TTE), and cardiac magnetic resonance imaging (CMR) were performed after CAG. Thoracic enhanced computed tomography with vascular three-dimensional imaging showed no abnormal lesions such as aortic dissection, pulmonary embolism, or inflammation. TTE showed enlargement of the left ventricle (inner diameter of 56 mm), a normal left ventricular ejection fraction

(61%), anterior and inferior wall dyskinesia, an akinetic apex, a thickened ventricular septum, enlargement of the inner diameter of the ascending aorta, and aortic valve calcification with regurgitation. The patient subsequently underwent myocardial contrast echocardiography (MCE) with two-dimensional speckle tracking imaging (2D-STI). MCE showed a large microvascular defect in the basal-mid segments of the inferoseptal and inferior walls in the apical four-chamber and two-chamber views and confirmed the absence of any other cardiomyopathy or left ventricular apical thrombosis. 2D-STI showed that the left ventricular global longitudinal strain was significantly reduced, especially in the basal-mid segments of the anterior-inferior septal and inferior walls (Figure 3). CMR performed 48 hours after the onset of chest pain showed an increased left ventricular volume (120.6 mL/m^2) and thinning of the apical ventricular wall with akinetic motion (Figure 4(c), (d)). Cardiac cine images showed mitral valve and aortic valve regurgitation and a small amount of pericardial effusion. No obvious filling defects were present in the first-pass

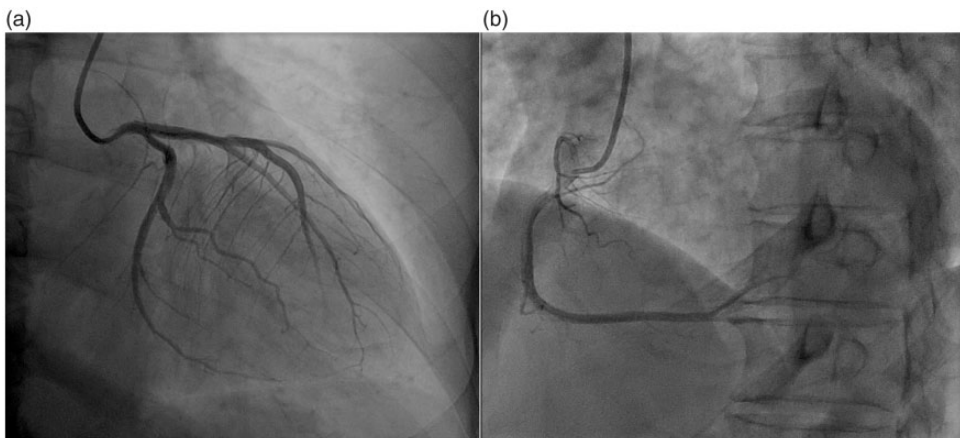


Figure 2. Coronary angiography showed normal coronary arteries. (a) Left coronary artery. (b) Right coronary artery.

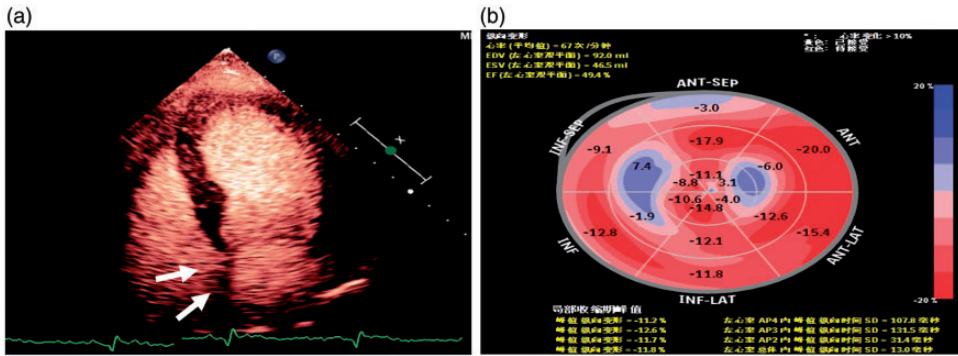


Figure 3. (a) During the myocardial contrast echocardiography procedure 24 hours after successful coronary angiography, a large microvascular defect was still present in the right coronary artery territory in the apical two-chamber view. (b) The 17-segment bulls-eye model showed that the longitudinal peak strain was significantly reduced in the basal-mid segments of the anterior-inferior septal and inferior walls.

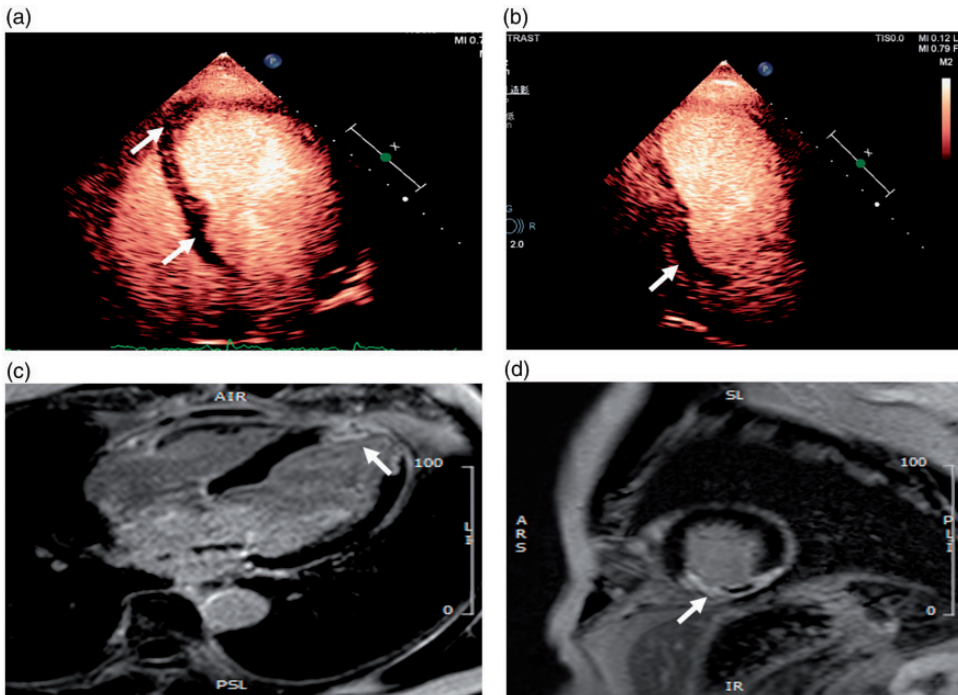


Figure 4. In the (a) apical four-chamber and (b) apical long-axis views of myocardial contrast echocardiography, perfusion defects were revealed in the apical and basal-mid segments of the inferoseptal and basal-mid segments of the inferior wall (arrows). The (c) four-chamber and (d) short-axis views of cardiac magnetic resonance imaging revealed subendocardial enhancement of the apical and mid segments of the inferoseptal and inferior walls (arrows). Recorded 24 hours after successful coronary angiography.

perfusion images, and late gadolinium enhancement showed abnormal transmural hyperintensity with a patch of signal void in the apical inferior wall of the left ventricle. Hence, myocarditis was excluded and myocardial infarction was considered.

After the CAG procedure, other underlying causes were taken into consideration according to the working diagnosis of MINOCA. The CMR findings then confirmed the diagnosis of subendocardial myocardial infarction, confirming the patient's AMI. MINOCA was therefore a plausible final diagnosis. The patient remained asymptomatic during the rest of the hospitalization and developed no recurrent ischemic events or other complications. He was discharged in good condition on the seventh day post-admission. He did not receive antithrombotic therapy because he had no evidence of a thrombus. The patient was discharged home on a daily aspirin, beta blocker, calcium channel blocker, angiotensin-converting enzyme inhibitor, and statin and was scheduled for a follow-up examination with his cardiologist and primary care physician. During follow-up 4 months later, TTE showed the same wall motion abnormalities, but the ejection fraction had improved to 68%. The First Affiliated Hospital of Kunming Medical University does not require ethical approval for the reporting of individual cases. Written informed consent was obtained from the patient for publication of this case report and its accompanying images.

Discussion

Fundamental to the definition of MINOCA is the diagnosis of AMI. According to the Fourth Universal Definition of Myocardial Infarction, the diagnosis of myocardial infarction indicates that an ischemic mechanism is responsible for the myocyte injury (i.e., nonischemic causes such as myocarditis have been excluded).¹ With this revised

concept of AMI, the term "MINOCA" should be reserved for patients who have an ischemic basis for their clinical presentation. In evaluating these patients whose CAG reveals no significant disease, it is important to first exclude nonischemic causes of the apparent AMI (i.e., nonischemic causes of an elevated troponin level) and clinically overt causes of the elevated troponin level (e.g., sepsis or pulmonary embolism). Given the absence of angiographic evidence of coronary artery disease, the present case fits the diagnostic criteria for MINOCA, which is characterized by the diagnosis of AMI and no clinically apparent cause of the presentation. Once these conditions have been excluded using available diagnostic resources, a diagnosis of MINOCA can be made. The next key step is to seek the underlying cause. The prognosis of MINOCA is unclear and likely to be heterogeneous, considering the various mechanisms responsible for the syndrome. Patients should be clinically re-evaluated in the light of the findings, with consideration given to the potential causes that were well illustrated by Pasupathy et al.³ and are listed in Table 1. Other possible mechanisms of AMI in this case include thrombosis due to a plaque disruption or coronary artery spasm. Prolonged coronary spasm can cause temporary occlusion of the coronary artery and resultant myocardial ischemia.⁴ Furthermore, coronary spasm can trigger acute thrombus formation via platelet activation, resulting in the progression of coronary atherosclerosis.^{5,6} Vasospasm may occur with normal coronary arteries or in combination with atherosclerotic lesions.⁷ Vasospasm should also be considered in patients with few risk factors for coronary artery obstruction and nonobstructive arterial occlusion.⁴ The AMI was considered to have been caused by plaque destruction or coronary artery spasm in this patient. It is therefore plausible that the patient had MINOCA without serious

Table 1. Diagnostic considerations regarding the cause of MINOCA.

Cardiac causes	Noncardiac causes
Coronary disorders	Renal impairment
Coronary artery spasm	Pulmonary embolism
Microvascular spasm	
Microvascular dysfunction	
Coronary slow-flow phenomenon	
Plaque disruption/coronary thrombus	
Coronary emboli	
Spontaneous coronary artery dissection	
Myocardial bridging	
Myocardial disorders	
Cardiomyopathy (takotsubo)	
Myocarditis	
Myocardial trauma or injury	
Tachyarrhythmia-induced infarct	
Thrombotic disorders	
Factor V Leiden	
Protein C and S deficiency	

MINOCA, myocardial infarction with nonobstructive coronary arteries.

consequences because of the prompt drug therapy performed.

Previous studies have shown that approximately one-fifth of patients with MINOCA have CMR findings of a delayed subendocardial increase, suggesting myocardial infarction. Despite further examination, the etiology remains unclear in a significant proportion of patients with MINOCA. In patients with an acute coronary syndrome-like presentation but normal coronary arteries, MCE combined with 2D-STI is useful not only to differentiate cardiomyopathy or left ventricular apical thrombosis from an ischemic event but also to identify alternative etiologies. For example, MCE with adenosine can

confirm clinicians' suspicion for takotsubo cardiomyopathy by showing reversible coronary microvascular contraction.⁸ MCE combined with 2D-STI and CMR can allow for evaluation of myocardial contractility and microvascular dysfunction, left ventricular regional systolic function, intracavitary thrombi, and microvascular flow within the infarct territory. In the 2018 American Society of Echocardiography Guidelines Update, MCE is recommended for patients presenting to the emergency department with suspected myocardial ischemia, in which case regional function assessment is performed with ultrasound enhancing agents.⁹ In the present case, we found good consistency between the CMR findings and MCE with 2D-STI findings (Figures 3, 4). At present, the management of MINOCA remains empirical, and regular follow-up of these patients is required to determine the therapeutic efficacy. We should also explore further investigation techniques that involve less risky and inexpensive noninvasive tests, such as CMR and MCE combined with 2D-STI to diagnose and treat MINOCA in a timely manner. Certainly, techniques such as intravascular ultrasound imaging, optical coherence tomography, provocative testing for coronary spasm, and thrombophilia testing can also be selected to further evaluate the underlying cause.

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Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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