

# A Case Report of ST-Segment Elevation Myocardial Infarction With Non-Obstructive Coronary Artery Disease: an Economical and Beneficial Investigative Algorithm

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## Abstract

Myocardial infarction with nonobstructive coronary arteries (MINOCA) is a puzzling clinical entity, coupled with potential multiple pathophysiological mechanisms, often makes disease classification, investigation and management difficult. Despite presence of myocardial damage and increased risk for the future, many patients are discharged undiagnosed, thus the specific cause is not effectively treated. A 59-year-old female was referred to our cardiac emergency department following a ST-elevation myocardial infarction (STEMI). Emergency coronary angiography showed no obstructive coronary artery disease. We made accurate diagnosis and patient-specific treatment for her under limited-resource conditions.

**Keywords:** Algorithm for investigation; Case report; MINOCA; Myocardial infarction.

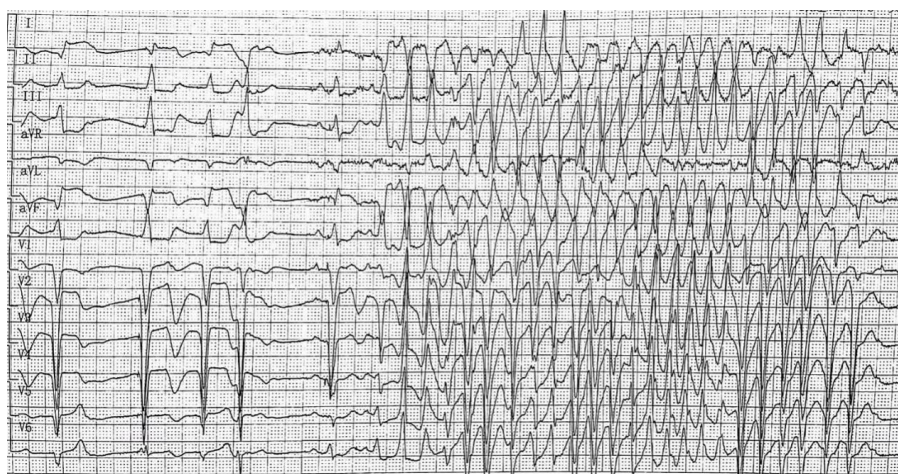
## Introduction

MINOCA is defined as myocardial infarction (MI) with normal or near-normal coronary arteries on angiography (stenosis <50%) [1,2]. The incidence of MINOCA not only varies from 5-15% in all patients with acute myocardial infarction (AMI), but has also been associated with a 5% 1-year mortality [3,4,5]. Previous studies have shown that MINOCA patients are more likely to be women, as evidenced by a prevalence of 10%–15% in relative to 3%–4% in males with AMI.7 MINOCA is less likely to exhibit ST segment elevation on the electrocardiogram (ECG), but mediate a smaller degree of troponin increase than AMI with obstructive coronary artery disease (AMI-CAD) [6,7].

The diagnosis of MINOCA must conform to three aspects, namely (1) Biochemical and clinical indications for AMI; (2) Coronary angiography (CAG) showing normal or < 50 % stenosis of coronary artery; and (3) No other signs of specific diseases causing AMI, such as myocarditis, pyemia and pulmonary embolism [3,4,8]. Previous studies have considered the condition to be a 'working diagnosis', thereby necessitating further identification of its cause [1,9,10]. Early identification and accurate diagnosis of MINOCA is important for optimizing patient prognosis and preventing future cardiovascular events. To date, Cardiac magnetic resonance imaging (CMRI), optical coherence tomography (OCT) and intravascular ultrasound scanning (IVUS) have been used to effectively diagnose the condition. However, many tests are unavailable due to high technical requirements and high costs in developing countries. In many cases, high costs limit availability, thus patients may not only go undiagnosed but also possibly face poor clinical prognosis.

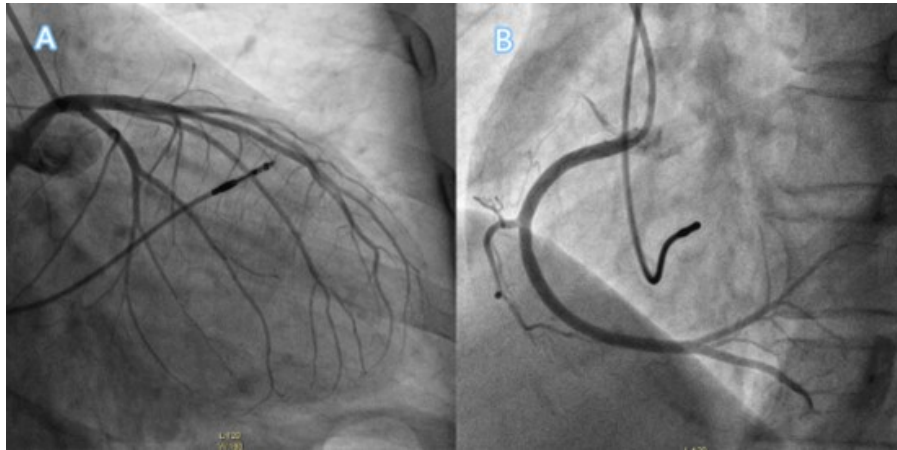
## Case presentation

A 59-year-old xanthoderm woman presented to the emergency room of our hospital because of persistent chest pain and syncope with loss of consciousness and urinary incontinence, which had developed 1 hour prior to her admission. She had a 7-day history of intermittent angina and palpitation, and aggravated chest pain following recovery from syncope. The patient mentioned she had a permanent right ventricular single-chamber pacemaker implantation, due to spontaneous third degree atrioventricular block 6 years ago. She also had a history of ventricular premature beats, albeit with no regular medication. On arrival, her blood pressure (BP) was 150/79 mmHg, with a heart rate of 78 b.p.m., and a temperature of 36.5<sup>0</sup>C. Results from cardiovascular examination were normal on auscultation, with no audible murmur but frequent premature beats. ECG at presentation revealed anterior/lateral wall ST-elevation myocardial infarction, ventricular pacing rhythm, frequent ventricular premature beats (VPB) and non-sustained torsade de pointes (TdP) (Figure 1).



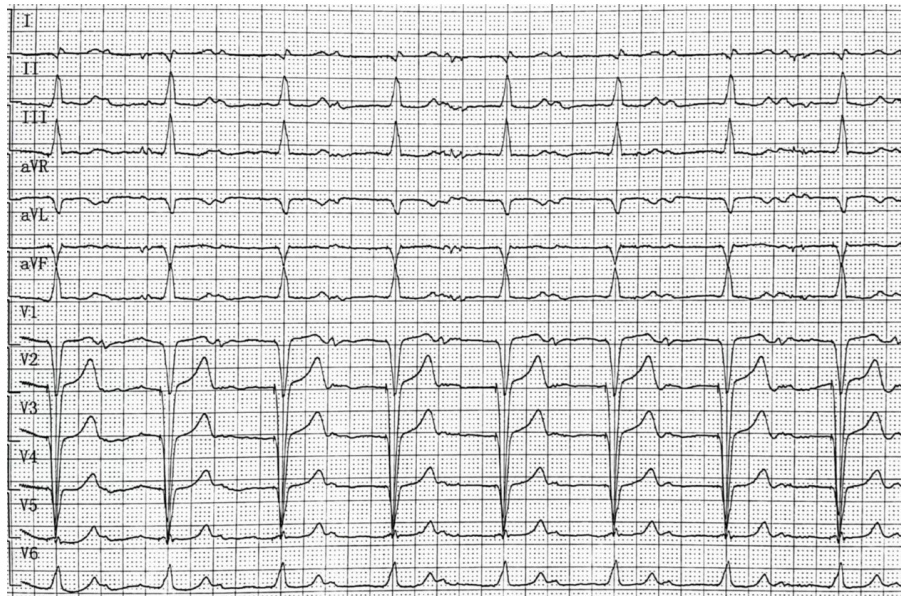
**Figure 1:** ECG results at presentation showing elevation of the ST segment in lead V1-V4, and aVF, frequent VPB with non-sustained TdP, corrected QT (QTc) interval was 522ms.

The patient was given continuous oxygen inhalation and loading doses of aspirin and ticagrelor in the field as a possible acute coronary syndrome. Blood results revealed leukocytosis of  $12.9 \times 10^9/L$  ( $3.5-9.5 \times 10^9/L$ ), potassium 4.1 mmol/L (3.5-5.5 mmol/L), D-dimer 1.35 mg/LEFU (0-0.55 mg/LEFU), N-terminal pro-B-type natriuretic peptide of 870 pg/mL (range 15-125 pg/mL), and creatine kinase-MB of 43.2 ng/mL (range 0-5.85 ng/mL). Her initial serum cardiac troponin I levels were high, at 0.84 ng/mL (0.025-0.1 ng/mL). She was urgently transferred to the cardiac catheterization room, and subjected to emergency coronary artery angiogram (CAG) which revealed unobstructed coronary arteries with thrombolysis in myocardial infarction-3 flow (Figure. 2). The patient refused IVUS examination, thus we ended the operation and transferred her to the intensive-care unit (ICU) for further treatment.



**Figure 2:** Angiogram results showing normal coronary arteries. (A) Left coronary artery. (B) Right coronary artery.

Assuming MINOCA, we maintained the patient on anti-platelet therapy with aspirin (100 mg once a day), ticagrelor (90 mg twice a day) with planned tirofiban infusion for 12 hours (considering the possibility of thrombosis autolysis). These treatments did not improve the patient's condition, as she still experienced chest pain with frequent TdP and hypotensive. Considering that she initially manifested syncope and anti-platelet therapy were ineffective, multiple discussions were carried out regarding whether malignant ventricular arrhythmia was the cause of MINOCA. Long QT syndrome (LQTS) and non-adherence to medication may be the cause of TdP and sympathetic storms, which leads to MINOCA resulting from a supply-demand mismatch. Generally, management of MINOCA caused by non-atherosclerotic would largely focus on reversal or treatment of the etiology. From the discussion, we administered continuous lidocaine and magnesium sulfate infusion, which subsequently improved her hemodynamics 2 hours later. Results of a repeat ECG, performed after her symptoms had been relieved, are shown in Figure 3. Transthoracic echocardiogram, performed the following day, revealed an ejection fraction (EF) of 57%, with hypokinesis of the anterior and basal walls of the left ventricle. Combined with the patient's clinical history and available examination results, we further identified tachyarrhythmias as the cause of MINOCA. In order to prevent LQTS and sympathetic storm, we increased the cardiac pacing frequency to 70 b.p.m, and added a maintenance dose of metoprolol 50 mg/d. She was transferred to the normal medical ward, on the fourth day in the ICU, upon which we observed a significant decrease in troponin I to 0.14 ng/mL. She was discharged on the eleventh day, after hospitalization, and no similar symptoms were evident during a 1-year follow-up period.



**Figure 3:** Results of repeat ECG, performed 6 hours after admission, showing resolution of ST changes and arrhythmia.

## Discussion

MINOCA, which involves multiple pathophysiological mechanisms, can be divided into two types, namely (1) Atherosclerotic causes of myocardial injury, such as plaque erosion with thrombosis, plaque rupture, intraplaque haemorrhage, ulceration; and (2) Non-atherosclerotic causes of myocardial injury, such as epicardial coronary vasospasm or microvascular spasm, coronary embolization, supply-demand mismatch caused by systemic diseases, like tachyarrhythmias, hypotension, respiratory failure, and anaemia [11]. The ESC guidelines suggest that coronary imaging, using IVUS or OCT, should only be applied when plaque erosion or rupture, or spontaneous coronary artery dissection are suspected [1]. Therefore, coronary imaging is not recommended for diagnosing MINOCA caused by non-atherosclerosis, and the algorithm for investigation should be more flexible, especially under limited resources.

For MINOCA with obvious causes, complex means of testing are unlikely to cause any modifications in diagnosis, especially if symptoms are controlled, or even delayed treatment and causes additional economic burden. According to our CAG results, the patient in this study did not manifest any signs of infarction-related artery disease that could explain ECG changes suggest anterior/lateral wall STEMI. Tachyarrhythmia-associated MINOCA is one of the common causes of type-2 MI [4]. Although tachyarrhythmia and MINOCA can be mutually causal, the patient was most likely to have MINOCA caused by tachyarrhythmia (myocardial injury caused by non-atherosclerosis), according to her clinical context. Notably, sympathetic storm and LQTS causes R on T phenomenon and TdP with significant hypotension. Supply-demand mismatch, caused by hemodynamic instability, also causes MINOCA. After antiarrhythmic treatment, the patient's TdP and chest pain gradually subsided, while her clinical indexes returned to normal. In order to shorten QT interval, we increased the frequency of cardiac pacing to 70 bpm, and found no evidence of frequent occurrence of premature ventricular contractions or tachycardia during a 1-year follow-up. Collectively, this data confirmed our judgment, that tachyarrhythmia was not a consequence but a cause of the MINOCA.

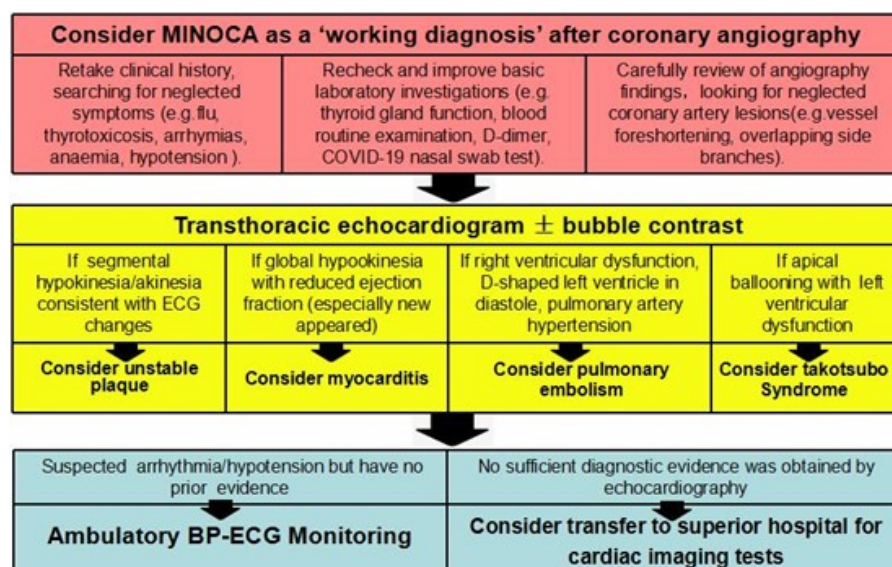
MINOCA patients exhibit extremely variable clinical outcomes, depending on disease etiology. To date, elucidating the underlying mechanism and identification of effective patient-specific treatments options represent a challenge for clinicians, owing to a lack of detailed evidence-based guidelines for the management of MINOCA. Considering that pathogenesis of atherosclerotic-associated MINOCA is similar to that of MI with obstructive coronary artery disease, prescribed cardioprotective therapies in accordance with the AMI guidelines are strongly recommended for MINOCA patients caused of atherosclerotic (type 1 MINOCA)



[1,3,12]. To date, efficacy of routine use of classical secondary prevention and treatment strategies for AMI-CAD to treat type 2 MINOCA remains unclear, with suggestions that it may be contraindicated [4]. Therefore, significant heterogeneity of MINOCA caused by non-atherosclerosis suggests that a more effective treatment strategy may solely depend on an individualized approach so as to improve patient's prognosis. In fact, most patients only get cardioprotective therapies, without consideration of the cause of the MINOCA instead of cause-targeted therapies, owing to the expensive costs associated with tests coupled with limited expertise.

An effective examination and treatment plan should not only aim at the inciting cause, but also be suitable for groups with different income levels. Some effective diagnostic tests, such as IVUS, OCT, and CMRI, are not yet widely used in most developing countries. Therefore, decisions requiring further testing of low-income groups should be based on patient history and economic status under limited-resource conditions. A thorough clinical history, physical examination, cardiac biomarkers, ECG, CAG, and echocardiography are the first-level diagnostic investigations resources for determination of MINOCA etiologies. Carefully collected clinical history often reveals risk factors and possible causes, such as unhealthy living habits and neglected symptoms, which can guide decisions on development and selection of empirical treatment therapies.

Some scholars have proposed a diagnostic approach for groups with inadequate resource conditions, with the aim of saving costs for subsequent treatment [13]. In the present study, we improved the investigative algorithm based on first-level diagnostic resources, and successfully benefited this patient (Figure. 4). Although she belonged to a low-income group from a developing country and could not afford expensive tests, she received rapid diagnosis and timely treatment at a limited cost. Notably, her hospitalization expenses were reduced by more than 40%.



**Figure 4:** Three-step algorithm for investigating MINOCA based on clinical history and echocardiography. (Step 1: The red part represents a review of the patient's medical history, laboratory investigations, and coronary angiography results. Step 2: The yellow part indicates the etiology of MINOCA according to different results of transthoracic echocardiogram. Step 3: The blue part indicates that the etiology can be further explored by ambulatory BP-ECG monitoring and cardiac imaging tests in the absence of clear etiology in the above two steps.)

## Conclusions

Accurate diagnosing MINOCA, a syndrome of diversity and heterogeneity resulting from different causes, is a great challenge to clinicians in countries with limited resources. Although cardiac testing and imaging are very important, they are unavailable in many hospitals across developing countries, indicating that patients cannot receive targeted treatment thus are at a risk of having poor prognosis. To effectively investigate causes of MINOCA, we support the investigative algorithm based on clinical history and

echocardiography under limited-resource conditions. This diagnostic approach is expected to benefit most patients, especially those with type 2 MINOCA, ultimately improving clinical outcomes and lowering medical costs.

## **Informed Consent**

The authors declared no conflicts of interest. Written informed consent was obtained from the patient.

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