

CASE REPORT

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# Delayed ventricular septal rupture complicated with ventricular aneurysm in a case of myocardial infarction with non-obstructive coronary arteries

Si Wang<sup>1†</sup>, Xu Huang<sup>1†</sup>, Qianfeng Xiao<sup>1</sup>, Ying Xu<sup>1\*</sup> and Xin Wei<sup>1\*</sup>

## Abstract

**Background** Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a rare cause of heart attack, which may not receive sufficient attention from patients during post-discharge treatment, especially among those with normal coronary angiography results.

**Case presentation** We present the case of a 65-year-old woman who was readmitted to the hospital with ventricular septal rupture (VSR) complicated by ventricular aneurysm, occurring 2 weeks after myocardial infarction. During the initial admission, coronary angiography revealed normal coronary arteries, leading to a diagnosis of MINOCA. Epicardial coronary vasospasm or coronary embolism was considered as potential causes; however, the patient did not adhere to standardized treatment upon initial discharge. The delayed VSR led to a decline in cardiac function but did not result in severe hemodynamic impairment. Following correction of heart failure with medications, the patient underwent percutaneous VSR repair 19 days after diagnosis and was discharged with a favorable recovery.

**Conclusions** The occurrence of delayed VSR complicated with ventricular aneurysm in patients with MINOCA is rare, highlighting the possibility of serious complications in MINOCA cases. Both cardioprotective therapies and cause-targeted therapies are essential in the management of patients with MINOCA.

**Keywords** Ventricular septal rupture, Ventricular aneurysm, Myocardial infarction, Non-obstructive, MINOCA

## Background

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is defined as an acute myocardial infarction (AMI) according to the “Fourth Universal

Definition of Myocardial Infarction” criteria, with no stenosis  $\geq 50\%$  in any major epicardial coronary artery and no other specific cause leading to myocardial injury [1]. The reported prevalence of MINOCA ranges from 5 to 6% in patients with AMI [1], and patients with MINOCA generally have a more favorable prognosis compared to patients with AMI due to coronary artery disease (CAD) [1, 2]. Consequently, identifying

<sup>†</sup>Si Wang and Xu Huang contributed equally to this work.

\*Correspondence:

Ying Xu

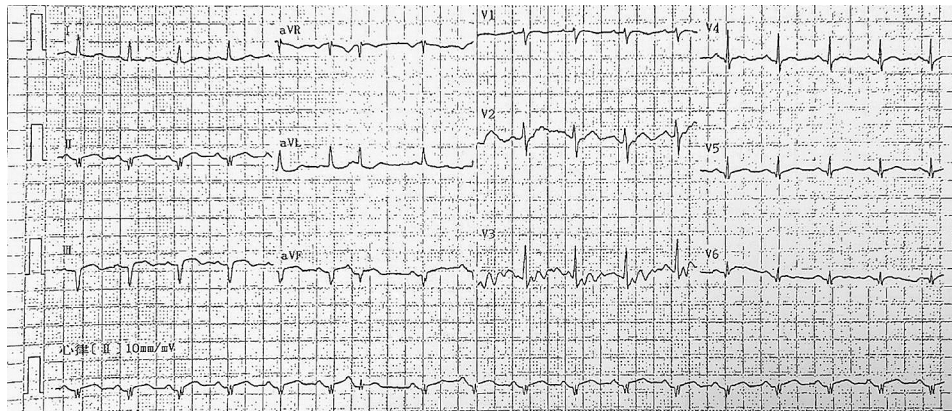
xy\_791030@wchscu.cn

Xin Wei

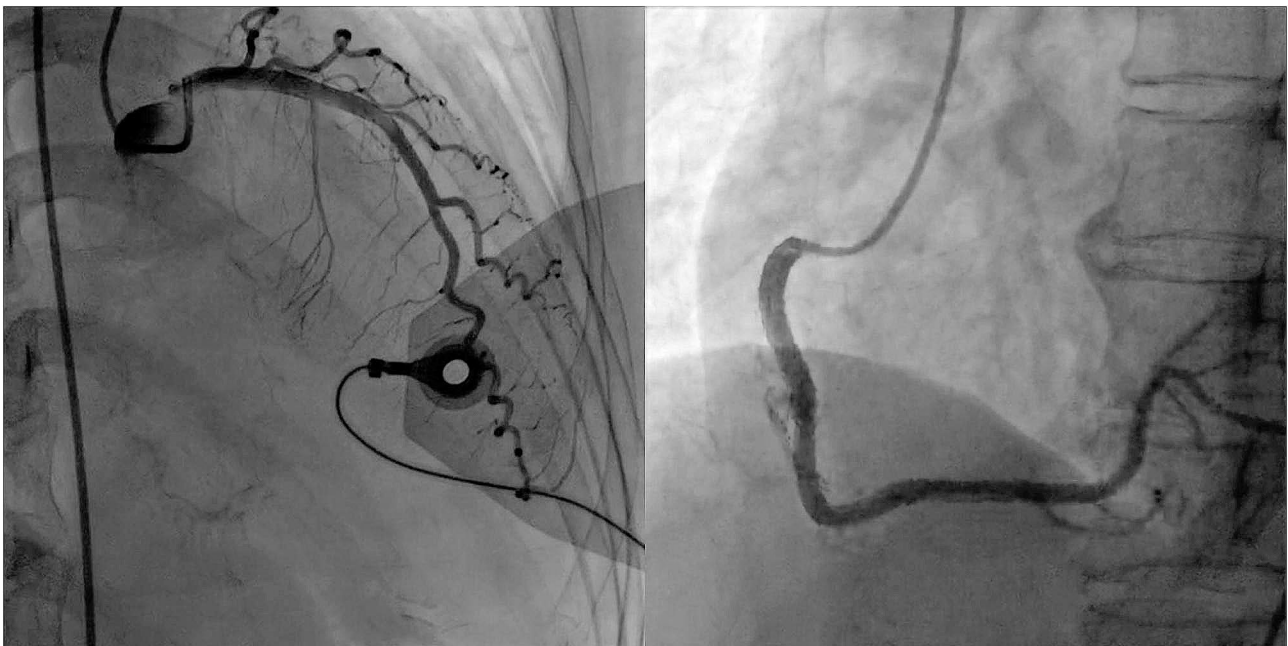
gates-w@wchscu.cn

<sup>1</sup>Department of Cardiology, West China Hospital, Sichuan University, 37 Guoxue Road, Chengdu 610041, China





**Fig. 1** The electrocardiogram upon the patient's first admission revealed sinus rhythm, ST-segment elevation and Q-wave formation in leads II, III, and AVF



**Fig. 2** Coronary angiography revealed normal coronary arteries

non-obstructive coronary arteries through coronary angiography in patients with AMI might lead to inappropriate discontinuation of medical therapy [3], particularly in those patients with normal coronary arteries. We present a case of MINOCA in a patient who did not adhere to standardized medications after discharge and was readmitted to the hospital 2 weeks later due to ventricular septal rupture (VSR) complicated with ventricular aneurysm.

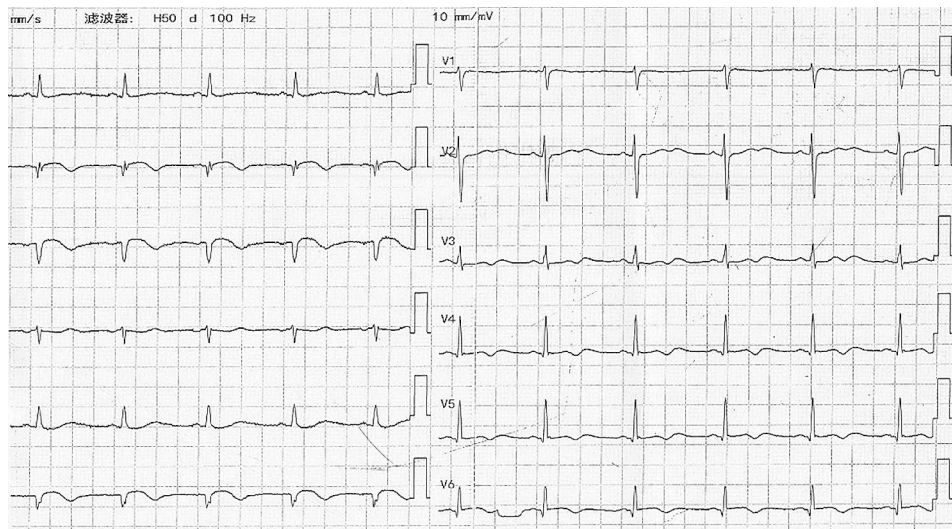
### Case presentation

A 65-year-old woman was admitted to our hospital due to recurrent chest pain for 30 days and dyspnea for 7 days. 30 days ago, the patient experienced intermittent chest pain following exposure to cold, with each episode lasting several minutes. She ignored the symptom and

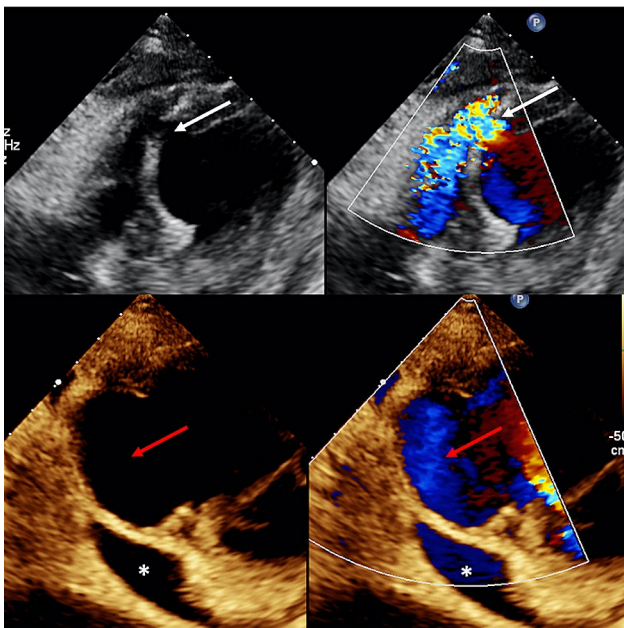
did not seek medical attention. 25 days ago, the patient experienced persistent chest pain without relief and went to a local hospital on September 24, 2021. She had no medical history of hypertension, diabetes mellitus, or hyperlipidemia. The electrocardiogram (ECG) revealed sinus rhythm, ST-segment elevation and Q-wave formation in leads II, III, and AVF (Fig. 1).

The patient's troponin-I level was 9.057 ng/ml (reference: < 0.02 ng/ml) upon admission. However, emergency coronary angiography revealed normal coronary arteries (Fig. 2, Supplementary materials), with no evidence of coronary artery stenosis, slow blood flow, or coronary artery spasm.

The patient was ruled out for hypercoagulable disorders but declined further provocative testing. Echocardiography demonstrated hypokinesia in the posterior septum,



**Fig. 3** The electrocardiogram upon the patient's second admission showed sinus rhythm, Q-wave formation in leads II, III, and AVF, without evident new ST-T changes



**Fig. 4** Echocardiography revealed a 10 mm rupture in the posterior interventricular septum near the apex (white arrow) and a ventricular aneurysm measuring 36 mm × 25 mm (red arrow) in the mid-lower portion of the left ventricular posterior septum, accompanied by moderate pericardial effusion (\*)

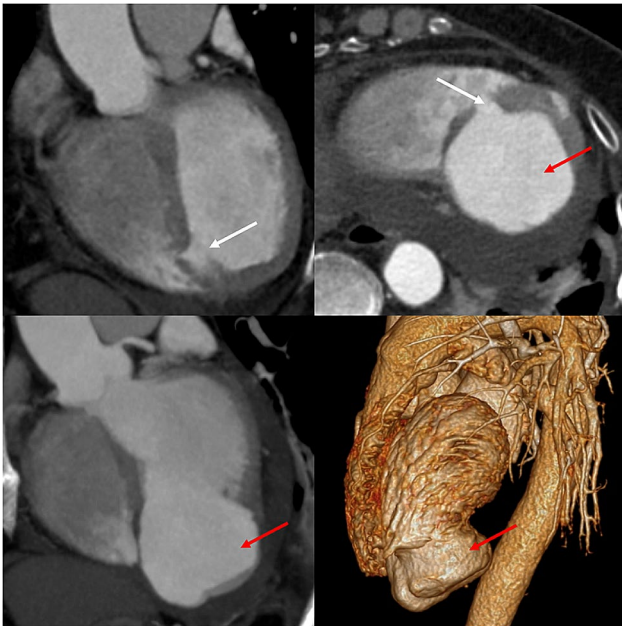
with a normal left ventricular end-diastolic dimension (LVEDD=42 mm) and a normal left ventricular ejection fraction (LVEF=68%). During hospitalization, the patient experienced atrial fibrillation, which reverted to sinus rhythm following intravenous administration of amiodarone. The patient was diagnosed with MINOCA and was discharged with rivaroxaban, metoprolol, and perindopril. However, she did not adhere to the prescribed medications after discharge as she no longer experienced

chest pain. 7 days ago, the patient was readmitted to the local hospital due to dyspnea. The ECG showed Q-wave formation in leads II, III, and AVF, without evident new ST-T changes (Fig. 3), and troponin I levels did not significantly elevate.

Echocardiography indicated a left ventricular aneurysm complicated with ventricular septal rupture (VSR). After receiving optimized heart failure management, the patient was transferred to our hospital for further management. The echocardiography conducted at our hospital revealed a 10 mm rupture in the posterior interventricular septum near the apex, and a ventricular aneurysm measuring 36 mm × 25 mm in the mid-lower portion of the left ventricular posterior septum, accompanied by moderate pericardial effusion (Fig. 4). The location of the ventricular aneurysm and VSR was further confirmed by the cardiac CT scan (Fig. 5). Considering the stable hemodynamics of the patient and the relatively small area of VSR, we performed a transcatheter closure of the VSR 19 days after its detection (Fig. 6, Supplementary materials). The patient recovered quickly, resumed a normal life, and was prescribed standardized secondary preventive medications. One year after the operation, the patient remained in good condition with a normal level of N-terminal pro-B-type natriuretic peptide (NT-proBNP). The echocardiogram showed normal left ventricular size, preserved ejection fraction, and a residual left ventricular aneurysm.

### Discussion and conclusions

Ventricular septal rupture (VSR) is a highly fatal complication following acute myocardial infarction (AMI) with an incidence of about 0.3% [4]. Advanced age, female gender, and delayed reperfusion are recognized as risk



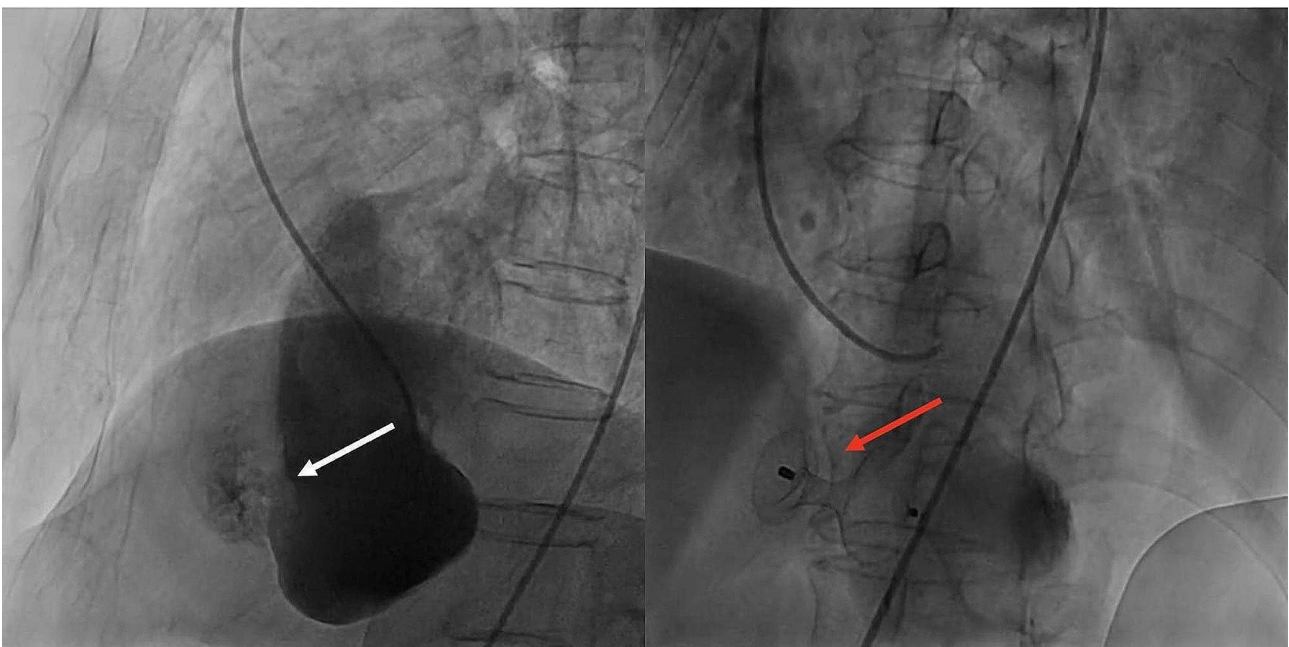
**Fig. 5** Cardiac CT scan indicated the location of the ventricular septal rupture (white arrow) and ventricular aneurysm (red arrow)

factors for VSR post-AMI [4]. VSR usually occurs within 24 h or 3–5 days post-AMI. The former may be attributed to intramural hematoma dissection or hemorrhage into ischemic myocardium, while the latter could result from ischemic tissue coagulation necrosis accompanied by neutrophil infiltration, leading to thinning and weakening of the interventricular septum [5]. The occurrence

of VSR more than 2 weeks post-AMI is relatively rare and may be associated with central perforation of a ventricular aneurysm that develops subsequent to AMI [5]. Reports regarding delayed VSR secondary to ventricular aneurysm formation following MINOCA are scarce in the literature.

VSR is also a rare and serious complication observed during the acute phase of Takotsubo Syndrome [6]. Differential diagnosis between MINOCA and Takotsubo syndrome is crucial. Takotsubo syndrome is a reversible condition where, although some patients may exhibit Q waves during the acute phase, both electrocardiography and echocardiography tend to normalize after several weeks [7]. In this case, the patient developed a left ventricular aneurysm 2 weeks after the onset of chest pain, accompanied by persistent Q waves in the inferior leads on ECG, suggesting myocardial fibrosis resulting from myocardial infarction. Despite the coronary angiography displaying normal results, the progression of the patient's condition does not align with the expected course of Takotsubo syndrome. Considering these factors, the diagnosis of MINOCA should be considered.

MINOCA can result from plaque disruption, coronary artery spasm, coronary thromboembolism, and coronary dissection [8]. It is essential to identify the underlying cause during the evaluation of patients with MINOCA. In this particular case, MINOCA was initially suspected to be associated with coronary artery spasm. The patient experienced recurrent episodes of chest pain preceding an AMI. However, during coronary angiography, the



**Fig. 6** Transcatheter closure of the ventricular septal rupture. The white arrow shows the shunt of ventricular septal rupture and the red arrow shows the ventricular septal occluder

chest pain diminished, and the angiography revealed normal coronary arteries without evidence of coronary artery spasm. Unfortunately, the patient declined provocative testing, preventing a definitive diagnosis. Moreover, the underlying cause of MINOCA in this patient cannot entirely exclude thrombotic occlusion. Although the patient had no history of palpitations, paroxysmal atrial fibrillation occurred during the initial hospitalization, suggesting the possibility of previous asymptomatic atrial fibrillation leading to thrombus formation. However, during subsequent follow-ups, the patient did not experience atrial fibrillation. Considering that the occurrence of atrial fibrillation during the initial hospitalization might be related to AMI and the absence of other risk factors for thrombus formation, the possibility of thrombotic occlusion causing myocardial infarction seems less likely.

In managing VSR post-AMI, delayed repair is often recommended for patients with stable hemodynamics [9]. When encountering hemodynamic instability, the utilization of mechanical circulatory support such as intra-aortic balloon pump (IABP) and extracorporeal membrane oxygenation is advised to prolong the preoperative interval, aiming to enhance the success rate of repair [4]. Currently, there is no precise recommendation regarding the optimal timing for repair. Hobbs et al. suggested performing repairs 7–10 days post-VSR [10]. However, the mortality rate for patients undergoing repair within 1 to 3 weeks after AMI remained at 30% [11]. Histologic studies have also indicated that the proliferation of connective tissue does not occur until the third week post-infarction [12]. Hence, according to the 2019 Chinese Society of Cardiology (CSC) guidelines, repair could be deferred to 3–4 weeks post-AMI through optimized medications and IABP in hemodynamically stable patients [13]. The patient underwent transcatheter repair 19 days after VSR and had a favorable recovery. Delayed VSR accompanied by a ventricular aneurysm is a rare complication post-AMI, especially rarely reported in patients with MINOCA. The conventional treatment for both complications is VSR repair combined with aneurysmectomy surgery [14]. However, surgical aneurysmectomy appears to yield minimal benefits and is only recommended for patients with large aneurysms and uncontrolled heart failure or recurrent ventricular arrhythmias that are not responsive to ablation procedures [15]. Transcatheter closure of VSR alone serves as an alternative treatment for these two complications, as observed in this patient.

MINOCA is a condition often overlooked in clinical practice. While patients with MINOCA generally have a better prognosis compared to those with AMI-CAD [1, 2], this finding is not consistently supported by all reports [1]. Additionally, patients with MINOCA may also be at risk of developing severe complications. After the occurrence of MINOCA, it is crucial to further investigate the

underlying causes and not disregard medication therapy solely due to normal findings in coronary angiography. Both cause-targeted therapies and cardioprotective therapies play a vital role in managing patients diagnosed with MINOCA.

### Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12872-024-04100-w>.

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Supplementary Material 4

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Not applicable.

### Author contributions

Si Wang and Xu Huang collected the data and wrote the main manuscript. Qianfeng Xiao prepared the figures. Ying Xu and Xin Wei revised the initial manuscript. All authors reviewed and approved the final version of the manuscript.

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### Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

### Declarations

#### Ethics approval and consent to participate

The ethics committee approval from the authors' institution and patients' informed consent have been obtained for this study.

#### Consent for publication

Written informed consent was obtained from the patients for publication of this Case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

#### Competing interests

The authors declare no competing interests.

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