Nonobstructive coronary artery myocardial infarction complicated by heart failure, ventricular aneurysm, and incessant ventricular arrhythmia

A case report

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Abstract

Rationale: Myocardial infarction with nonobstructive coronary artery (MINOCA) is one of the common causes of cardiac related death. While patients with MINOCA usually do not need coronary artery intervention treatments, the prognosis could be worsened if the condition is not appropriately managed. This report describes a case of MINOCA patient suffered with multiple complications.

Patient concerns: A 63-year-old female was admitted to the emergency department with acute onset of chest pain, electrocardiogram showed ST segment elevation in precordium leads.

Diagnosis: Emergency coronary artery angiogram no significant coronary artery stenosis being observed. The diagnosis of MINOCA was established. She subsequently developed ventricular thrombus, heart failure, and ventricular arrhythmia.

Interventions: The patient’s ventricular tachycardia (VT) was refractory to different treatments. Due to the presence of a ventricular thrombus, epicardial ablation was performed but the VT recurred. The patient was then successfully treated with an implantable cardioverter defibrillator and surgical removal of the ventricular aneurysm.

Outcomes: The patient was free of events during the 3-month follow-up period after the surgery.

Lessons: MINOCA can occur with multiple complications, and surgical removal of a ventricular aneurysm can successfully recover the cardiac geometry during contraction leading to an improvement of the left ventricular bump function, as well as the removal of the potentially pathological loci of the VT.

Abbreviations: AMI = acute myocardial infarction, ATP = antitachycardia pacing, CAD = coronary artery disease, CAG = coronary artery angiogram, ECG = electrocardiogram, EF = ejection fraction, HF = heart failure, ICD = implantable cardioverter defibrillator, LVED = left ventricular end-diastolic, LVEF = left ventricular ejection fraction, MI = myocardial infarction, MINOCA = myocardial infarction with nonobstructive coronary artery, NT-ProBNP = N-terminal pro-B-type natriuretic peptide, SCD = sudden cardiac death, STEMI = ST-segment-elevated myocardial infarction, TIMI = thrombolysis in myocardial infarction, TTE = transthoracic echocardiography, VT = ventricular tachycardia.

Keywords: incessant ventricular tachycardia, myocardial infarction with nonobstructive coronary artery, ventricular aneurysm, ventricular thrombus

1. Introduction

Myocardial infarction with nonobstructive coronary artery (MINOCA) is the term used to describe cases of acute myocardial infarction (AMI) without evidence supporting the diagnosis of obstructive coronary artery disease (CAD). The vast majority of cases of CAD involve occlusion and require reperfusion therapy, including percutaneous coronary intervention. Whereas cases of MINOCA may not need intervention treatment because of the absence of coronary artery occlusion.[1] However, patients with MINOCA may have a worse prognosis if the condition is not appropriately managed. Here, we report a case of MINOCA that was complicated by heart failure (HF), ventricular aneurysm, ventricular thrombus, and refractory incessant ventricular tachycardia (VT). The patient’s refractory incessant VT was unresponsive to antirrhythmia medications and had to be terminated by shock. However, VT recurred after ablation and was finally managed with an implantable cardiac defibrillator (ICD) and surgical ventricular aneurysm repair.

2. Case report

A 63-year-old female was admitted to the Emergency Department of our hospital because of a persistent pressure, like chest pain, which developed 3 hours prior to her admission. She had a history of hypertension for approximately 10 years but had no history of diabetes. She was a nonsmoker. The electrocardiogram (ECG) results showed ST-segment elevation at the precordial leads (Fig. 1), and ST-segment elevation myocardial infarction
(STEMI) was therefore suspected. The patient was transferred to the catheter lab for an emergency coronary artery angiogram (CAG). The CAG failed to show any significant stenosis in the large coronary artery (Fig. 2). The thrombolysis in myocardial infarction (TIMI) flow was graded at 3. The patient’s chest pain relieved shortly after admission, and she was transferred to the coronary care unit (CCU) for further observation. The patient’s troponin levels were monitored every 6 hours, and the pattern was consistent with that of STEMI. The peak value of troponin was 123 ng/mL. Contrast-enhanced computed tomography was performed and revealed decreased contractility of the anterior wall with a reduced ejection fraction (EF). Consistent with these findings, transthoracic echocardiography (TTE) showed that the left atrial diameter was 31 mm, the left ventricular end-diastolic (LVED) diameter was 48 mm, and the EF was 50%. Based on the findings above, a MINOCA diagnosis was made. The patient was discharged 5 days after admission, following an uneventful observation period, with guidelines for the recommended medical therapies including dual antiplatelet therapy, statin, angiotensin converting enzyme inhibitor, and β-blockers.

Ten days after discharge, the patient developed shortness of breath, which exacerbated with exertion. Physical examination

Figure 1. Electrocardiogram at admission shows ST-segment elevation at the precordial leads (V2-V6), Q-wave, and subsequent T-wave inversion (V2-V5). These observations are consistent with acute anterior wall ST-segment-elevated myocardial infarction.

Figure 2. Angiography shows grade 3 thrombolysis in myocardial infarction flow. Angiography was performed 3 hours after the acute onset of chest pain. (A) The caudal view shows the left coronary artery including the left main artery, left descending artery, and circumflex artery, which are all normal. (B) The normal right coronary artery.
showed that the point of maximal impulse was laterally displaced. TTE revealed that the left atrial diameter was dilated from 31 to 41 mm, the LVED was dilated from 48 to 60 mm, and the EF had dropped from 50% to 38%. An apex ventricular aneurysm with a thickness of 5.7 mm, a range of approximately 30 mm without contractility, and a hypo-echo appendage attached to the left ventricular apex area without movement with cardiac cycle were observed and the N-terminal pro-B-type natriuretic peptide (NT-ProBNP) level was 7420 pg/mL. Cardiac magnetic resonance imaging revealed a left ventricular thrombus and decreased EF. Based on these clinical findings, the patient was diagnosed as having HF, ventricular aneurysm, and mural thrombosis. The clinical symptoms of this patient were relieved by diuretic treatment and the patient was discharged with the addition of Rivaroxaban to the treatment plan.

One week later, the patient came to the emergency room again with palpitations and shortness of breath. ECG revealed VT (Fig. 3). The patient was admitted to the CCU for further treatment. Treatment with a loading dose of amiodarone, followed by a maintenance dose, failed to terminate the VT. Thereafter, amiodarone was replaced by lidocaine and esmolol. However, the VT persisted with deteriorating HF. The incessant VT lasted for 5 days before it was successfully terminated by 100 J synchronized electrical cardioversion. Epicardium radiofrequency ablation was performed to treat the VT, and the pericardium was punctured with the xiphoid process. Through electrophysiology mapping, the epicardium was ablated, and no VT was observed immediately after the ablation.

Fourteen days after the ablation, the VT recurred. About 100 J synchronized cardioversion was used to terminate the VT. Informed consent was obtained from the patient and an ICD was implanted. During the 7-day follow-up period, there were no shock delivered discharges and several episodes of VT were successfully terminated by antitachycardia pacing (ATP). After consultation with a cardiac surgeon, the patient was transferred to the Surgery Department for the removal of the ventricular aneurysm, to restore the heart geometry. One month after the surgery, TTE revealed that the left atrial diameter was reduced to 31 mm, the LVED diameter was reduced to 50 mm, the EF was increased to 50%, and the mitral valve regurgitation was greatly improved. The patient was free of events during the 3-month follow-up period after the surgery.

3. Discussion

Over the past decades, the prevalence of MI has increased. While over 90% of cases of MI are due to the occlusion of the coronary artery, approximately 10% of the cases of myocardial infarction.

![Figure 3. Electrocardiogram (ECG) findings support the diagnosis of ventricular tachycardia. The ECG, taken during a palpitation episode, shows the QRS deformity, which is not associated with P-wave, the main QRS complex upward at the avR lead, and a ventricular heart rate of 150 beats/min.](image-url)
(MI) are MINOCA.[1] MINOCA usually occurs in younger patients and females, who may have a lower risk of cardiovascular events than patients with obstructive CADs.[3] A diagnosis of MINOCA requires the following criteria: the presence of AMI; no stenosis of over 50% revealed by coronary angiography; and no overt clinical cause for AMI. In our case, the angiogram revealed a normal coronary artery. Thus, MINOCA was affirmatively diagnosed. The previous literature indicates that MINOCA may have a favorable prognosis compared with MI with occluded arteries.[3] However, the 2017 European Society of Cardiology guideline on the management of STEMI emphasized that this might not be the case.[6] Although patients with MINOCA were more likely to survive 30 days after MI, a 2.6-year follow-up study indicated no difference the mortality rates in the obstructive coronary artery group and the MINOCA group.[5] Moreover, the Acute Catheterization and Urgent Intervention Triage Strategy Trial showed that the adjusted risk ratio for the 1 year mortality in the MINOCA group was even higher than that in the obstructive artery group.[6]

In the current case, the patient’s HF was managed successful with optimal medical therapy. However, her VT was refractory. Due to the consideration of the ventricular thrombus, epicardial mapping was performed. Currently, there is no conclusive data regarding the efficacy of catheter ablation after AMI. However, multicenter studies have shown that ablation reduces the recurrence of VT in patients with prior MI by ≥75% in 67% of patients.[3] Thus, this treatment is a reasonable option for the reduction of the episodes of recurrent VT in patients with prior MI. Epicardial ablation is the most commonly employed treatment after failed endocardial ablation.[7] In our case, despite the instant success of ablation in the treatment of VT, there was still a recurrence of VT 2 weeks after the ablation therapy.

Several large randomized trials have evaluated the use of ICD in patients at risk of sudden cardiac death (SCD) due to HF or left ventricular dysfunction after MI. These studies showed that ICD implantation reduces arrhythmia-linked mortality. For secondary prevention of SCD, ICD therapy is an option in patients that develop sustained VT/ventricular fibrillation more than 48 hours after STEMI, provided that the arrhythmia was not due to transient or reversible ischemia, reinfarction, or metabolic abnormalities.[9,10] The indications for ICD therapy ≥40 days after STEMI are based on the LVEF and the New York Heart Association class, as derived from the results of the landmark Multicenter Automatic Defibrillator Implantation Trial 2 (MADIT 2) and Sudden Cardiac Death in Heart Failure trials.[11–14] If the LVEF remains ≤0.35 and the patient has a New York Heart Association class II or III HF symptoms, or if the LVEF is ≤0.30 independent of symptoms, then ICD implantation is recommended. Additionally, ATP is an important component of the ICD function. Previous reports have shown that ATP significantly reduces the frequency of shock and improved patient life quality.[15,16]

Ventricular aneurysm is a serious complication after AMI. The results may cause geometrical changes that further compromise the already impaired contractility and filling capacity.[17,18] The scar tissue in the loci of an aneurysm usually accounts for the electric disturbance that leads to the malignant VT. Indications for the surgical removal of the ventricular aneurysm include angina pectoris, ventricular arrhythmias, dyspnea, and the presence of systemic embolism.[19]

In summary, in this study, we describe a case of a patient with MINOCA that was complicated by HF, ventricular aneurysm and refractory VT. The patient was successfully managed with ICD implantation and surgical ventricular aneurysm repair.

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