Ventricular Septal Defect Complicating Myocardial Infarction: A Case of Delayed Percutaneous Transcatheter Closure

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Abstract

A 57-year-old man suffered chest pain during the COVID-19 pandemic, but he delayed medical treatment due to fear of infection. Four months later, symptoms chest tightness and shortness of breath appeared. Electrocardiogram (ECG) revealed old myocardial infarction; color sonography and myocardial CT revealed apical myocardial defect. He refused surgery and percutaneous transcatheter closure, and follow-up observation. After 22 months, the symptoms of chest tightness and shortness of breath aggravated. He recovered after percutaneous transcatheter closure, and was discharged. This case shows delayed closure is one of the possible options for the patients without severe organ dysfunction or hemodynamic disturbance.

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Abstract

A 57-year-old man suffered chest pain during the COVID-19 pandemic, but he delayed medical treatment due to fear of infection. Four months later, symptoms chest tightness and shortness of breath appeared. Electrocardiogram (ECG) revealed old myocardial infarction; color sonography and myocardial CT revealed apical myocardial defect. He refused surgery and percutaneous transcatheter closure, and follow-up observation. After 22 months, the symptoms of chest tightness and shortness of breath aggravated. He recovered after percutaneous transcatheter closure, and was discharged. This case shows delayed closure is one of the possible options for the patients without severe organ dysfunction or hemodynamic disturbance.

Key Words : Acute myocardial infarction, Complication, Ventricular assist device, Radial percutaneous coronary intervention, Covid-19, Case report

Ethics approval statement regarding

This case report was approved by the ethics committee of The Third Affiliated Hospital of Zunyi Medical University. We certify that the case report was performed in accordance with the 1964 declaration of HELSINKI and later amendments.

Informed consent statement regarding

Written informed consent was obtained from all the participants prior to the enrollment of this case report.

Introduction

Post-AMI VSD is a rare complication of AMI, with various clinical manifestations, including hemodynamic instability, cardiac dysfunction and cardiogenic shock. Its incidence is about 0.17%-0.34%[1], but the in-hospital mortality can reach 41-80%[2].

At present, the development of interventional therapy has opened up a new idea for the treatment of post-AMI VSD. Percutaneous transcatheter closure therapy which has the advantages of less trauma and no need for general anesthesia, provides a new treatment idea for the patients with post-AMI VSD who cannot undergo early surgical treatment.

We reported a patient with post-AMI VSD who underwent delayed percutaneous transcatheter closure.

Case Report:

A 57-year-old man suffered sudden chest pains, mainly in the middle and lower sternum, accompanied by sweating, while he rested at home 4 months ago. He chose to delay medical care because of the COVID-19 epidemic. Three months later, he developed chest tightness, shortness of breath and other discomfort. The symptoms of depression and shortness of breath aggravated significantly after a little activity, complicating systemic edema, especially in the extremities. Physical examination after admission revealed heart rate of 98 beats/min, blood pressure of 131/103mnHg, no rales in both lungs, intermittent rales in the third and fourth ribs and systolic ejection murmur on the left sternal border. Besides, Troponin was 0.01μg/L; CK-MB was 12.8U/L; BNP was 880.6pg/ml. Electrocardiogram indicated myocardial infarction of anterior interwall and anterior wall (Figure 1 A). Color echocardiography revealed abnormal movement of the anterior and interanterior walls of the left ventricle with septal perforation (Figure2 A). Myocardial CT showed myocardial defects at the apex of the ventricular septum, being consistent with myocardial perforation (Figure 3). Coronary angiography revealed a three-vessel lesion.((Figure4 A)(174)

The patient improved slightly after the treatment of vasodilating, diuresis and heart strengthening. For ventricular septal defect, there was high risk of surgery according to the cardiac surgery consultation. His family members gave up surgical treatment and interventional plugging treatment. The patient was treated with anti heart failure drugs after risk assessment. The patient was followed up in our hospital every 3 months. During the period, his chest tightness, shortness of breath and other symptoms did not worsen. Color echocardiography reveals there was no ventricular septal defect enlargement and cardiac function decline. The patient developed chest tightness, and increased shortness of breath, with significant restrict of activity after 22 months. Physical examination revealed heart rate of 98 beats/min, blood pressure of 131/103mnHg, no rales in both lungs, intermittent rales in the third and fourth ribs and systolic ejection murmur on the left sternal border. Supplementary examination revealed that Troponin and CK-MB were within normal limits. Electrocardiogram indicated old myocardial infarction (Figure1 B). Color echocardiography showed no enlarged perforation (Figure 2 B).

Percutaneous transcatheter closure was chosen after communication with family members (Figure4 B). Post-operative echocardiography showed changes after ventricular septal closure (Figure2 C). After treatment with anti-heart failure and symptomatic support, the patient's symptoms of chest tightness and shortness of breath were relieved. And he was discharged from the hospital upon recovery.

Discussion

In our report, the patient did not seek medical treatment. Studies have shown that most VSD occurs 3-5 days after AMI[3]. The patients receiving drug therapy alone have a median survival time of 5 days; more than 75% of patients die within 12 days after the onset of VSD symptoms, with 93.6% of 30-day mortality rate, and 96.2% of long-term mortality rate[4]. Therefore, based on the experience, the patient's onset of VSD might be approximately 4 weeks before the first visit. The patient had no discomfort such as chest pain

and no obvious hemodynamic disorder at the first visit. Based on the above-mentioned factors, we dared to make the clinical decision of agreeing to the patient's temporary refusal of closure and choosing follow-up observation. Regular follow-up is a vital guarantee for delayed ventricular septal closure. For patients who need to perform delayed closure, it is crucial to closely monitor whether patients have organ dysfunction and hemodynamic disorder.

Closure of ventricular septal perforation is the key to the treatment of post-AMI VSD. Current guidelines still recommend surgical treatment for post-AMI VSD[5]. According to the STS database report on the results of VSD surgery after infarction, however, only 2,876 patients received surgery in 666 participating institutions[6], and most surgeons operated on no more than 1 patient per year. Moreover, patients were often complicated with complications such as shock and high surgical trauma. The patients undergoing surgical treatment still face the mortality rate of 40%[7]. For the patients with severe hemodynamic disorders, it is particularly important to close ventricular septal defects as early as possible[8]. Such patients, however, often cannot be treated surgically immediately. Transcatheter closure of the ventricular septum may be a more feasible option for these patients. In addition, increasing number of evidences show that percutaneous transcatheter closure of post-AMI VSD is an effective treatment. It can immediately reduce shunt and thus prevent hemodynamic deterioration[9]. Besides, it does not require general anesthesia and has little trauma and is tolerable for vast majority of patients.

At present, the optimal intervention time for post-AMI VSD remains controversial. Guidelines recommend early ventricular septal closure for the patients with severe hemodynamic disorders. However, current studies have shown that using supportive treatments including cardiopulmonary bypass, stable hemodynamics and delay closure can also benefit patients[10] and reduce the mortality of patients[11].In this case, the patient underwent the occlusion 22 months after the onset of VSD, much later than the recommended time in the guidelines. It could provide clinical evidence for delayed closure of patients with post-AMI VSD. This case shows that it is necessary to reevaluate whether early ventricular septal closure is needed for all patients with post-AMI VSD.

The patient reported in this case delayed medical care due to COVID-19, which is a common phenomenon during the epidemic. According to related data, during the epidemic period, the number of emergency visits has been reduced by nearly 50%, and the out-of-hospital mortality has increased accordingly because of patients' fear of coronavirus infection and other factors[12]. Evidence has shown that the incidence of mechanical complications of AMI is significantly higher than that in previous years due to patients' delayed medical treatment during COVID-19[13]. Enough attention should be paid to the consequences of delayed medical care during the COVID-19 pandemic. Besides, the impact of the COVID-19 epidemic on patients' long-term health-seeking habits still needs to be observed in the longer term.

Conclusion

Percutaneous transcatheter closure has become an alternative treatment strategy for patients with post-AMI VSD. Delayed closure is one of the feasible options for patients without severe organ dysfunction or hemodynamic disturbance. The optimal time to perform percutaneous septal closure still needs further study and evaluation.

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Figure legends

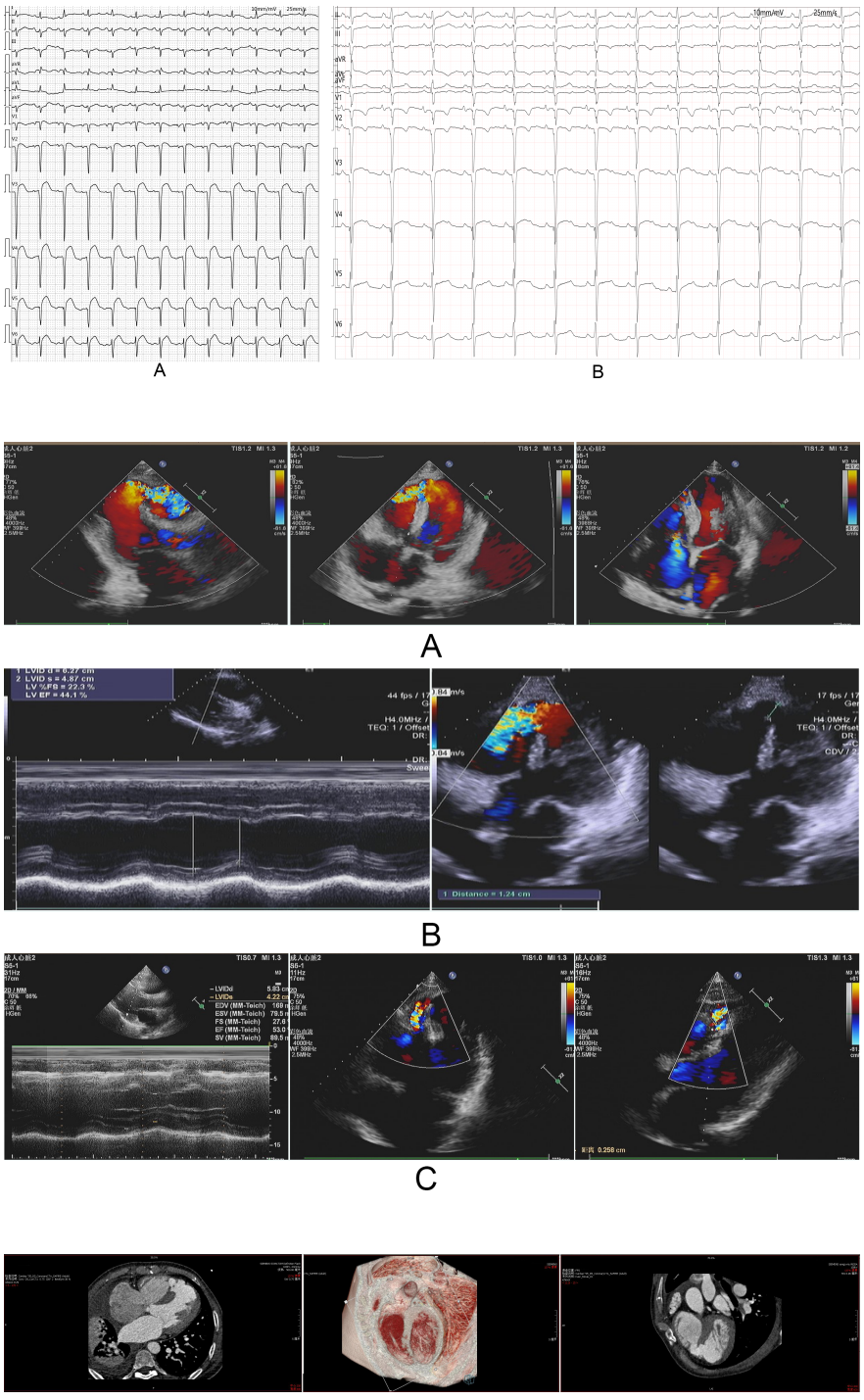
Figure1: A)V1-V5 shows QS pattern, and V1-V6 arch elevates 0.05-0.5Mv;B) V2-V4 shows QS pattern, and V2-V4 arch elevates 0.05-0.15mv.

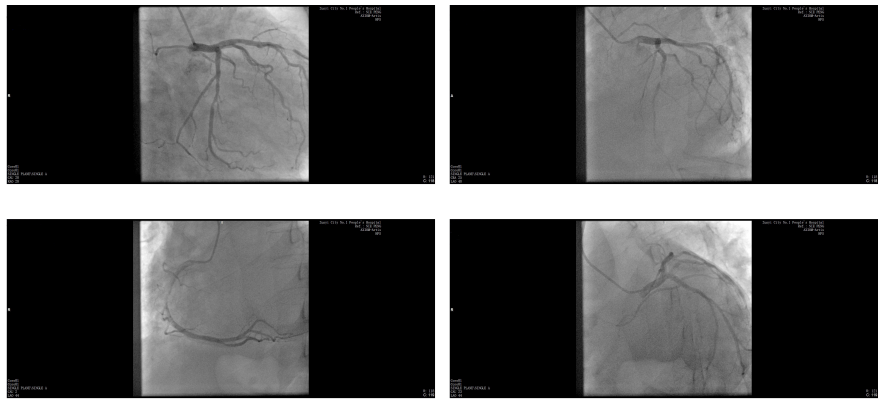
Figure 2 A) Echo-interruption in the left ventricular septum near the apex was observed by color echocardiography, with a range of about 18.5mm and EF: 51%; B) Echo interruption at the apex of the ventricular septum, and the shunt layer at the ventricular septum stump was dissected. The echo interruption in the left ventricular plane and the right ventricular plane was 19mm, and 12mm, respectively; C) The enhanced echo of the occluder can be seen at the ventricular septum near the apex of the heart, and a small gap can be seen below the occluder with a width of about 1.9mm.

Figure 3: Myocardial CT reveals myocardium defect of 2.2x2.1cm in maximum cross-section at the apex of the ventricular septum.

Figure4: A) Coronary angiography (CAG) shows that the LAD is subtotal occlusion in the middle and distal segments, with blood flow TIMI2: 60% stenosis in the middle segment of LCX; TIMI3: diffuse 50-70% stenosis in the proximal segment of RCA; 60% stenosis in the proximal segment of posterior descending branch, with blood flow TIMI3; B) Angiography confirms ventricular septal perforation after apical myocardial infarction, with the defect of about 17mm in diameter. A right heart catheter was placed through the femoral vein under fluoroscopy. The right ventricular and pulmonary artery pressures were measured as RVP53/13mmHg (26mmHg) and PAP52/12 MMHG (24mmHg), respectively. A 260cm guide wire was successfully inserted into the right ventricle through the ventricular septal defect, and then sent into the pulmonary artery. The guide wire snare was inserted through transvenous sheath and pulled out through transvenous sheath pipe. The 28mm(14g) interventricular septum wall packer was sent to left ventricular lateral through14F transmit

sheath pipe after successfully establishing the track. Next, the packer left tray umbrella was released and the sheath tube retreated at the same time. The right tray umbrella was released after completely blocking the ventricular septal defect. The left ventricular angiography confirmed there was no shunt in local part. Moreover, the auscultation noise was obviously reduced, and X-ray images showed that the occluder was in good shape, thus, the occluder was completely released. The postoperative left ventricular pressure was 114/6(41)mmHg.





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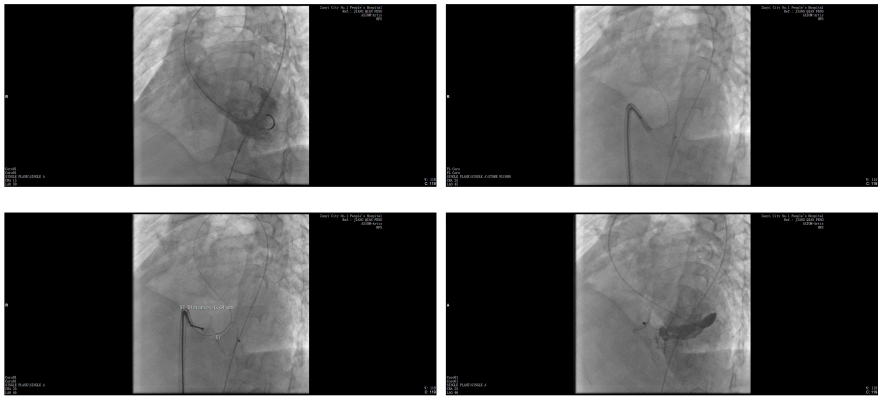

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