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Emergence of Ventricular Septal Rupture During Primary Coronary Intervention for Myocardial Infarction Manifested as Unexpected Coronary Blood Flow Disturbance

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Female, 85
Final Diagnosis: Ventricular septal rupture
Symptoms: Chest discomfort
Medication: —
Clinical Procedure: Coronary angiography
Specialty: Cardiology

Objective: Unusual clinical course
Background: As primary percutaneous coronary intervention (PCI) has been commonly performed for acute myocardial infarction (AMI), we rarely encounter ventricular septal rupture (VSR), which is one of the mechanical complications of AMI. However, the associated mortality rate is still very high unless treated appropriately.
Case Report: We encountered a very rare case of VSR that was considered to have occurred during primary PCI for AMI. The manifestation of sudden coronary flow disturbance may correspond with the emergence of VSR. We introduced a veno-arterial extracorporeal membrane oxygenation (ECMO) system for sudden hemodynamic instability. As a result, the existence of VSR under the operation of the ECMO system led to unusual hemodynamics in the heart, but the vital signs were stabilized by ECMO. VSR was surgically treated and the patient fully recovered without any neurological or physical sequelae.
Conclusions: Although we now encounter markedly fewer mechanical complications of AMI in this era of primary PCI, we should always be conscious of its possibility in the acute phase of myocardial infarction.

MeSH Keywords: Acute Coronary Syndrome • Case Reports • Ventricular Septal Rupture

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Background

In the present era of primary percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI), ventricular septal rupture (VSR) is a very rare mechanical complication of AMI. However, the associated mortality rate is still very high unless treated appropriately. Therefore, early recognition and management of this pathology can improve the prognosis.

Case Report

We present the case of an 85-year-old woman with intermittent chest discomfort at rest persisting overnight for about 16 hours. She never complained of any other chest symptoms preceding this episode. She had been receiving medication for pleural tuberculosis and she had no coronary risk factors. Her blood pressure was 87/65 mmHg, and pulse was 84/min and regular. No pathological cardiac murmur was audible and respiratory sounds were attenuated in the bilateral lower pulmonary fields. Laboratory examinations revealed a positive sign of Troponin T and a high level of brain natriuretic peptide (2526.3 pg/mL). None of the cardiogenic enzymes were increased. An electrocardiogram (ECG) showed a sinus rhythm and an elevated ST-segment in V2-V5 (Figure 1A). A trans-thoracic echocardiogram demonstrated abnormality of the apico-anterior wall motion with about 40% visually estimated ejection fraction. Each cardiac chamber was non-dilated and neither notable valvular dysfunction nor shunt flow was visible. A chest

X-ray showed central vascular congestion and bilateral pleural effusion (Figure 1B). We diagnosed acute coronary syndrome based on clinical findings and examinations such as ECG and echocardiogram. We immediately performed coronary angiography (CAG), which showed an obstruction of the middle segment of the left anterior descending (LAD) artery (Figure 2A). Then, we conducted PCI and successfully passed a guidewire through the occlusion. When we injected contrast medium into the left coronary artery (LCA) to check the position of the guidewire, no blood flow appeared in either the LAD or left circumflex arteries (Figure 2B). Soon after this phenomenon, hemodynamics collapsed. Her systolic blood pressure persisted at about 50–60 mmHg, which caused depressed consciousness. Therefore, we promptly established a veno-arterial extracorporeal membrane oxygenation (ECMO) system for stabilization of the hemodynamics. Under extracorporeal oxygenation support, CAG showed coronary blood flow, which recovered similarly to that seen on the initial angiography in the LCA (Figure 2C). Then, we checked a trans-thoracic echocardiogram, and surprisingly found stasis of blood flow in the aorta (Figure 3A). Moreover, we detected the existence of a large VSR near the apex (Figure 3B). Most of the blood filling the left ventricle moved toward the right atrium through the septal defect caused by being aspirated by the pump of the ECMO system in the right atrium (Figure 3C). Therefore, there was little blood outflowing from the left ventricle to aorta, causing the stasis of blood flow in the aorta (Figure 3A). Her condition and vital signs were stably maintained with ECMO support. On the next day, she was transferred to the Department

Figure 1. (A) An electrocardiogram showing a sinus rhythm and an elevated ST-segment in V2–V5. (B) A chest X-ray showing bilateral pulmonary congestion and pleural effusion.

Figure 2. (A) Initial CAG showing obstruction of the middle segment of the LAD artery (arrows). (B) Sudden coronary blood flow disturbance occurring in both the LAD and left circumflex arteries (arrows). (C) CAG showing restored coronary blood flow in the LCA under extracorporeal oxygenation support.

Figure 3. (A) Trans-thoracic echocardiography showing the stasis of blood flow in the aorta. (B) Most of the blood filling the left ventricle is flowing out toward the right ventricle through the large ventricular septal defect. (C) Tricuspid regurgitation deteriorated by being sucked up by the pump of the ECMO system in the right atrium.

Figure 4. (A) Intra-operative findings showing ventricular septal defect via the right ventricle. (B) VSR closed by the extended double-patch technique.

of Cardiovascular Surgery and the VSR was successfully closed by open surgery (Figure 4). Because the distal segment of the occluded LAD artery was not clearly detected during surgery, coronary artery bypass grafting was not performed. The patient was weaned off the ECMO support simultaneously with the end of surgery. The patient fully recovered without any neurological or physical sequelae. One month later, we confirmed spontaneous recanalization of the occluded LAD artery by coronary computed tomography.

Discussion

We encountered a case of VSR, which is a very rare mechanical complication accompanied by myocardial infarction in the acute phase. In the pre-thrombolytic era, VSR was considered to complicate 1–2% of the population with AMI. On the other hand, contemporary clinical series showed that 0.17% to 0.31% of patients with AMI are complicated by VSR [1]. Because the mortality rate associated with this condition is extremely high, ranging from 41% to 80% [2,3], early recognition and management of this pathology can improve the prognosis. Our case is a very typical case of VSR in that she had higher risk factors such as being an elderly woman, LAD as culprit lesion, and the missing opportunity of revascularization in acute phase.

The typical mechanism of VSR involves coagulation necrosis of infarcted tissue with neutrophilic infiltration, which causes thinning and weakening of the septal myocardium. This sub-acute process takes 3–5 days, which corresponds to the conventional period from AMI onset to the emergence of VSR. In our clinical setting, we tried to perform primary PCI in accordance with the ACCF/AHA guidelines [4], considering the patient had ST-elevation myocardial infarction with clinical and ECG evidence of ongoing ischemia at 12–24 hours after symptom onset. However, the patient's chief complaint seen from the previous day of her admission was thought to have derived from congestive heart failure following AMI. Also, cardiogenic enzyme levels did not increase during hospitalization, which indicated that the acute phase of myocardial infarction had already passed by the time of hospital admission.

There is another critical issue regarding the onset of VSR. Because no finding suggesting VSR had been observed until the beginning of PCI, we consider that the sudden coronary flow disturbance during PCI directly corresponded with the emergence of VSR. Retrospectively, we can see stasis of

contrast medium in the sinus of Valsalva at the moment of coronary flow disturbance (Figure 2B). Our speculation is that the emergence of VSR caused reduced cardiac output, which led to coronary flow disturbance. Following this phenomenon, we had to introduce the ECMO system to correct sudden hemodynamic instability. As a result, the existence of VSR under the operation of the ECMO system produced unusual hemodynamics in the heart.

There has been discussion about the optimal timing of surgical repair for this pathology. The short-term mortality rate was reported to be lower in patients who underwent surgical repair at ≥ 8 days after presentation, in comparison with those who underwent repair within 7 days after presentation [5]. The improved outcome with delayed surgery was associated with the stabilization of the infarcted cardiac tissues. However, the instability of hemodynamics can be a major obstacle for delayed repair of VSR. In other words, critical patients with unstable hemodynamics who cannot wait for delayed surgery inevitably have poor prognosis. Also, there are developing percutaneous options for the closure of VSR, which are promising methods in patients with significant risk for surgery. However, there are several anatomic concerns in selecting patients for this kind of percutaneous repair. For the purpose of maintaining a favorable hemodynamic condition until VSR closure, intra-aortic balloon pumping is usually considered as routine care for mechanical afterload reduction and augmentation of the cardiac output. On the other hand, there have been several reports on the use of ECMO systems to stabilize hemodynamics in this situation until surgery, but the effect of this strategy remains controversial [6–8].

Conclusions

This case is very specific in that VSR occurred during the PCI procedure. The prompt introduction of the ECMO system stabilized the hemodynamics in the presence of this unanticipated mechanical complication. Although we encounter markedly fewer mechanical complications of AMI in this era of primary PCI, we should always be conscious of its possibility in the acute phase of myocardial infarction.

Conflicts of interest

None

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