Severe Mechanical Hemolysis After Transcatheter Closure of a Traumatic Ventricular Septal Defect Using the Amplatzer Atrial Septal Occluder

Liang TANG,¹ MD, Jian-jun TANG,¹ MD, Zhen-fei FANG,¹ MD, Xin-qun HU,¹ MD, Xiang-qian SHEN,¹ MD, and Sheng-hua ZHOU,¹ MD

SUMMARY

Traumatic ventricular septal defect (VSD) resulting from chest trauma, either penetrating or blunt, is a relatively rare occurrence. Herein, we describe the case of a previously healthy 26-year-old man who presented with congestive heart failure, which was secondary to a large traumatic VSD following violent blunt chest trauma. The traumatic VSD was initially closed percutaneously using an Amplatzer atrial septal defect occluder. Post-device closure, however, the patient developed severe intravascular hemolysis refractory to medical treatment. The patient subsequently underwent surgical removal of the Amplatzer device, with concomitant VSD patch closure. (Int Heart J 2016; 57: 519-521)

Key words: Blunt chest injury, Traumatic VSD, Complication

Traumatic ventricular septal defect (VSD) is a relatively uncommon but potentially life-threatening complication of both penetrating and blunt chest trauma. The optimal therapy of this rare entity has not been well established and there are few publications reporting the safety and efficacy of transcatheter closure of traumatic VSD. Herein, we describe the case of a young, otherwise healthy male presenting with a large muscular VSD secondary to violent blunt chest trauma that was initially managed with transcatheter closure using an Amplatzer atrial septal defect occluder (ASO). Post-device closure, however, the patient developed severe intravascular hemolysis due to residual shunt. The patient subsequently underwent surgical removal of the Amplatzer device, with concomitant VSD patch closure without complication.

CASE REPORT

A previously healthy 26-year-old male was admitted with progressive exertional dyspnea for one week and recent-onset orthopnea with features of congestive heart failure. Ten days prior to admission, the patient was trapped in a pyramid selling scheme and he had experienced violent blunt chest trauma when he attempted to escape from the pyramid selling scheme. At presentation, his blood pressure was 85/50 mmHg, heart rate was 110 bpm, and respiratory rate was 30 breaths/min with an oxygen saturation of 93% on room air. Physical examination revealed jugular vein distension, a positive hepatojugular reflex, and severe lower extremity edema. Cardiac exami-

nation was notable for an accentuated pulmonary component of S2, and a grade 4/6 harsh holosystolic murmur at the left lower sternal border with a thrill. Serum cardiac biomarker levels were elevated, with a creatine kinase of 9,493 U/L (normal: 24-195), a CK-MB level of 166 U/L (normal: 0-24), and a hypersensitive troponin-T of 162.4 pg/mL (normal: 0-100). A 12-lead electrocardiogram showed sinus tachycardia with ST elevation in the inferior leads. Transthoracic echocardiography (TTE) demonstrated a large muscular VSD measuring approximately 15.2 mm with an aneurysm (measuring 31 × 25 mm) located in the apical interventricular septum as well as a severely dilated right heart chamber with inferior wall hypokinesis (Supplemental Figure 1A1 and A2). Because of severely low cardiac output, he was treated with inotropic drugs and intra-aortic balloon counterpulsation. Despite these medical treatments, multi-organ failure subsequently developed. Cardiothoracic surgeons were consulted for possible surgical repair of the VSD, but in view of the early stage after VSD occurrence and poor clinical condition, they recommended device closure as the preferred option or a bridge to late surgical repair. Cardiac catheterization was subsequently performed, which showed a PAP of 64/36 (46) mmHg, calculated Qp/Qs of 2.4, and PVR of 3.7 wood units. Coronary angiography showed no significant coronary artery disease (Figure A). Left ventriculography further characterized the defect as a large muscular defect with an aneurysm of the apical interventricular septum (Figure B). The procedure was conducted under general anesthesia and continuous TTE monitoring. The VSD was traversed with a JR catheter (Cordis, USA) and an arterio-

From the ¹ Department of Cardiology, The Second Xiangya Hospital of Central South University, Changsha, China.

Address for correspondence: Sheng-hua Zhou, MD, Department of Cardiology, The Second Xiangya Hospital of Central South University, No.139, Middle Ren-min Road, Changsha, Hunan, 410011, China. E-mail: zhoushenghua_guo@163.com

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Figure. **A:** Coronary angiography revealed normal coronary arteries without obstructive lesions. **B:** Left ventriculography revealed an aneurysm of the apical interventricular septum, with a large defect. **C, D:** Repeated LV angiogram showed good position of the device and reduction in shunt. The device was then released.

enous loop was created by snaring a hydrophilic Terumo guidewire from the right jugular vein. A 12 Fr delivery sheath was then advanced over the guidewire from the right jugular vein across the VSD. Following this, an 18 mm Amplatzer muscular VSD occluder (AmVSDO) was deployed in routine fashion under fluoroscopic guidance. However, with only mild tension on the device, it slipped into the right ventricle side and was then retrieved. Due to the unavailability of a larger AmVSDO, we decided to use a 24-mm ASO, which was similarly delivered. Repeated LV angiograms showed a good position of the occluder and a significant reduction in the shunt. Subsequently, right heart catheterization was again performed to determine the severity of the residual shunt. The PAP decreased significantly to 38/18(26) mmHg and calculated Qp/Qs decreased to 1.3:1, which was not above the significant threshold, and then the device was released (Figure C and D).

On the following day, the patient was noted to have red colored urine highly suggestive of hemolysis (Supplemental Figure 2). The blood tests revealed a significant drop in hematocrit and hemoglobin fell from 12.1 to 8.5 g/dL. Urinalysis showed significant hemoglobinuria and a peripheral blood smear showed fragmented erythrocytes. TTE showed both discs of the ASO appeared to be deformed, allowing the high-velocity flow cross through the device (Supplemental Figure 1B and C). Over the next few days the patient developed soy-colored urine (Supplemental Figure 2); jaundice and the hematocrit fell progressively, while the lactate dehydrogenase and total and indirect bilirubin showed progressive elevation. This was initially managed conservatively with multiple blood transfusions, fluid liberalization, and urine alkalinization to minimize renal damage. However, the hemolysis was refractory to medical treatment. In light of these findings, the patient underwent surgical removal of the device, with concomitant VSD closure with a Teflon patch and ventricular aneurysm placcation (Supplemental Figure 1D-F). By the next day, the clinical and laboratory signs of hemolysis were alleviated. The patient's recovery was uneventful and he was discharged on the 10th postoperative day in stable condition. One month later, a repeat TTE showed a tiny residual shunt at the apical interventricular septum.

DISCUSSION

The incidence of VSD complicating severe blunt chest

trauma has been reported to be only 5.5%, a relatively rare occurrence.¹⁾ The mechanisms of traumatic VSD occurring from blunt chest injury include cardiac contusion caused by acute compression of the heart between the sternum and the spine (especially during late diastole or isovolumetric systole when the ventricles have maximum volume and the valves are closed), or acute laceration of the septum, and rapid deceleration injury that causes myocardial infarction due to the coronary artery intimal tear with thrombosis, spasm, and dissection.^{1,2)} The thin apical interventricular septum is most commonly affected, and on-going necrosis results in complex structure, progressive enlargement, and the development of prompt signs and symptoms of left-to-right shunting. In our patient, we believe the VSD occurred immediately as a result of cardiac contusion and coronary damage at the time of the initial chest trauma resulted in an AMI and liquefaction necrosis of the muscular septum.

Transcatheter device closure of congenital VSDs has been described as an effective and acceptable alternative to surgical intervention with encouraging short-term and long-term results. However, owing to the rarity of traumatic VSD, there is little data regarding indications and results of closure of these defects. Closure of traumatic VSD has been based on a combination of heart failure symptoms, hemodynamics, and defect size. Small defects may be well tolerated and may spontaneously close over time but symptomatic and moderate-to-large shunts with Qp/Qs greater than 1.5:1 should be considered for closure.³⁾ Surgical closure of such defects in the acute setting is associated with high mortality especially in patients with poor clinical status and multi-organ failure, as in our case. Additionally, many surgeons recommend surgical closure after a 4-6 week delay to allow more room for recovery and fibrosis of the surrounding tissue to occur, enabling more secure suture placement.¹⁾ In such patients, transcatheter device closure has been described as an effective and acceptable alternative. As a less invasive approach, catheter-based device closure might allow for immediate VSD closure or initial hemodynamic stabilization. Immediate reduction of the left-to-right shunt, even if the VSD is not completely closed, may stabilize the patient enough to function as a bridge to surgery as in our case.

Accurate measurement of the defect and proper device selection are critical for optimal closure. However, in contrast with congenital defects, traumatic VSD often have a serpiginous course within the muscular septum and have jagged edges and are asymmetric,⁴⁾ making accurate sizing of the defect

difficult, which was the case in our patient. Because of the limited experience, varying characteristics, and difficulties in sizing the defect, no standard method of device selection for transcatheter closure of traumatic VSD exists and the selection of a device is usually individualized. For very large defects, the largest AmVSDO (18 mm) may not be large enough and either the Amplatzer postinfarct VSD occluder (with diameters from 18-24 mm) or the ASO may be the only alternative.⁵⁾ A review of the literature shows that a few cases of traumatic VSD have thus far been managed with an ASO device.^{4,6-9)} In our patient, we decided to use an ASO device because the initial 18 mm AmVSDO easily prolapsed through the defect and due to the unavailability of a larger AmVSDO. Our case showed that transcatheter closure with ASO appears to be an option for patients with a large post-traumatic muscular VSD and significant hemodynamic compromise. In such patients, immediate catheter based closure with ASO can significantly reduce the left-to-right shunt and improve the hemodynamic status. Although the defect is not completely closed, the initial transcatheter closure with ASO may significantly reduce the risk of late surgical repair.

It is worth noting that hemolysis is a potentially intractable complication following transcatheter closure of traumatic VSD with an ASO. The length of the waist of the ASO is 4 mm compared with 7 mm for the AmVSDO and the muscular interventricular septum is much thicker than the interatrial septum and its thickness increases during systole; this reduced length of the waist can prevent the ASO device from maintaining its optimal configuration, leading to device deformation and residual shunt.⁶⁾ High-velocity blood flow going through the wire mesh of the device can potentially produce mechanical fragmentation of erythrocytes and hemolysis. Suh, *et al*⁴⁾ reported a case of a large VSD resulting from penetrating chest trauma that was closed percutaneously using a 28 mm ASO device. The patient developed severe hemolysis attributed to residual shunt, which resolved spontaneously. In their report, they advise against using an ASO device for traumatic VSD closure. Ali, *et al*⁷⁾ recently also utilized a 24 mm ASO device to close a traumatic VSD and they also reported hemolysis as a complication that was resolved by conservative medical management. However, our case demonstrated intractable hemolysis that was managed with surgical device removal. Taken together, we can conclude that in patients with traumatic VSD, the ASO should be used with caution and specialized larger devices, and a VSD occluder may be needed to close such defects and minimize potential complications of hemolysis.

Conclusion: In conclusion, this case highlights the importance

of the “no residual shunt” strategy to minimize the occurrence of severe hemolysis complications following transcatheter closure of traumatic VSD. Furthermore, this case suggests that in the setting of traumatic VSD, the ASO device should be used with caution. Specifically designed devices such as the post-infarction VSD occluder may be better alternatives to provide total closure.

DISCLOSURE

Conflict of interest: None to declare.

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SUPPLEMENTAL FILES

Supplemental Figure 1, 2

Please find supplemental files;

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