Transcatheter Closure of a Traumatic VSD with an ASD Occluder

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Introduction
Traumatic ventricular septal defects (VSD) are exceptionally rare. They can be a consequence of either blunt or penetrating trauma. It is believed that most patients die before reaching the hospital, which makes this condition even more challenging.

Percutaneous closure of traumatic VSD has been presented as an alternative to open conventional surgery.¹ Transcatheter intervention may have some benefits. The objective of this case report is to present a situation where the defect was closed using an Amplatzer septal occluder.

Case Report
A 23-year-old man was admitted in the emergency department after a frontal car collision. He had suffered severe blunt trauma, which included cervical subcutaneous emphysema, bilateral pulmonary contusion, left hemotorax, pneumomediastinum and complex fractures of both femurs. He was in hemorrhagic shock and was immediately taken to the operative room. After external fixation of both femurs and reaching hemodynamic stability, he was transferred to the Intensive Care Unit. The following morning the presence of a loud holosystolic murmur was noted. The 12-lead electrocardiogram showed only sinus tachycardia. The condition remained stable and resolved without the need of transient self-limited hemolysis. Initial blood analysis showed a Qp/Qs ratio of 35 mmHg and a Qp/Qs ratio of 1.9/1.0 and the Qp/Qs ratio was estimated in 1.8/1.0. Cardiac catheterization showed a Qp/Qs ratio of 2.95/1.0. Because the shunt increased significantly, it was decided to close the defect percutaneously.

The procedure was done under general anesthesia and guided by transesophageal echocardiography. Cardiac catheterization was performed using the right femoral artery (6-Fr sheath) and vein (7-Fr sheath) and unfractionated heparin was administered. Angiogram of the LV confirmed a VSD with an oblique entry from the LV into the right ventricular outflow tract. The VSD was crossed using a retrograde arterial approach with a floppy guidewire, which was advanced into the pulmonary artery. The guidewire then snared and brought out the femoral venous sheath. This created an arteriovenous loop to allow the delivery of the closure device. A NuMed sizing balloon catheter was subsequently utilized to measure the defect, but it was not possible to maintain it steady. Therefore, the echocardiographic calculations were used to choose the device size. An 8-mm Amplatzer septal occluder was first selected and loaded into the sheath. This created an arteriovenous loop to allow the delivery of the device. The device was advanced across the VSD, but failed attempt, a slightly different approach was used. The VSD was crossed once more using the guidewire, this time in the opposite direction into the right subclavian artery. Once again, it was snared to make an arteriovenous loop, but on this occasion pulled out through the femoral arterial sheath. For this second attempt, it was decided to employ a 10-mm Amplatzer septal occluder. The device was advanced through the venous sheath and this time was successfully placed (Figure 2). LV angiogram after the procedure revealed a mild residual shunt and the Qp/Qs ratio reduced to 1.53/1.0.

A transesophageal echocardiography was repeated a month after the procedure, which showed the device well adapted to the defect. Nevertheless, a residual shunt remained in the superior border of the device with a peak gradient estimated in 90 mmHg (Figure 2). Another complication of this procedure was the appearance of transient self-limited hemolysis. Initial blood analysis showed a LDH value > 2000 U/L and haptoglobin < 6 mg/dL. The condition remained stable and resolved without the need of blood transfusions.

The patient continued to be asymptomatic and has returned to his previous professional life.

Discussion
There are a few possible mechanisms that explain the development of traumatic VSDs. In this case, cardiac contusion after compression between the sternum and the spine or due to high intrathoracic pressures at impact seems to be the most probable explanation.¹
During his stay our patient was clinically improving, which led to our decision to delay the intervention. Furthermore, it is known that in VSDs fibrotic tissue facilitates the device placement in elective closures. However, the progressively increasing shunt, led to the decision of closing it. He was initially considered for surgery, but given the risks associated with this procedure, the alternative approach was pondered. Transcatheter closure can be a successful substitute with some advantages. It removes cardiopulmonary bypass, avoids arrhythmogenic scar formation related with ventriculotomy and reduces hospital stay and recovery time.

Because these are rare cases with diverse features, it can be challenging to size accurately the defect. In this case, imprecise echocardiographic measurements and the difficulty in operating the sizing balloon catheter, led to the inappropriate choice of the first device.

A possible complication of selecting this type of devices is the appearance of hemolysis. The probable mechanism is the passage of high-velocity turbulent blood flow through
the device, which causes mechanical fragmentation of erythrocytes. Although there are reports of chronic hemolysis, it is usually self-resolving. Like previous cases, we encountered the same complication. Our patient remained asymptomatic and the hemolysis resolved without the need of blood transfusions.

Reference

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Conclusion

Transcatheter devices can be selected as the first choice for closing traumatic VSD. We demonstrate that ASD Occluder can be successfully implanted and that acceptable clinical effectiveness can be achieved.

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