Case Reports

Spontaneous Resolution of Residual Shunting

in 2 Compromised Patients after AMPLATZER Occlusion of Postinfarction Ventricular Septal Defects

Takashi Yanagiuchi, MD
Norio Tada, MD
Taro Suchi, MD
Yukiko Mizutani, MD
Takashi Matsumoto, MD
Mie Sakurai, MD
Tatsushi Ootomo, MD

Ventricular septal defect (VSD) is a rare, potentially fatal complication of acute myocardial infarction. When surgical closure is contraindicated, transcatheter closure may be an alternative. Residual shunting after transcatheter closure of postinfarction VSDs has been reported; however, we found few cases of this in patients who also had severe heart failure or hemolysis.

We report 2 closures of postinfarction VSDs with use of the AMPLATZER Septal Occluder. Both elderly patients—one with severe heart failure, one with persistent hemolysis, and neither a surgical candidate—had high-velocity residual shunting through the occluders. We intensively managed the patients' conditions and used angiography and transthoracic echocardiography to record the gradual disappearance of each shunt over 4 months—the first such serial monitoring of which we are aware. We think that even substantial shunting in the presence of severe heart failure or hemolysis can eventually resolve spontaneously, assuming effective management of the concomitant medical conditions. ***(Tex Heart Inst J 2019;46(1):44-7)***

Key words: *Aged; heart septal defects, ventricular/complications/diagnostic imaging/therapy; myocardial infarction/therapy; percutaneous coronary intervention/instrumentation; septal occluder device; time factors; treatment outcome; ventricular septal rupture/therapy*

From: *Department of Cardiology, Sendai Kousei Hospital, Sendai 980-0873, Japan*

Address for reprints: *Takashi Yanagiuchi, MD, Department of Cardiology, Sendai Kousei Hospital, Hirosemachi, Aoba-ku, Sendai 980-0873, Japan*

E-mail: *takashi.yanagiuchi@gmail.com*

Postinfarction ventricular septal defect (VSD), a rare and severe complication of acute myocardial infarction (MI),[1] is typically treated surgically.[2] When surgery is contraindicated, transcatheter closure may be an alternative.

Residual shunting is not unusual after transcatheter closure of postinfarction VSDs[3]; however, we found few reports of substantial residual shunting in patients who also had severe heart failure or hemolysis.[4] In 2 such patients (one case, a follow-up study of an earlier report[5]; the other, more recent), substantial residual shunting spontaneously resolved over time. Using coronary angiography and transthoracic echocardiography (TTE), we serially recorded the disappearance of shunt flow, and we report our observations here.

Case Reports

Patient 1

A 77-year-old woman, diagnosed with ventricular septal rupture after a subacute anteroseptal MI, was not a candidate for surgical intervention. She underwent transcatheter closure of postinfarction VSD 28 days after admission. The defect was 11 mm in diameter, with thin surrounding tissue. We deployed a 20-mm AMPLATZER™ Septal Occluder (ASO) (St. Jude Medical, an Abbott company). A TTE and left ventriculogram showed small residual shunts.[5] We started the patient on intra-aortic balloon pump (IABP) support and prescribed anticoagulant and antiplatelet medications.

The patient's hemodynamic status improved dramatically, so we terminated IABP support on postoperative day (POD) 1. However, TTE on POD 2 revealed increasing left-to-right shunt flow (peak velocity, 2 m/s) across the ventricular septum through the lower half of the ASO (Fig. 1A), and the patient's condition deteriorated because of progressive hypotension and right ventricular failure. Hoping to promote thrombosis inside the ASO, we discontinued the anticoagulant and antiplatelet therapy. After intensive heart-failure management consisting of IABP reinsertion and diuretic therapy, the patient's hemodynamic status resolved. We ended IABP support on POD 28. Subsequently, although the patient was taking oral diuretic agents only (including 15 mg/d of tolvaptan), her condition improved, and positive-pressure ventilation was ended on POD 40. The tolvaptan therapy was stopped on POD 50. Two months after the procedure, we reduced the patient's oxygen supplementation to a minimum. At 4 months, the shunt flow disappeared (Figs. 1B and C).

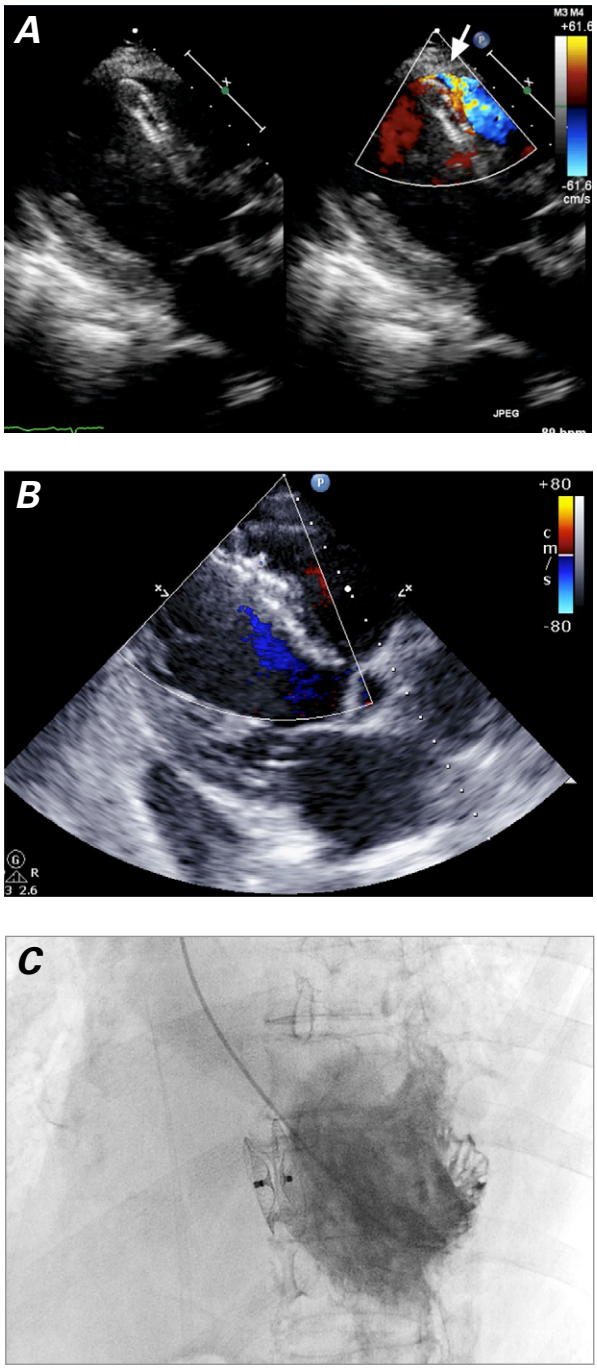

Fig. 1 *Patient 1.* ***A****) Transthoracic echocardiogram (color-flow Doppler mode, apical long-axis view) on postoperative day 2 shows increasing left-to-right shunt flow (peak velocity, 2 m/s) across the ventricular septum through the lower half of the* Amplatzer *occluder (arrow). At 4 months, a* ***B****) transthoracic echocardiogram (apical long-axis view) and* ***C****) left ventriculogram show no shunting.*

Patient 2

A 79-year-old man was admitted to a hospital with a 2-month history of dyspnea and anorexia. An electrocardiogram showed abnormal Q waves and negative T waves in leads V_1 through V_3. Echocardiograms showed an akinetic aneurysm of the left ventricular apex. He was referred to our hospital because of a suspected subacute anteroseptal MI, time of onset unknown. Coronary angiograms revealed an occluded mid-left anterior descending coronary artery with no collateral vessels; percutaneous coronary intervention was not performed because of the unknown delay in diagnosis.

Despite continuous inotropic support, the patient's pulmonary edema could not be controlled; 21 days after admission, we placed him on invasive positive-pressure ventilation and inserted an IABP. An apical pansystolic murmur was audible, and TTE showed an apical muscular VSD (Fig. 2A). We suspected ventricular septal rupture, with VSD closure the only solution for the refractory severe heart failure. Surgery was contraindicated because of the patient's age, severely impaired consciousness, heart failure, and poor kidney function, so we performed transcatheter VSD repair that same day.

Because the patient's hemodynamic status was Forrester class IV, we started percutaneous cardiopulmonary support (PCPS). Real-time transesophageal echocardiography (TEE) showed a defect at the apex with a diameter of 11 mm on both the left and right ventricular sides; the rim was thin but looked stable from all angles. After passing a guidewire from the left ventricle through the VSD into the left subclavian vein, we deployed a 22-mm ASO. Angiography and TEE showed a well-placed device with a small leak at its inferior margin.

The patient's hemodynamic status improved dramatically, and we ended PCPS and IABP support on POD 1. Therapy with aspirin (100 mg/d) and clopidogrel (75 mg/d) was begun. On POD 3, TTE revealed rapid left-to-right flow (peak velocity, 4 m/s) across the ventricular septum through the lower half of the ASO (Fig. 2B). The patient's dark brown urine suggested hemolysis, which was confirmed after blood tests (Table I). We discontinued his antiplatelet therapy. The conservatively managed hemolysis, which developed into hemoglobin nephropathy,[4] necessitated more than 30 units of transfused blood. The patient's serum creatinine level peaked at 4.89 mg/dL on POD 14 and gradually resolved without the need for hemodialysis. Serial TTE over one month revealed gradually diminishing flow across the ventricular septum (Fig. 2C), and the blood indicators of hemolysis returned to normal (Table I). Two months after VSD closure, the biochemistry and hematology test results were normal despite a small residual shunt. At 4 months, color-flow Doppler TTE showed no shunting (Fig. 2C).

Discussion

We detected residual shunting after deploying Amplatzer occluders to close postinfarction VSDs in 2 patients, one who had severe heart failure and one who

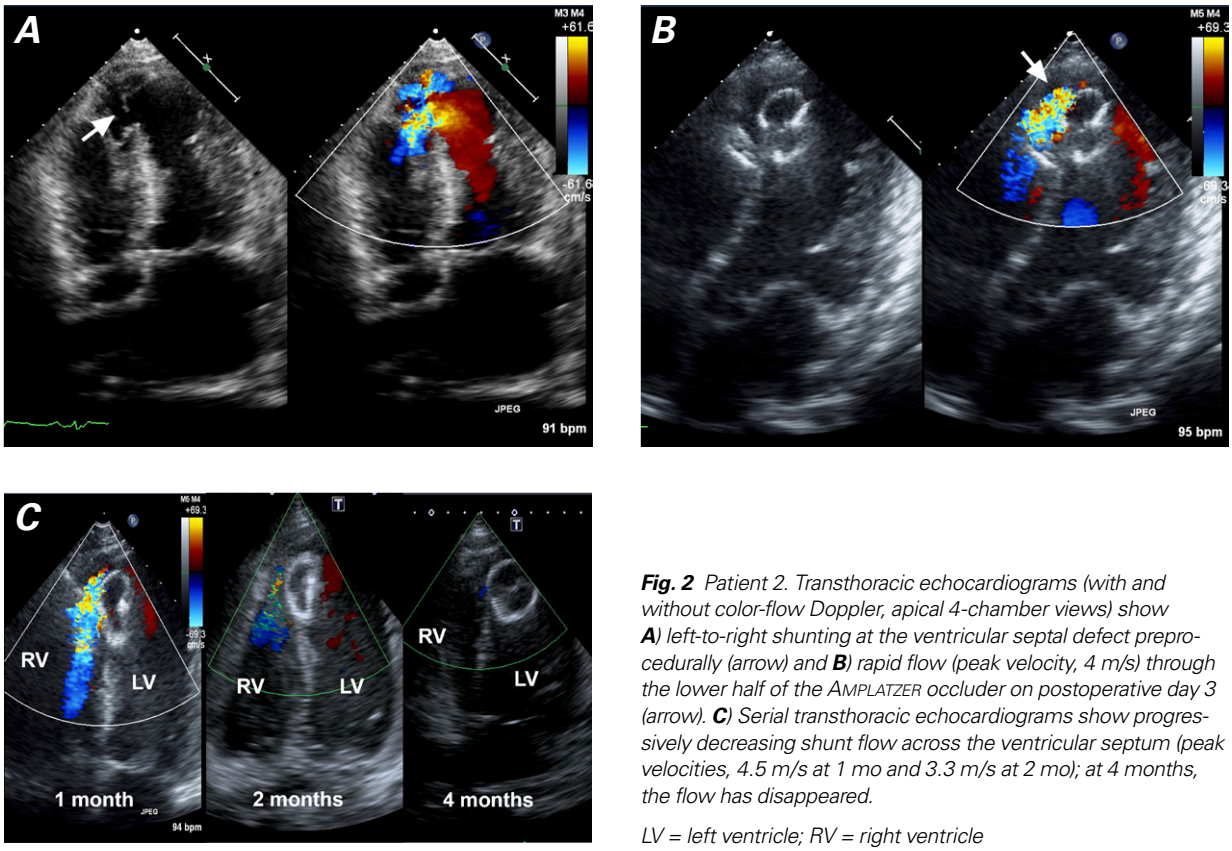

Fig. 2 *Patient 2. Transthoracic echocardiograms (with and without color-flow Doppler, apical 4-chamber views) show* ***A****) left-to-right shunting at the ventricular septal defect preprocedurally (arrow) and* ***B****) rapid flow (peak velocity, 4 m/s) through the lower half of the* AMPLATZER *occluder on postoperative day 3 (arrow).* ***C****) Serial transthoracic echocardiograms show progressively decreasing shunt flow across the ventricular septum (peak velocities, 4.5 m/s at 1 mo and 3.3 m/s at 2 mo); at 4 months, the flow has disappeared.*

LV = left ventricle; RV = right ventricle

TABLE I. Blood Indicators of Hemolysis in Patient 2

Variable	Before Closure	POD				POM	
		3	7	14	28	2	4
Hemoglobin (g/dL)	12.2	8.9	8.9	7.9	8.3	8.4	10.4
Serum creatinine (mg/dL)	1.41	1.71	3.39	4.89	2	0.99	0.97
LDH (U/L)	371	1,620	1,933	1,147	968	324	265
Total bilirubin (mg/dL)	1.4	—	11.31	4.17	2.75	0.85	0.45

LDH = lactate dehydrogenase; POD = postoperative day; POM = postoperative month

had persistent hemolysis. Neither was a surgical candidate. Although repeat transcatheter intervention might have shortened their hospital stays, we could not find appropriate strategies for this because the shunt flow was through the ASOs, not around them. Accordingly, we had to treat the patients conservatively. Intensive management maintained their condition and renal perfusion until the heart failure and hemolysis improved, this when the shunt flow weakened. We serially recorded the gradual decrease in flow by using angiography and TTE.

In preliminary animal studies, investigators reported complete endothelialization of AMPLATZER septal occluders within 3 months after deployment.[6] However, data in clinical studies were insufficient. The healing response to VSD and atrial septal defect occluders in human beings is largely known: the initial formation of thrombotic material between metal wires and around polyester fibers is transformed into connective tissue, and the cellular organization of fibrin deposits is advanced 2 months after deployment.[7] Hoping to activate thrombosis inside the ASOs, we discontinued our patients' anticoagulant and antiplatelet therapy. No thrombotic sequelae or cerebral infarction resulted; otherwise, the effectiveness of our action was unclear. It took approximately 2 months for the severe heart failure and hemolysis to resolve, and approximately 4 months for the shunting to disappear. From the literature and

our cases, we hypothesize that it takes approximately 2 months for residual shunting to decrease dramatically, perhaps consequent to the healing response to the occluders over time. Incomplete endothelialization of Amplatzer occluders has been detected >6 months after device deployment,[8,9] and endothelialization of only some of the metallic mesh might decrease shunting.

Of note, we implanted each ASO in the ventricular septum, because of acceptable prior results.[10] Most transcatheter closure of postinfarction VSD is performed with Amplatzer muscular VSD occluders, which are not approved for use in Japan. The ASO and the muscular VSD occluder are both double-disc devices made from nitinol wire mesh, and their discs and waists contain identical polyester fabric; the only difference is waist length. In our patients, the 4-mm waist length of the ASO—shorter than that of the muscular VSD occluder (7 mm) and the Amplatzer PI Muscular VSD Occluder (10 mm)—seemed ideal, because the septum around each defect had thinned from remodeling after MI. We considered possibly recurrent shunting through the thin scar tissue; regardless, the device selection was appropriate.

Occluders 1.5 to 2 times the size of the defect appear to be best for postinfarction VSD closure.[10] We deployed a 20-mm ASO in Patient 1 and a 22-mm ASO in Patient 2, approximately twice the size of their 11-mm defects.

To our knowledge, this is the first reported use of coronary angiography and TTE to record serial decreases in shunt flow after transcatheter closure of postinfarction VSDs. Although more data are needed for confirmation, we think that even substantial shunts in the presence of severe heart failure or hemolysis will spontaneously close over time, assuming effective management of the concomitant medical conditions.

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