Flail Tricuspid Leaflet During the Percutaneous Closure of Post-Myocardial Infarction Ventricular Septal Defect

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INTRODUCTION

Iatrogenic tricuspid regurgitations caused by the presence of pacemaker or intracardiac defibrillator or repeated right ventricle biopsies particularly in transplanted patients have been reported in the literature[1]; however, there have been no reports of iatrogenic ruptured chordae tendineae of the tricuspid valve following percutaneous closure of post-myocardial infarction ventricular septal defect (post-MI VSD).

The post-MI VSD is a devastating complication of ST-elevation myocardial infarction. The incidence and the clinical course of the disorder have changed dramatically in the last few decades due to improved and fast reperfusion strategies. Despite this decreasing incidence and an improved standard of care, mortality following post-MI VSDs remains high with medical treatment alone. While traditionally post-MI VSDs were only closed surgically, percutaneous closure is a promising treatment strategy that has been introduced with success in recent years.[2,3]

CASE PRESENTATION

A 92-year-old hypertensive female presented to an outside hospital with nausea and chest pain radiating down her left arm beginning 2 days earlier. Electrocardiogram on arrival revealed ST-segment elevations in the anterior lateral leads prompting emergent coronary angiography, which revealed complete occlusion of the mid left anterior descending artery and a tight stenosis of the intermediate coronary artery. Percutaneous transluminal coronary angioplasty, with implantation of two stents, was successfully performed in both the left anterior descending artery and the intermediate coronary artery. On the following day, a systolic murmur was detected and Doppler echocardiography revealed an apical muscular VSD (Figure 1). The patient was in cardiogenic shock with evidence of both renal and liver failure. Given the high risk associated with surgery—i.e., an elderly patient in a state of shock—we opted for the alternative emergency transcatheter approach. After obtaining informed consent from the patient's relatives, we performed the procedure via the right femoral vein and left femoral artery. The implantation technique was similar to that described originally by Landzberg *et al.*[4] The process was monitored with transthoracic Doppler echocardiography and fluoroscopically. The patient received 100 U/kg heparin and a dose of cefazolin (1,000 mg). A counterpulsation balloon was ready for implantation but not implanted. A contrast left ventriculography confirmed an apical muscular VSD (Video 1). The VSD was crossed from the left ventricle using a left internal mammary artery (LIMA) guiding catheter, to create an arteriovenous loop as described in previous publications.[4] After crossing the defect, the hydrophilic wire was replaced by a soft J-tipped 260-cm exchange 0.035-inch guide wire, which was advanced into the pulmonary artery, where it was snared using a 10- to 15-mm gooseneck snare and exteriorized through the right femoral vein, creating a stable arteriovenous loop. An 8-Fr AGA delivery system (AGA Medical, Golden Valley, MN) was advanced over the wire from the femoral vein until the tip of the dilator touched the tip of the retrograde catheter. In general, the retrograde catheter is advanced across the VSD over the exchange wire with its end left in the junction of the inferior vena cava and the right atrium; however, we could not reach this area due to the patient's long aortic tortuosity, therefore, the "kissing catheter" technique was applied in the right ventricle. The muscular VSD device was selected to be 2 mm larger than the post-MI VSD diameter, as estimated by echocardiography and angiography during diastole. Then an Amplatzer 18-mm muscular VSD occluder (AGA Medical Corp., Plymouth, MN) was advanced through it. The left ventricular disk was deployed and pulled gently against the septum, which was both felt and observed by transthoracic echocardiography and contrast ventriculography, and the device was finally deployed with minimal residual shunt (Videos 2-5). However, we detected unusual severe tricuspid regurgitation caused by chordal rupture of the tricuspid leaflet (Videos 6-8), whereas the tricuspid valve was normal in preprocedural echocardiography (Video 9). On the following day, the patient showed hemodynamic deterioration and died from multiorgan failure due to disseminated intravascular coagulation secondary to sepsis, despite the echocardiograms confirming the correct placement of the device.

DISCUSSION

Septal rupture is more common in older, hypertensive females with late presentation of MI. Echocardiogram is diagnostic in over 90% of patients and demonstrates the new post-MI VSD and the left to right shunt across the septum (best demonstrated by color Doppler).[5] Since the precise timing of surgical or percutaneous intervention is critical in determining the outcome,[6] post-MI VSDs have been broadly categorized into two phases: acute phase

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Keywords: Post-myocardial infarction ventricular septal defect, Percutaneous closure, Iatrogenic, Tricuspid chordal rupture

Conflicts of interest: The authors reported no actual or potential conflicts of interest relative to this document.

2468-6441

http://dx.doi.org/10.1016/j.case.2017.04.009

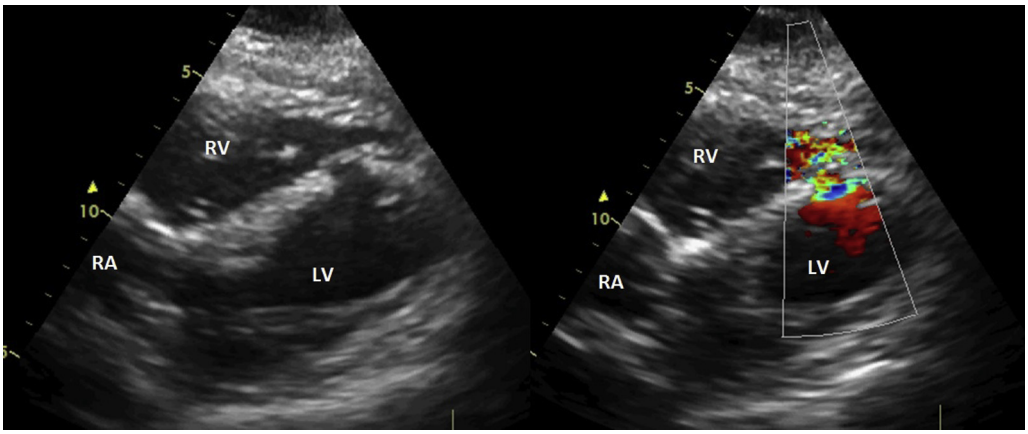

Figure 1 Echocardiography showing post-MI apical muscular ventricular septal defect. *LV*, Left ventricle; *RA*, right atrium; *RV*, right ventricle.

(the first 2 weeks after MI) and chronic phase (3–4 weeks after MI).[7] In the acute period, the defect evolves. Within the first 3–5 days after the infarction, coagulation necrosis develops and lytic enzymes released by neutrophils cause the disintegration of the necrotic myocardium. The site of the septal rupture becomes surrounded by the fragile necrotic tissue.[8] Therefore, both surgical[9] or percutaneous[2,7] closure of post-MI VSD is associated with high mortality especially in the acute phase due to this friable necrotic tissue and the comorbidities of these patients.[6] However, the primary transcatheter closure of post-MI VSDs is a less invasive palliative measure to clinically stabilize patients with suitable anatomy.[8,10] Since the defect evolves into the chronic phase, the septum becomes fibrotic, and the scar develops within several weeks,[8] it has been long believed and recommended that surgery be delayed ideally for 3–6 weeks to allow the margins of the infarcted muscle to develop a firm scar.[8] Probably the same should now apply to transcatheter closure in clinically stable patients. On the other hand, the mortality of post-MI VSD remains high particularly in patients with cardiogenic shock by both percutaneous and surgical closure.[3] Therefore, the patient's preoperative/preprocedural hemodynamic status has been found to be a major determinant of postoperative/postprocedural survival.[6] In post-MI VSDs, failure of transcatheter VSD closure can be classified into three subtypes: failure to implant the device, failure to close the shunt even after proper device implantation, and clinical deterioration irrespective of the shunt closure in a dying patient.[8] We can also add this classification to procedure-related complications. In the current case, the severe tricuspid regurgitation due to ruptured chordae tendineae of the tricuspid valve might have been another contributing factor for mortality in addition to the patient's advanced age, late presentation, and multiorgan failure caused by cardiogenic shock. Combinations of such factors could lead to multiplicative effects. Unfortunately, we could not control the degree of tricuspid regurgitation during the creation of the arteriovenous loop. All care should be taken with arteriovenous loop formation to prevent such a fatal complication. If the loop passes through the valve, regurgitation should be minimal; however, if the loop passes through chordae or leaflets, the regurgitation is greater, and this well-known finding should be taken as a warning sign that when the sheath is advanced, resistance may be felt or chordal rupture may develop. Therefore, care should be taken with the echocardiographic degree of tricuspid regurgitation by either intracardiac echocardiography transesophageal echocardiogram, or transthoracic echocardiogram during the creation of the arteriovenous loop using a "push-me-pull-you" kissing technique of catheters from both venous and arterial sides, to prevent wire injury to the tricuspid valve in addition to the septum. If there is an injury, then one should stop, rewire in a different place, and check again. Once a satisfactory wire position has been achieved, only then should guide catheters and a device be advanced.

CONCLUSION

Although the gold standard is still surgical repair with concurrent bypass surgery, the application of this therapy to all patients might not be reasonable, especially when patients are critically ill or have multiple comorbidities. Percutaneous closure may also be a viable option for patients in the subacute-to-chronic period whose comorbidities preclude surgical repair, and whose septal anatomy is favorable to device placement. However, device embolization, hemolysis, ventricular perforation, or death have been reported as rare major complications of percutaneous VSD closure. The iatrogenic tricuspid injury should also be kept in mind during the percutaneous closure procedures.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.case.2017.04.009.

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