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2022 Symposium Presentation

A Case of Inferior Myocardial Infarction Complicated by Ventricular Septal Rupture Leading to Cardiogenic Shock: Tandem Heart to the Rescue!

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Abstract

Ventricular septal rupture (VSR) is a rare but life-threatening complication of acute myocardial infarction. We present a case of VSR-related refractory cardiogenic shock that was successfully managed with TandemHeart® followed by surgical repair.

Keywords: ventricular septal rupture, myocardial infarction, cardiogenic shock

Background

Ventricular septal rupture (VSR) is a life-threatening, albeit rare, mechanical complication of ST-segment elevation myocardial infarction (STEMI), occurring in 0.3-2% of patients.¹ If left untreated, VSR in this setting can progress to cardiogenic shock, associated with a mortality rate of 94%.² Although the definitive treatment is surgery, this can often be challenging due to friable necrotic myocardial tissue and unstable hemodynamics. Surgical repair is associated with a mortality rate of 42.9%;² therefore, a multi-disciplinary approach should be considered when managing this grave complication.

We describe a case of inferior STEMI complicated by VSR and cardiogenic shock.

Case Description

A 30-year-old Hispanic male with a past medical history notable for hypertension and hyperlipidemia presented to the emergency room with a 1-week history of substernal chest heaviness and shortness of breath. Upon evaluation, the patient was hemodynamically stable with a blood pressure of 111/80 mmHg, heart rate of 105 beats per minute, and oxygen saturation of 97% on 2 liters of oxygen. Physical examination

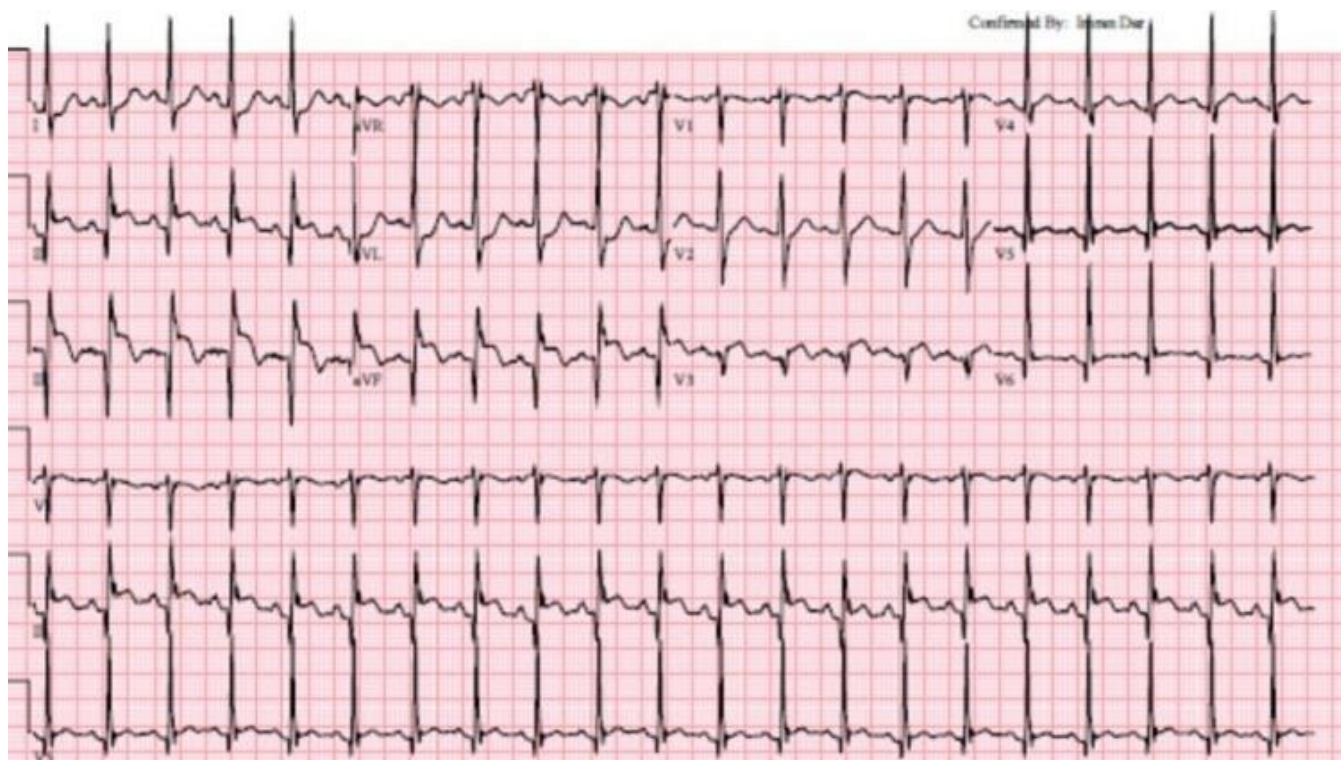


Figure 1. Electrocardiogram. ST-segment elevation in leads II and III, AVF associated with Q waves, reciprocal ST-segment depression in lead I, and AVL consistent with a subacute inferior ST-segment elevation myocardial infarction.

was positive for a new soft pan-systolic murmur at the left lower sternal border in addition to cold lower extremities.

Electrocardiogram revealed ST-segment elevation in leads II and III, AVF associated with Q waves, reciprocal ST-segment depression in lead I, and AVL consistent with a subacute inferior STEMI (Figure 1).

Pertinent laboratory work revealed a high sensitivity troponin I of 5500 ng/L (reference < 14.0 ng/L), serum creatinine of 3.2 mg/dL (0.7-1.3 mg/dL), blood urea and nitrogen of 50 mg/dL (6.0-24.0 mg/dL), aspartate

transaminase of 2500 U/L (8.0-24.0 U/L), alanine transaminase of 3250 U/L (4.0-36.0 U/L), and lactic acid of 2.5 mmol/L (0.5-2.2 mmol/L), suggestive of end-organ hypoperfusion. Selective coronary angiography was performed, which showed a 100% ostial right coronary artery (RCA) thrombotic lesion, 70% stenosis in the mid and distal left anterior descending artery (LAD), diffusely diseased first obtuse marginal branch, and 70% mid left circumflex disease (Figure 2).

As a temporizing measure, balloon angioplasty of the culprit RCA ostial lesion was done, partially restoring blood



Figure 2. A) 100% thrombotic ostial right coronary artery lesion, B) significant mid and distal left anterior ascending artery disease, C) diffusely diseased obtuse marginal 1 and significant mid left circumflex disease.

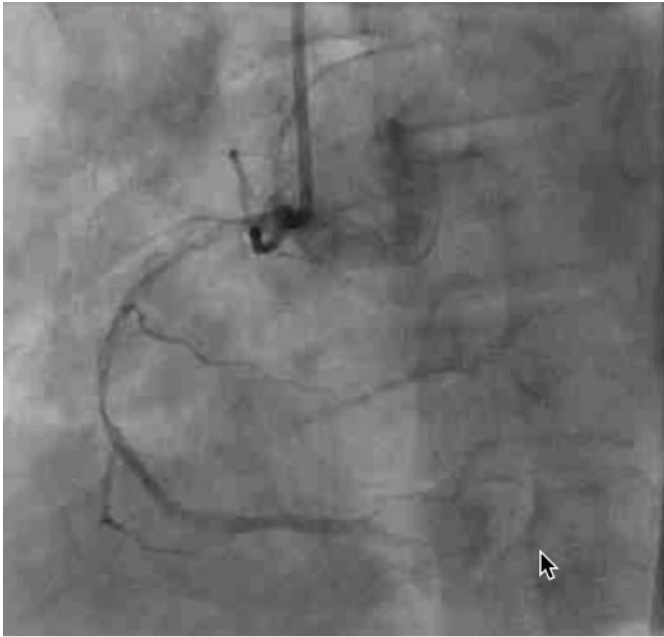


Figure 3. Patient status after balloon angioplasty of ostial right coronary artery lesion, demonstrating TIMI 2 flow.

flow (thrombolysis in myocardial infarction 2 flow) (Figure 3). Aspiration thrombectomy was attempted but unsuccessful due to technical difficulties. Ventriculography revealed left to right contrast shunting suggestive of a ventricular septal defect (VSD). This was confirmed with a bedside echo which illustrated a muscular VSD with evidence of left to right shunting and features of right ventricular dysfunction (Figure 4). Left ventricular function was preserved. Due to concerns for clinical and hemometabolic cardiogenic shock, an intra-aortic balloon pump was placed.

Although the patient's vitals remained stable 24 hours later, end-organ status continued to deteriorate with worsening lactic acidosis and liver and kidney function. The decision was made to proceed with placing a TandemHeart® to stabilize the patient. Within forty-eight hours of escalating mechanical circulatory support (MCS) to TandemHeart, the aforementioned hemometabolic parameters returned to normal.

Twelve days after initial presentation, the patient underwent two-vessel coronary artery bypass grafting with a left internal mammary artery to LAD and saphenous vein graft to RCA, patch repair of the VSD, and a switch of the TandemHeart to veno-arterial extracorporeal membrane oxygenation (V-A ECMO) intra-operatively to allow for right ventricular function recovery. The patient was eventually

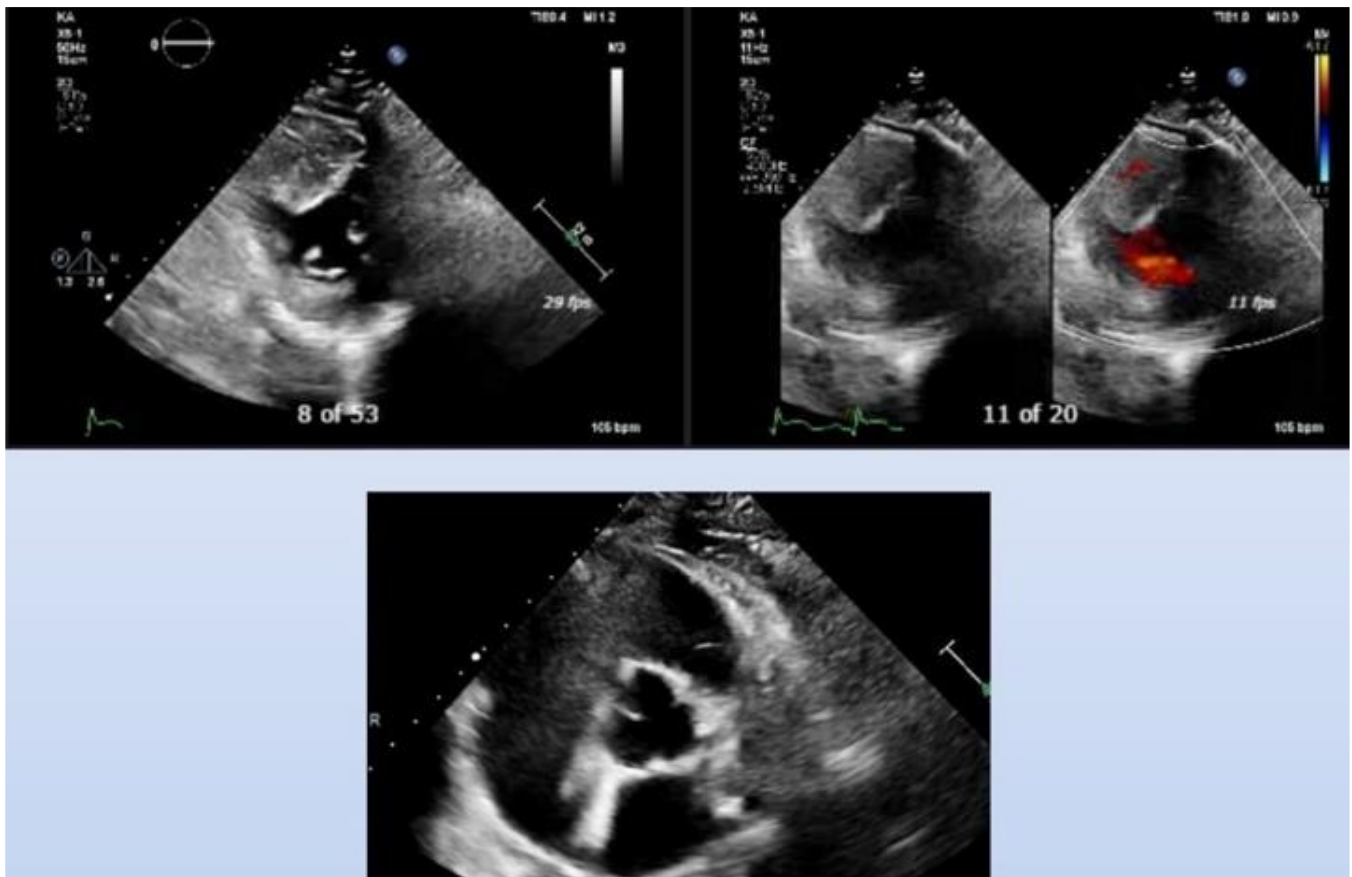


Figure 4. Echocardiographic images demonstrating ventricular septal defect with left to right shunting and an enlarged right ventricle.

discharged home. Trans-esophageal echo revealed no residual shunting across the VSD.

Discussion

Although the incidence of post-STEMI VSR-related cardiogenic shock has decreased in the reperfusion era, it still carries a high mortality rate.² This case highlights the importance of prompt recognition and initial hemodynamic stabilization of patients experiencing this complication using contemporary MCS before surgical repair. We felt that TandemHeart was most suitable in this case as it is a left atrial to femoral artery bypass system that does not involve interacting interventricular septum. The theoretical risks of left ventricular unloading with an Impella device (Abiomed) include interacting with the friable necrotic myocardium around the VSR and inducing hypoxia by right to left shunting at the level of the ventricle. V-A ECMO can be used in STEMI complicated by VSR; however, accurately assessing right ventricular function before patch repair and its ability to recover after patch repair may be hindered, and as such, we feel the use of TandemHeart in acute VSR offers the best strategy to stabilize patients.

Conclusion

The optimal timing of definitive surgical repair of VSR remains controversial.¹ In this case, surgery was delayed to allow time for hemodynamic stabilization and healing of the myocardial tissue to increase the chances of successful surgical repair.

Acknowledgments

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