

## Double Trouble: Ventricular Septal Rupture (VSR) and Ventricular Wall Aneurysm (VWA) Complicating Acute Myocardial Infarction

Shafi I<sup>1</sup>, Oviedo C<sup>2</sup>, Reddy V<sup>2</sup>, Abu-Mahfouz<sup>3</sup>, Omar AE<sup>3</sup>, Baciewicz FA<sup>4</sup> and Afonso L<sup>5\*</sup>

<sup>1</sup>Department of Internal Medicine, Wayne State University, Detroit, MI, USA

<sup>2</sup>Division of Cardiovascular Medicine, Department of Internal Medicine, Wayne State University, Detroit, MI, USA

<sup>3</sup>DMC Heart Hospital, Detroit Medical Center, Detroit, MI, USA

<sup>4</sup>Division of Cardiothoracic Surgery, Department of Surgery, Wayne State University, Detroit, MI, USA

<sup>5</sup>Division of Cardiology, Wayne State University, Detroit, MI, USA

\*Corresponding author: Afonso L, Program Director, Division of Cardiology, Wayne State University, Detroit, MI, USA, Tel: +3137452620; E-mail: lafonso@med.wayne.edu

Received: November 18, 2019; Accepted: December 09, 2019; Published: December 16, 2019

Copyright: © 2019 Shafi I, et al. This is an open-access article distributed under the terms of the creative commons attribution license, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

### Abstract

Ventricular septal rupture (VSR) is a rare but fatal complication of acute myocardial infarction. Prompt diagnosis with noninvasive imaging is key and surgical repair, though challenging remains the treatment of choice. We describe a rare case of inferior wall AMI complicated with VSR and posterior true ventricular wall aneurysm.

**Keywords:** Ventricular septal rupture; Ventricular wall aneurysm; Acute myocardial infarction

**Abbreviations:** VSR: Ventricular Septal Rupture, EKG: Electrocardiogram; NYHA: New York Heart Association; STEMI: ST Segment Elevated Myocardial Infarction; AMI: Acute Myocardial Infarction; GUSTO: Global Use of Strategies to Open Occluded Coronary Arteries; SHOCK: SHould we emergently revascularize Occluded Coronaries for cardiogenic shock; RCA: Right Coronary Artery; RV: Right Ventricle

### Case Presentation

A 49-year-old male patient with recent ST elevated inferior wall myocardial infarction (3 weeks prior to coming to our hospital) presented to our emergency department with complaints of worsening shortness of breath and lower extremity swelling. The patient was discharged from another facility 2 weeks prior to his current presentation after being admitted for an acute ST-segment elevation myocardial infarction (STEMI). Cardiac catheterization done there revealed 100% total occlusion of the RCA, two drug eluting stents (DES) were placed in the RCA. The patient was discharged on aspirin 81 mg daily, ticagrelor 90 mg BID, atorvastatin 80 mg at night and metoprolol succinate 25 mg daily. Three days following discharge, he started to experience worsening shortness of breath on minimal exertion, paroxysmal nocturnal dyspnea, increased fatigue and progressive lower extremity swelling. On presentation to our facility the patient was significantly dyspneic, NYHA class IV, tachycardic with a heart rate of 115, blood pressure of 94/64 and mean arterial pressure (MAP) of 74 mmHg. Physical examination was remarkable for a holosystolic murmur pronounced at the left lower sternal border, elevated JVP and bilateral basilar crackles.

### Medical history

Hypertension, diabetes mellitus, chronic obstructive pulmonary disease, recent inferior wall myocardial infarction, 50 pack year smoking history.

### Differential diagnosis

Acute decompensated heart failure, Ventricular septal rupture post myocardial infarction.

### Investigations

EKG on presentation (Figure 1) revealed sinus tachycardia with right axis deviation and q waves present in the inferior leads with T wave inversions. CXR (Figure 2) showed bilateral vascular congestion accompanied by bilateral effusions. Transthoracic echocardiography revealed a small area of discontinuity in the posterior-inferior septum and the inferior wall was suspicious for a ventricular septal defect. Trans-esophageal echocardiography (TEE) was done which showed a large basal-mid inferior septal saccular aneurysm with thinned out wall (2.2 mm). Aneurysm neck measured 5.0 cm across, and approximately 4.0 cm deep. VSR was noted along apical aspect with systolic color flow turbulence and left-to-right shunting seen at the level of rupture (Figure 3). 3D Full volume and 3D zoom images were acquired for further analysis offline. 3D planimetry measurement of apical infero-septal VSR showed an area of 2.91 cm<sup>2</sup> (Figure 4).

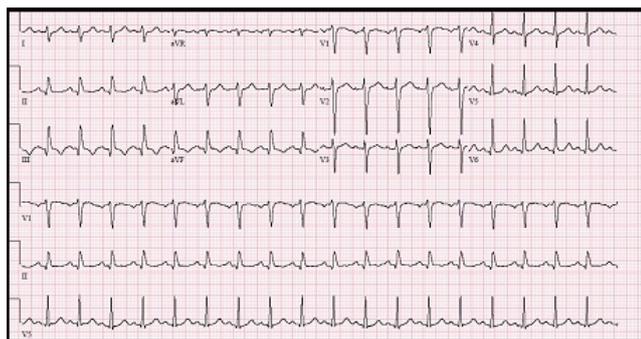
### Management

The patient was transferred to the coronary care unit for clinical stabilization before surgical repair. Cardiothoracic surgery was consulted, and the patient was taken to the operating room. A 3.0 × 1.5 cm defect distal to the mid ventricular septum was repaired with a bovine pericardial patch along with repair of a large posterior ventricular aneurysm. Post operatively the patient required vasopressor support with dobutamine, norepinephrine, epinephrine

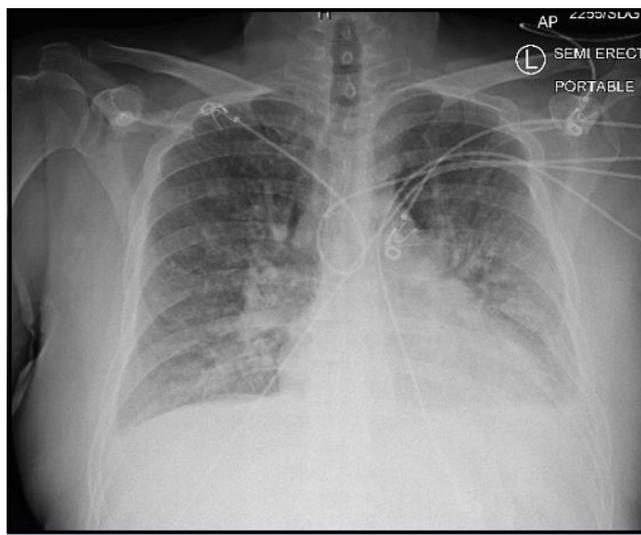
and vasopressin. Vasopressor support was weaned off over the subsequent week and the patient was extubated at day 3 post procedure.

### Follow-up

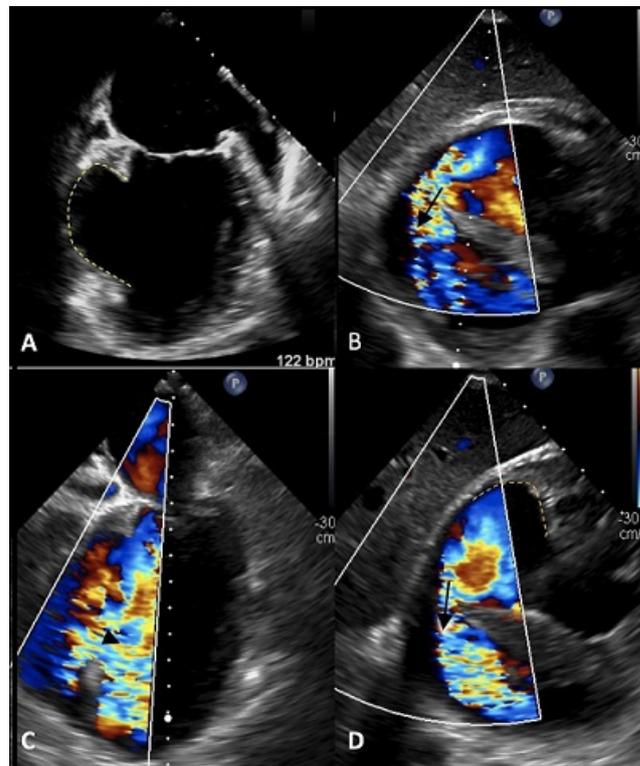
The patient had good recovery following cardiac rehabilitation, on 5 months follow-up, the patient is doing well.



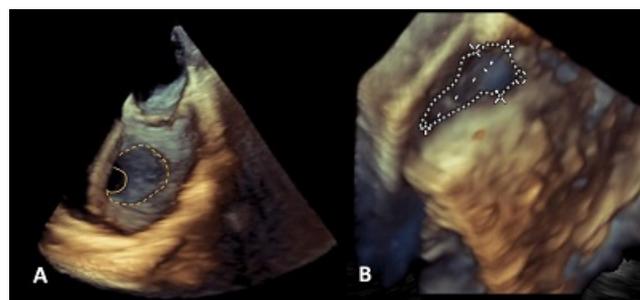
**Figure 1:** EKG shows sinus tachycardia with right axis deviation and q waves present in the inferior leads with T wave inversions.



**Figure 2:** Vascular congestion and bilateral interstitial changes are present, accompanied by bilateral effusions.



**Figure 3:** Trans esophageal echocardiographic images of VSD and Inferior basal aneurysm. A) Trans gastric view showing Inferior-basal aneurysm in dotted line. B) Off axis trans gastric view showing High velocity turbulent flow of left-to-right shunt (black arrow) by color Doppler across the ventricular septal rupture. C) Off axis trans gastric view showing VSR (black arrow) and Inferior-basal aneurysm. D) Off Axis trans gastric view of inferior basal aneurysm (dotted line) and VSD (black arrow)



**Figure 4:** 3D reconstruction of VSR. A) 3D Trans esophageal echocardiograph showing VS (Solid line) in the far field within the ventricular aneurysm (broken line). B) 3D-cropped, en-face view outlining the perimeter and extent of the VSR as viewed from the RV perspective.

## Discussion

Cardiac ruptures following acute myocardial infarction (AMI) include ventricular free-wall rupture (FWR), ventricular septal rupture (VSR), and papillary muscle rupture. Ventricular septal rupture (VSR) is a rare but lethal complication of acute myocardial infarction. With the advancement in reperfusion strategies for AMI, VSR has become increasingly rare and is recognized earlier in the post infarction course. However, the mortality of patient who develops VSR post MI still remains high at 48-87% [1]. The incidence of VSR was 1-2% before the thrombolytic era but has since declined to 0.17-0.31% with the advent of reperfusion therapy [2,3]. VSRs have been reported to occur 16-24 hr after acute myocardial infarction in the reperfusion era as opposed to 3-5 days previously [4,5]. In the SHOCK [4] trial registry and the GUSTO-1 [6] (The Global Use of Strategies to Open Occluded Coronary Arteries) trial, VSR occurred most often within the first 24 hours in patients with ST-segment elevation myocardial infarction treated with thrombolytic therapy. Older age, female sex, prior stroke, chronic kidney disease, and CHF are independent risk factors for VSR in patients presenting with AMI. Ventricular septal rupture seems to occur with similar frequency in anterior and inferior/lateral infarctions. Anterior infarctions are more likely to cause apical defects and inferior or lateral infarctions are more likely to cause basal defects at the junction of the septum and the posterior wall. Regardless of location, the newly formed communication results in left to right shunting of oxygenated blood from the left to the right ventricle. Clinical presentation varies from complete hemodynamic stability to frank circulatory collapse depending on the size of the defect, presence of RV infarction, ongoing RV ischemia, or stunning of the RV from volume overload.

Medical management of VSR carries high mortality and surgery remains treatment of choice. GUSTO-1 trial showed 94% mortality at 30 days without surgery suggesting that conservative management is associated with very high mortality [6]. Timing of surgery remains controversial the American College of Cardiology and American heart association guidelines recommend early surgical repair of VSR in setting of AMI [7], however the largest study of society of thoracic surgeons (STS) database showed improved outcomes with delayed surgery: 18.4% mortality for patients who underwent surgery after 7 days vs 54.1% mortality for those who underwent surgery within 7 days [1]. The improved outcome with delayed intervention is hypothesized to be related to evolution of the infarct, which allows a more effective surgical repair. Our patient had delayed surgical repair with good outcome, however the delay in surgery was unplanned as patient presented to our facility 2 weeks after the initial episode of AMI.

Our patient also had posterior true wall aneurysm, which is rare. Rupture of the free wall of the left ventricle is a catastrophic complication occurring in 4% [2] of patients after myocardial infarction. If the rupture is contained by an adherent pericardium a pseudo-aneurysm is created. A true aneurysm is an abnormal protrusion of the vascular wall, containing all layers of the original structure. About 85% of LV aneurysms are located antero-laterally near the apex of the heart and only 5% to 10% are posterior [2]. Clinically persisting ST-segment elevation, enlarged cardiac silhouette and calcifications adjacent to the LV border on the chest X-ray are suggestive for aneurysms. Pseudo-aneurysms, unlike true aneurysm, are prone to spontaneous rupture; hence, immediate surgical intervention is the treatment of choice.

## Conclusion

This rare case of inferior wall AMI complicated by both ventricular septal rupture and true posterior ventricular wall aneurysm presenting with impending cardiogenic shock, highlights the importance of noninvasive imaging for prompt diagnosis of these life-threatening complications. 3D-TEE done in pre-operative period can be useful to define the site and size of septal rupture, left and right ventricular function, and help plan a tailored surgical repair to the site of tear. The clinician needs to be aware of all the mechanical complications of AMI due to the fact that although the mortality associated with AMI has fallen substantially in the thrombolytic era; the mechanical complications post AMI still carries very high mortality. Our patient despite late presentation had a favorable outcome.

## Learning Points

- 1) 3D TEE is a useful technique in the assessment of patients with VSR post MI. It facilitates the understanding of the anatomy, and site of the lesion which in turn aids in tailored surgical approach to the site of lesion.
- 2) The mortality associated with mechanical complications post AMI still remains very high. In patients presenting with acute onset heart failure or shock post MI, prompt diagnosis with noninvasive imaging studies and multidisciplinary care is warranted for better outcomes.

## Conflicts of Interest

There are no conflicts of interest for the present study.

## References

1. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, et al. (2012) Surgical repair of ventricular septal defect after myocardial infarction: Outcomes from the Society of Thoracic Surgeons National Database. *Ann Thorac Surg* 94: 436-444.
2. Durko AP, Budde RPJ, Geleijnse ML, Kappetein AP (2018) Recognition, assessment and management of the mechanical complications of acute myocardial infarction. *Heart* 104: 1216-1223.
3. Moore CA, Nygaard TW, Kaiser DL, Cooper AA, Gibson RS (1986) Postinfarction ventricular septal rupture: the importance of location of infarction and right ventricular function in determining survival. *Circulation* 74: 45-55.
4. Menon V, Webb JG, Hillis LD, Sleeper LA, Abboud R, et al. (2000) Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: A report from the SHOCK Trial Registry. *J Am Coll Cardiol* 36: 1110-1116.
5. Jones BM, Kapadia SR, Smedira NG, Robich M, Tuzcu EM, et al. (2014) Ventricular septal rupture complicating acute myocardial infarction: A contemporary review. *Eur Heart J* 35: 2060-2068.
6. Crenshaw BS, Granger CB, Birnbaum Y, Pieper KS, Morris DC, et al. (2000) Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. *Circulation* 101: 27-32.
7. Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, et al. (2004) ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction). *J Am Coll Cardiol* 44: e1-e211.