INTERNATIONAL JOURNAL OF ADVANCE RESEARCH AND DEVELOPMENT

(Volume3, Issue4)

Available online at: www.ijarnd.com

Ventricular septal defect closure in ventricular septal rupture post-myocardial infarction: A case report

Dr. Uday Rajaram Patil

Student, Dr. D. Y. Patil Medical College, Navi Mumbai, Maharashtra

ABSTRACT

Ventricular septal rupture (VSR) after acute myocardial infarction is increasingly rare in the percutaneous coronary intervention era but mortality remains high. The event occurs 2-8 days after an infarction and often precipitates cardiogenic shock. [1] The differential diagnosis of postinfarction cardiogenic shock should exclude free ventricular wall rupture and rupture of the papillary muscles Prompt diagnosis is key. Alternatively, delaying surgery in stable patients may provide better results. Prolonged medical management is usually futile, percutaneous VSR closure may be used as a temporizing measure to reduce shunt, or for patients in the sub-acute to chronic period.

Keywords: *VSR, Ventricular septal rupture, Acute myocardial infarction, Percutaneous closure.*

1. PRESENTATION OF CASE

A 76-year-old woman presented to the Cardiology outpatient unit at Dr. D.Y Patil School of Medicine, Mumbai with intermittent chest pain for four hours that was found compatible with angina. She reported no previous history of cardiac disease, dyslipidemia or any other medical conditions, which could be considered cardiovascular risk factors.She denied previous symptoms of similar characteristics.

On admission, her blood pressure was 105/60 mmHg and heart rate was 90 bpm; she reported mild persistent chest pain. Physical examination revealed no abnormalities except for a systolic murmur (III/VI) on the left sternal border and mesocardium. Her ECG showed a complete absence of R waves and ST-segment elevation in leads V1–V2 (Figure 1).

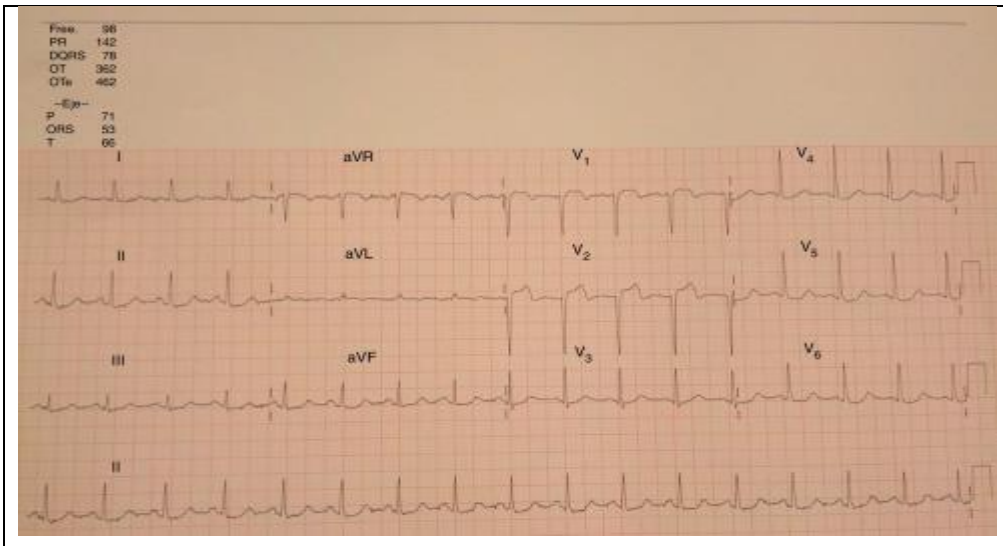

Figure 1. ECG showing 2 mm ST elevation, with Q waves and T-wave inversion, in V1–V2. ST depression can also be observed in the inferolateral leads.

2. DIFFERENTIAL DIAGNOSIS

ACUTE PERICARDITIS, MYOCARDITIS, AORTIC DISSECTION, OESOPHAGEAL RUPTURE

CLINICAL DIAGNOSIS

IMAGING DIAGNOSIS

Hand-held echocardiography showed no significant pathologic flows, although the patient's clinical situation made it difficult to carry out a complete assessment. She was immediately referred to the catheterization laboratory for percutaneous coronary intervention (PCI).

Coronary angiography showed no coronary lesions or signs of partially dissolved thrombus; coronary flow velocity was normal (Figure 2).

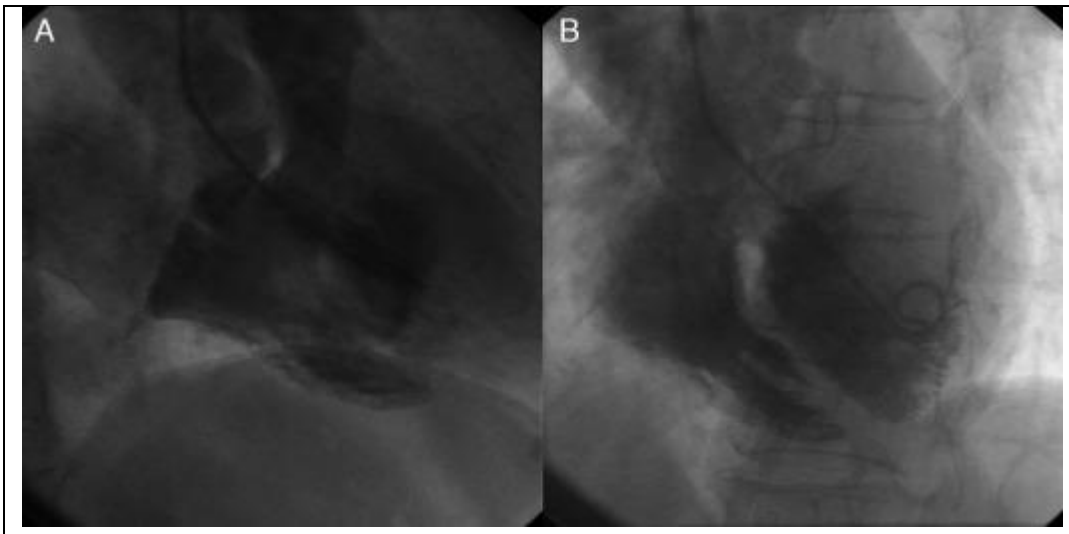

Figure 2

The septal branches from the left anterior descending and posterior descending arteries were reviewed carefully, but missing branches, significant atherosclerotic plaques or signs of ostial occlusion were ruled out. Ventriculography, performed to assess wall motion abnormalities, showed a ventricular septal defect (VSD). (Figure 3).

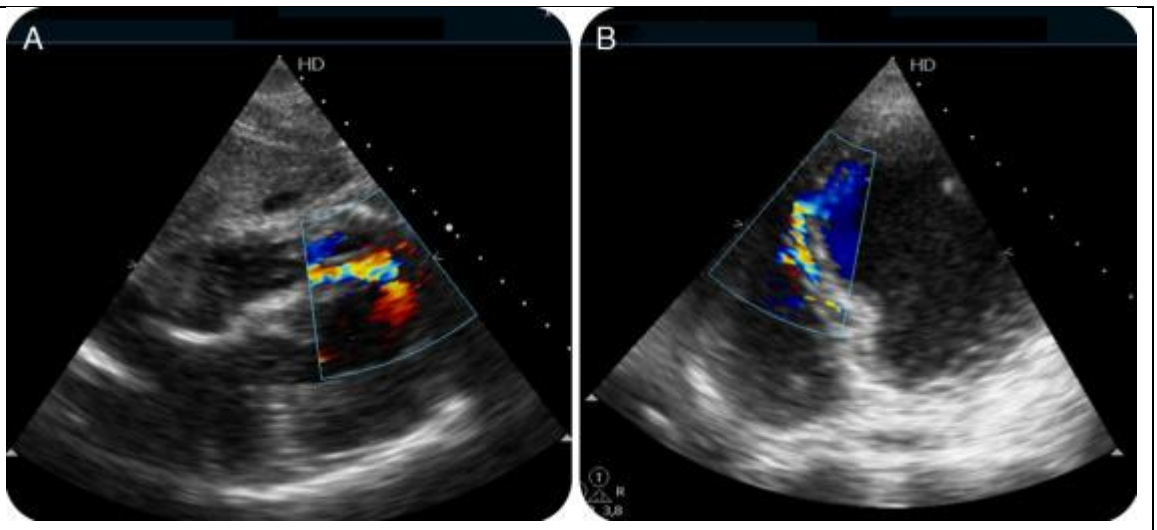

Figure 3

Intravascular ultrasound was not performed given the progressive worsening in the patient's clinical situation and evidence of septal rupture after ventriculography. She was transferred to the coronary care unit for clinical stabilization before surgical repair. Transthoracic echocardiography (TTE) confirmed the presence of a 7 mm dissection involving the apical third of the septum (Figure 4).

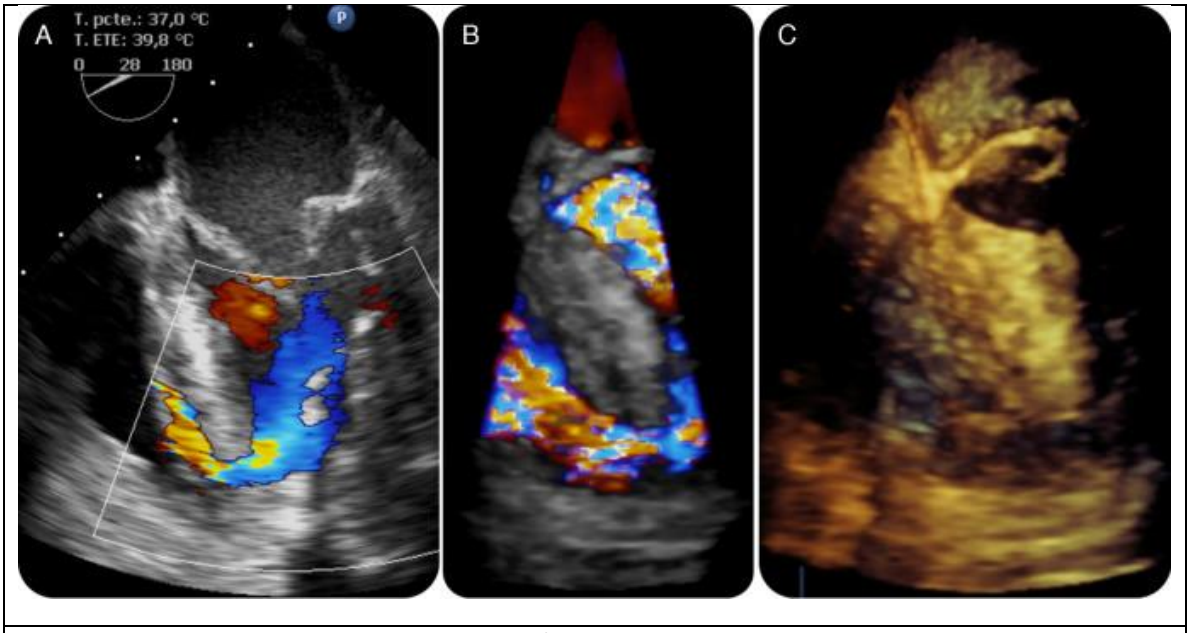

Figure 4

Initially, a direct through-and-through defect resembling a simple rupture was observed. However, additional images suggested a possible complex rupture with dissection tracts opening to the right ventricle in its mid-segment.

3. MANAGEMENT

Measurements of cardiac troponin I and creatine kinase were 6.3 ng/ml and 267 IU/L, respectively. The patient's clinical condition progressively worsened over 1 hour, requiring inotropic support and intra-aortic balloon counterpulsation.Emergency VSD closure was done but the patient developed cardiac arrest on cath table.Steps consisted of the following

The right femoral artery and right internal jugular vein were cannulated under local anesthesia. After a left ventricular angiogram, ventricular septal defect was crossed using 5 french right Judkin's (JR) catheter and a 0.035” × 260 cm angled tip Terumo wire (Terumo, Tokyo, Japan) using a retrograde arterial approach. It was passed into the right ventricle, to the main pulmonary artery and then into the left pulmonary artery. The right Judkin's catheter was advanced a little more into Right ventricular outflow tract (RVOT) and the Terumo wire was exchanged with 0.035”× 260 cm Teflon exchange length wire (Medtronic). The distal end of that wire was then caught with a 10 mm Amplatzer goose-neck snare (Microvena, MN) in the left pulmonary artery [Figure 7], and extracted outside via the right internal jugular sheath. A 12 Fr 180 degree curve device delivery sheath with its dilator (Star Way Medical Technology, Inc) was advanced from the jugular vein into the left ventricle (LV) across the VSD. Then dilator and the guide wire were removed. A 14 mm Cardio-O-Fix septal occluder was screwed onto the delivery cable and introduced into the sheath. The distal disc (left ventricle) was initially opened and pulled back onto the LV side of the septum under trans-thoracic echocardiography (TTE) and fluoroscopic guidance. After confirming the septal alignment, the proximal disc (right ventricle) was also opened. Once adequate placement was ascertained by TTE and left ventricular angiogram, the device was released consent for autopsy was not granted by the relatives.

4. DISCUSSION

The mortality rate in VSR with cardiogenic shock has been reported at 88% while in our study we had 75% mortality. In patients with RV dysfunction and inferior wall VSRs, mortality had been reported to be close to 100% and surgery is reported to be futile in these patients. Even in this series, it was found that patients with inferior wall VSRs and RV dysfunction had a mortality of 100%. Even those patients who have a successful device closure of the VSR often succumb to antecedent causes. The post VSR closure course in the ICCU is often stormy and requires careful monitoring to detect complications early and manage them appropriately. This study highlights that coronary lesions should be addressed on a later date for better results and primary focus should be on correction of mechanical complication.

5. FINAL DIAGNOSIS

VENTRICULAR SEPTAL DEFECT CLOSURE IN VENTRAL SEPTAL RUPTURE POST-MYOCARDIAL INFARCTION

Conflicts of interest

The authors have none to declare.

ETHICAL CONSIDERATIONS

Permission of ethical committee was taken and all ethical and standard protocols were followed during the study.

6. ACKNOWLEDGEMENTS

Special thanks to Dr Satish vaidya, Dr shantanu Deshpande, Dr Keshav kale, Dr. Amey chavan,Dr Somnath mallakmir, Dr Avinash Pawar, Dr vaibhav Patil, Dr Rohit Kumar,KP Goswami,Jeevansingh

7. CONCLUSION

Mechanical complications such as ventricular rupture are rare but is a possible complication of MI with normal coronary arteries. Although these are generally believed to have a more benign prognosis, the need for intensive care and observation may not differ significantly from cases of MI with coronary lesions.

8. REFERENCES

[1] Y.Y. Nan, M.S. Lu, K.S. Liu, et al.Blunt traumatic cardiac rupture: therapeutic options and outcomes Injury, 40 (2009), pp. 938-945
[2] C. De Diego, P. Marcos-Alberca, R.K. PaiGiant periprosthetic vegetation associated with pseudoaneurysmal-like rupture Eur Heart J, 27 (2006), p. 912
[3] Y. Birnbaum, M.C. Fishbein, C. Blanche, et al.Ventricular septal rupture after acute myocardial infarction N Engl J Med, 347 (2002), pp. 1426-1432
[4] B.S. Crenshaw, C.B. Granger, Y. Birnbaum, et al.Risk factors, angiographic patterns and outcomes in patients with ventricular septal defect complicating acute myocardial infarction Circulation, 101 (2000), pp. 27-32
[5] W.Y. Kang, M.H. Jeong, Y.K. Ahn, et al., Korea Acute Myocardial Infarction Registry InvestigatorsAre patients with angiographically near-normal coronary arteries who present as acute myocardial infarction actually safe? Int J Cardiol, 146 (2011), pp. 207-212
[6] G. Binetti, A. Maresta, G. Piovaccari, et al.Rupture of interventricular septum and ventricular aneurysm secondary to acute myocardial infarction in a case with normal coronary arteries G Ital Cardiol, 11 (1981), pp. 144-150
[7] P. St Louis, J.M. Rippe, J.R. Benotti, et al.Myocardial infarction with normal coronary arteries complicated by ventricular septal ruptureAm Heart J, 107 (1984), pp. 1259-1263
[8] L. Mendes, L. Raposo, R. Estefania-Fernandez, et al.Myocardial infarction without obstructive coronary disease complicated by ventricular septal rupture Rev Port Cardiol, 27 (2008), p. 859
[9] G. Nappi, L.S. De Santo, M. Torella, et al.Postinfarction ventricular septal defect in a patient without coronary lesions
Ann Thorac Surg, 75 (2003), pp. 1315-1317
[10] A. Viveiros Monteiro, A. Galrinho, L. Moura Branco, et al.Multiple mechanical complications in ST-segment elevation myocardial infarction with angiographically normal coronary arteries Rev Port Cardiol, 34 (2015) 209.e1–3
[11] B.S. Edwards, W.D. Edwards, J.E. EdwardsVentricular septal rupture complicating acute myocardial infarction: identification of simple and complex types in 53 autopsied hearts Am J Cardiol, 54 (1984), pp. 1201-1205
[12] C. Perdigão, A. Andrade, M. Ribeiro, et al.Incidence and forms of clinical presentation of the various morphologic types of myocardial laceration in the acute phase of infarct. A 4 years' caseload at a coronary unit. A clinico-anatomic study of 193 successive cases Rev Port Cardiol, 9 (1990), pp. 587-598

