Intraoperative Ventricular Septal Rupture: A Case Report

Hannah Rando[1] and Ahmet Kilic[2]

[1]Johns Hopkins Medicine School of Medicine
[2]The Johns Hopkins Hospital

June 15, 2022

Abstract

Ventricular septal rupture (VSR) is a rare complication of myocardial infarction that requires surgical repair. Herein we describe a case of intraoperative VSR requiring a second bypass run and patch repair. This case highlights the importance of early reperfusion and poses concern for delayed patient presentation during the COVID-19 pandemic.

Abstract

Ventricular septal rupture (VSR) is a rare complication of myocardial infarction that requires surgical repair. Herein we describe a case of intraoperative VSR requiring a second bypass run and patch repair. This case highlights the importance of early reperfusion and poses concern for delayed patient presentation during the COVID-19 pandemic.

Introduction

Ventricular septal rupture (VSR) is a rare complication of myocardial infarction that classically presents within one week of infarction[1] as coagulation necrosis occurs within the infarcted tissue. Prior to the widespread use of reperfusion therapy, VSR was estimated to complicate between 1-3% of acute myocardial infarctions,[2-4] whereas more recent estimates quote an incidence of 0.17%-0.34%.[5,6] While uncommon, VSR carries a high risk of mortality when it occurs, estimated between 19-54% even with optimal treatment.[2, 7-10]

Although some controversy exists regarding the optimal timing of repair, surgical treatment remains the mainstay of therapy. Without surgical consultation and repair, blood flow is shunted from the high-pressure left ventricle (LV) into the low-pressure right ventricle (RV), resulting in RV overload, pulmonary congestion, and eventual biventricular failure. Herein we present a rare case in which VSR occurred during a cardiopulmonary bypass (CPB) run for urgent coronary artery bypass grafting (CABG).

Case History

A 65-year-old male presented to our institution with sudden onset chest pain, nausea, and diaphoresis and was found to have ST elevation in leads I and aVL and ST depression in leads II, III, and aVF (**Figure 1**). He was diagnosed with an inferolateral wall myocardial infarction and was taken for emergent cardiac catheterization. Coronary angiography demonstrated 100% thrombotic mid-RCA occlusion with 80% mid-LAD and 80-90% mid-circumflex/proximal OM2 stenoses (**Figure 2**). He underwent balloon angioplasty and drug eluting stent placement to the RCA, and cardiac surgery was consulted for CABG given the presence of multi-vessel disease.

Treatment

Five days following initial presentation, the patient was taken to the operating room for CABG, with planned anastomoses of the left internal mammary artery (LIMA) to the left anterior descending artery (LAD),

and vein graft to the obtuse marginal artery. Transesophageal echocardiography (TEE) after induction of anesthesia demonstrated normal LV function and mild inferior wall hypokinesis, with no evidence of left-to-right shunting (**Figure 3a**).

Of note, while cannulating the right atrial appendage for venous drainage, the right atrium (RA) and RV immediately gave way and there was an abrupt change of hemodynamics. Out of concern for an RCA infarct or an abrupt obstruction of the RCA, an additional piece of vein was harvested, and the operative procedure was adjusted to include a vein graft to the right side.

Upon initiation of CPB, the patient was noted to have visibly infarcted myocardium in the inferior diaphragmatic aspect of the RV with significant hypokinesis. For this reason, the PDA was bypassed first before proceeding with the previously planned bypasses of the left circumflex with vein graft and LIMA to the LAD.

On attempted weaning of the CPB circuit, the aortic valve did not open, the LV did not distend, and any weaning maneuvers resulted in an overloaded RV. At this time, echocardiography demonstrated a new finding of a post-myocardial infarction VSR (**Figure 3b**). The location of the defect was in the basilar portion emanating from the mid-papillary muscle, consistent with a basal infarction in the distribution of the PDA.

A left ventriculotomy was made parallel to the LAD. This allowed visualization of a large, linear VSR with necrotic septum emanating between the papillary muscles and near the LVOT. The defect was repaired with a large piece of bovine pericardium secured with interrupted 2-0 Tycron pledgeted sutures. The ventriculotomy was closed and the cross-clamp removed.

After completion of the repair, the patient was able to be slowly weaned from bypass, but there was significant RV and LV strain despite placement of an intra-aortic balloon pump (IABP). Given the residual hemodynamic instability, the CPB circuit was converted to an ECMO circuit, with biatrial venous cannulae placed to minimize left to right shunting. The chest was left open and the patient was taken to the ICU for further management.

The patient's hemodynamics improved with resuscitation, but he demonstrated persistent RV dysfunction due to RV infarction as well as some residual left to right shunting suggesting an ongoing defect. Seven days after the index operation, he was returned to the operating room for sternal washout and attempted VSR repair. On direct visualization he was noted to have progression of myocardial necrosis. Although the patch was largely intact, there were small holes near the papillary muscles, necessitating reinforcement of the entirety of the patch with 2-0 Tycron pledgeted interrupted sutures.

Outcome and follow-up

The patient was continued on ECMO and returned to the ICU. Unfortunately, despite definitive surgical management of the VSR, he continued to demonstrate RV failure over the upcoming weeks and was unable to be weaned from ECMO. The decision was ultimately made to withdraw care.

Discussion

In this report, we describe a patient who presented with an inferolateral wall myocardial infarction and subsequently developed the mechanical complication of VSR during a planned CABG procedure. In this case, we elected to perform immediate surgical repair and utilized ECMO as a perioperative support strategy to alleviate biventricular dysfunction and allow for revision of the repair after the patient's condition had stabilized. Despite the ultimate patient outcome, we believe this case highlights several important points regarding perioperative management of VSR.

First and foremost, we acknowledge that the widespread use of early reperfusion therapy has made the incidence of VSR relatively minimal,[5,6] and for that reason it may not be high on the clinician's differential diagnosis when a patient decompensates. Particularly for the surgeon, who does not often encounter such a problem intraoperatively, it is important to be aware of this potential complication. We believe this to

be of particular importance during the COVID-19 pandemic, as patients are more likely to delay seeking care in an effort to avoid exposure to the virus in the healthcare setting.[11] Indeed, multiple studies have demonstrated a longer time from symptom onset to first medical contact in the setting of MI during the COVID-19 pandemic.[12-14]

Exemplifying this fact, we were surprised to encounter a similar case report by Kok et al. published in 2021.[15] The authors similarly describe a case of VSR during a CABG operation, in which the patient was managed with ECMO cannulation and delayed surgical repair. Despite the differences in management, this phenomenon of intraoperative VSR had not been described in the literature prior to 2021, and may reflect a rising rate of mechanical complications of MI due to delayed patient presentation. To date, there have been several case series and single institutional reviews that have demonstrated an increased incidence of mechanical complications during the COVID-19 pandemic,[16-18] but this has not yet been explored with a large database or multi-institutional review.

It is also worth noting the differences in patient management between the case report presented here and that of Kok et al, and more specifically, the differences in timing of surgical repair. The literature is divided in terms of optimal timing of VSR repair. While several studies have reported lower operative mortality in patients with delayed surgical repair,[19-22] these studies may have a significant component of selection bias, given that patients with smaller defects and preserved LV function have more favorable overall prognosis and are able to be medically temporized until definitive surgical repair. The advocates for delayed surgical repair argue that the delay allows for fibrosis of the septum and a more durable repair,[19,21] however without prompt repair many patients will develop progressive heart failure and will not survive the delay. Ultimately, we would argue that patient selection is critical, and the timing of surgical repair does not fall into a "one-size-fits-all" approach.

Finally, this case highlights the utility of ECMO as an adjunctive therapy in management of patients with VSR. Although no large prospective or retrospective studies have evaluated the efficacy of ECMO in the setting of VSR, several case reports and case series have reported good results when using ECMO in the perioperative period.[23-27] By affording complete cardiopulmonary support in the setting of cardiogenic shock, ECMO serves as a salvage therapy to allow time for myocardial rest and recovery. In this case, ECMO cannulation and delayed sternal closure provided the opportunity for myocardial recovery and hemodynamic stabilization with subsequent re-evaluation and revision of the ventricular septal repair.

In summary, VSR is a rare and often fatal mechanical complication of MI that merits clinical awareness and discussion. Although uncommon, given the additional myocardial manipulation that occurs in the operating room, the surgeon should be aware of the potential for intraoperative development of this complication. In these cases, the decision between immediate and delayed repair should be based on the size of the defect and the patient's overall clinical picture. In cases such as this, where immediate repair was needed to alleviate biventricular dysfunction, ECMO is a viable option both for temporary mechanical support and as a bridging modality to allow for re-evaluation and revision of the VSR repair at a later date.

References

1. Birnbaum Y, Fishbein MC, Blanche C, Siegel RJ. Ventricular septal rupture after acute myocardial infarction. N Engl J Med. 2002;347(18):1426-1432.
2. Topaz O, Taylor AL. Interventricular septal rupture complicating acute myocardial infarction: From pathophysiologic features to the role of invasive and noninvasive diagnostic modalities in current management. The American Journal of Medicine. 1992;93(6):683-688.
3. Radford MJ, Johnson RA, Daggett WM, et al. Ventricular septal rupture: A review of clinical and physiologic features and an analysis of survival. Circulation. 1981;64(3):545-553.
4. Pohjola-Sintonen S, Muller JE, Stone PH, et al. Ventricular septal and free wall rupture complicating acute myocardial infarction: Experience in the multicenter investigation of limitation of infarct size. The American Heart Journal. 1989;117(4):809-818.
5. Crenshaw BS, Granger CB, Birnbaum Y, et al. Risk factors, angiographic patterns, and outcomes

in patients with ventricular septal defect complicating acute myocardial infarction. Circulation. 2000;101(1):27-32.
6. Moreyra AE, Huang MS, Wilson AC, Deng Y, Cosgrove NM, Kostis JB. Trends in incidence and mortality rates of ventricular septal rupture during acute myocardial infarction. The American Journal of Cardiology. 2010;106(8):1095-1100.
7. Takahashi H, Arif R, Almashhoor A, Ruhparwar A, Karck M, Kallenbach K. Long-term results after surgical treatment of postinfarction ventricular septal rupture. European Journal of Cardio-Thoracic Surgery. 2015;47(4):720-724.
8. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: Outcomes from the society of thoracic surgeons national database. Ann Thorac Surg. 2012;94(2):436-444.
9. Coskun KO, Coskun ST, Popov AF, et al. Experiences with surgical treatment of ventricle septal defect as a post infarction complication. J Cardiothorac Surg. 2009;4:3.
10. Pang PY, Sin YK, Lim CH, et al. Outcome and survival analysis of surgical repair of post-infarction ventricular septal rupture. J Cardiothorac Surg. 2013;8:44.
11. Czeisler MÉ, Marynak K, Clarke KEN, et al. Delay or avoidance of medical care because of COVID-19–Related concerns — united states, June 2020. MMWR Morb Mortal Wkly Rep. 2020;69(36):1250-1257.
12. Mao Q, Zhao J, Li Y, et al. Impact of COVID-19 Pandemic on Mechanical Reperfusion in ST-Segment-Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention: A Multicenter Retrospective Study From a Non-epicenter Region. Front Cardiovasc Med. 2021;8:698923.
13. Aldujeli A, Hamadeh A, Briedis K, et al. Delays in Presentation in Patients With Acute Myocardial Infarction During the COVID-19 Pandemic. Cardiol Res. 2020;11(6):386-391.
14. Gao J, Lu P, Li C, et al. Reconsidering treatment guidelines for acute myocardial infarction during the COVID-19 pandemic. BMC Cardiovascular Disorders. 2022;22(1):194.
15. Kok CL, Balzereit A, Stooker W, Plonek TP. Intraoperative postinfarct ventricular septal rupture during coronary bypass grafting. BMJ Case Reports. 2021;14(9):e243824.
16. Lin TW, Tsai MT, Wu HY, Roan JN, Luo CY. Mechanical Complications of Acute Myocardial Infarction during the COVID-19 Pandemic. Acta Cardiol Sin. 2021;37(1):114-116.
17. Kitahara S, Fujino M, Honda S, et al. COVID-19 pandemic is associated with mechanical complications in patients with ST-elevation myocardial infarction. Open Heart. 2021;8(1):e001497.
18. Pilato E, Pinna GB, Parisi V, Manzo R, Comentale G. Mechanical complications of myocardial infarction during COVID-19 pandemic: An italian single-centre experience. Heart & Lung. 2020;49(6):779-782.
19. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database. Ann Thorac Surg. 2012;94(2):436-444.
20. Jeppsson A, Liden H, Johnsson P, Hartford M, Rådegran K. Surgical repair of post infarction ventricular septal defects: a national experience. Eur J Cardiothorac Surg. 2005;27(2):216-221.
21. Giuliani ER, Danielson GK, Pluth JR, Odyniec NA, Wallace RB. Postinfarction ventricular septal rupture: surgical considerations and results. Circulation. 1974;49(3):455-459.
22. Daggett WM, Guyton RA, Mundth ED, et al. Surgery for post-myocardial infarct ventricular septal defect. Ann Surg. 1977;186(3):260-271.
23. Rozado J, Pascual I, Avanzas P, et al. Extracorporeal membrane oxygenation system as a bridge to reparative surgery in ventricular septal defect complicating acute inferoposterior myocardial infarction. Journal of Thoracic Disease. 2017;9(9).
24. Matos D, Madeira M, Nolasco T, Neves JP. The role of extracorporeal membrane oxygenation in an acute basal ventricular septal rupture. European Journal of Cardio-Thoracic Surgery. 2020;57(4):799-800.
25. Neragi-Miandoab S, Michler RE, Goldstein D, D'Alessandro D. Extracorporeal membrane oxygenation as a temporizing approach in a patient with shock, myocardial infarct, and a large ventricle septal defect; successful repair after six days. J Card Surg. 2013;28(2):193-195.
26. Rohn V, Spacek M, Belohlavek J, Tosovsky J. Cardiogenic shock in patient with posterior postin-

farction septal rupture–successful treatment with extracorporeal membrane oxygenation (ECMO) as a ventricular assist device. J Card Surg. 2009;24(4):435-436.

27. Liebelt JJ, Yang Y, DeRose JJ, Taub CC. Ventricular septal rupture complicating acute myocardial infarction in the modern era with mechanical circulatory support: a single center observational study. Am J Cardiovasc Dis. 2016;6(1):10-16.

Figure Legends

Figure 1: EKG on initial presentation with ST elevation in leads I and aVL and ST depression in leads II, III, and aVF.

Figure 2: Coronary angiography with A) complete thrombotic mid-RCA occlusion, B) partial occlusion of the mid-LAD (80%) and mid-circumflex/proximal OM2 (80-90%).

Figure 3: Intraoperative echocardiography demonstrating A) no left to right shunting on initiation of bypass and B) a large VSR with left to right shunting on attempted weaning of cardiopulmonary bypass.