CASE REPORTS

VENTRICULAR SEPTAL RUPTURE AFTER ACUTE MYOCARDIAL INFARCTION – CAN VA-ECMO GIVE US EXTRA TIME?

Sara Teixeira[1]*, Inês Mendonça[1], Rita Ferreira[1], Sérgio Gaião[1], José Artur Paiva[1,2]

[1] Intensive Care Medicine Department, Centro Hospitalar Universitário de São João, Porto, Portugal
[2] Faculty of Medicine of University of Porto, Portugal

* Corresponding author: sara_isabel_10@hotmail.com

Abstract

Ventricular septal rupture (VSR) after myocardial infarction (MI) is a rare but life-threatening complication. Although surgery is the gold standard treatment, best surgical timing is still a matter of debate. Studies are showing a tendency towards survival improvement with delayed intervention on the assumption that scarring of the infarcted tissue may facilitate patch suturing and avoid relapse of the defect. Mechanical circulatory support (MCS) may be useful, not only for hemodynamic stabilisation and optimization before surgery, but also as a bridge to decide best surgical timing. Allowing myocardial recovery into a resistant scar, recurrence of VSR may be reduced and promotes a longer-lasting surgical repair.

The aim of this case report is to share the outcomes and management of two MI-associated VSR patients submitted to delayed surgery after VA-ECMO stabilisation. Although VSR repair failed, VA-ECMO allowed the patient's stabilisation, time for decision and became a bridge for successful heart transplant.

Keywords: *Ventricular septal rupture; myocardial infarction; mechanical circulatory support.*

INTRODUCTION

Ventricular septal rupture (VSR) after myocardial infarction (MI) is a rare but life-threatening complication. Since reperfusion became standard of care, its incidence decreased from 1-3% to 0.17-0.31%. However, mortality remains high reaching up to 94% without surgery in some reports[1-4]. Surgical mortality varies between 20-80% and worsens in the presence of cardiogenic shock (CS) before surgery. VSR develops, in most cases, with the occlusion of the left anterior descending or right coronary arteries, that supply the anterior-two thirds and the posterior one-third of the septum, respectively. The resultant ischemia evolves to necrosis and gives place to a friable tissue with territory for a left-to-right shunt to develop[1,3]. Not only size and location of the defect will influence the magnitude of the shunt, but also, both right and left, ventricular function (previous and after MI)[1,2]. It will cause a decrease in stroke volume and, without any intervention, in cardiac output. With reduced CO, there is an increase in systemic vascular resistance (SVR) to maintain mean arterial pressure (MAP). This rise in afterload associated with a poor contractility will promote biventricular overload leading to enhanced shunt, congestion and, ultimately, to CS[2]. Ineffective stabilization may prompt emergent surgery and worsen mortality rate.

Surgical treatment is the gold standard practice. However, although studies are showing a tendency towards survival improvement with delayed intervention, best surgical timing is still a matter of debate[1,2]. The choice of surgical technique is based on the localization, defect size, extent of ventricular dysfunction and also local expertise. Ventricular apical amputation may be an option for apical

defects[5]. For mid/basal VSR the 'infarct excision' and the 'infarct exclusion' techniques can be used[5]. The "excision technique" includes resection of the septal necrotic tissue by single patch reconstruction[5]. The "exclusion technique" consists of the use of pericardial or prosthetic material for the creation of a new ventricular septum sutured far from the necrotic edge at a non-infarcted site[5]. In both, the defect is approached via a ventricular anterior or inferior incision[5]. Basal and posterior defects represent a bigger challenge, due to their proximity to the subvalvular, mitral and tricuspid apparatus. Alternative approaches include a trans-mitral or trans-tricuspid valve access which avoids further injury of the ventricular myocardium[5]. For the "triple-patch technique," a smaller patch is used to close the defect and two large patches are used to exclude both ventricles[5].

After surgery, a residual shunt may persist due to the fragility of the necrotic tissue.

Delaying closure is based on the assumption that scarring of the infarcted tissue may facilitate patch suturing and avoid relapse of the defect[5]. In this context, mechanical circulatory support (MCS) can be used, not only for preoperative hemodynamic stabilization and optimization before surgery, but also as a bridge to decide best surgical timing. Allowing myocardial recovery into a resistant scar, recurrence of VSR may be reduced and promote a longer-lasting surgical repair.

CLINICAL CASE

Two male patients with 46 years (Patient A) and 58 years (Patient B), diagnosed with inferior MI due to right coronary artery occlusion. Both complicated with VSR and left-to-right shunt.

Patient A, with relevant medical history of smoking and alcohol consumption, was first admitted in the emergency department with complaints of dyspnoea, excessive sweating, and asthenia within 3 days of evolution. He was normotensive, slightly tachycardic and congestive. During examination a new holosystolic murmur was found and electrocardiogram had Q waves and ST-segment elevation in inferior leads. Echocardiogram showed ventricular septal rupture (VSR) with left-to-right shunt, located in the mid portion with a maximum gradient of 55mmHg. Septal and inferior wall akinesia, and biventricular dysfunction were present. During heart catheterization, right coronary artery occlusion and anterior descending and circumflex arteries anomalies were identified. He was then admitted in the coronary care unit where an intra-aortic balloon pump (IABP) was inserted for hemodynamic stabilisation. On the same day, the patient was transferred to our intensive care unit (ICU). A few hours later and because he maintained severe biventricular dysfunction despite IABP and dobutamine (maximum dose of 1.5mcg/kg/min) he was put on femoral-femoral veno-arterial extracorporeal membrane oxygenation (VA-ECMO) as a temporary bridge to shunt correction. Since admission and until the surgery, lactate levels were always maintained lower than 2mmol/L as well as adequate urine output. At day 8 (D8), he was submitted to scheduled VSR correction - mid-septal defect approach through ventricular incision and closure of both, access and defect, with bovine pericardium patch. Additionally, coronary artery bypass graft (CABG) to the right coronary artery was done and the patient was kept post-operatively on VA-ECMO. Several complications arose: difficulty to control intraoperative bleeding, neading several blood products; cardiac tamponade in the first 24h submitted to emergency resternotomy in the ICU, and septal patch malfunction a few hours later, with resurgence of ventricular shunt (maximum gradient of 80mmHg). Percutaneous closure was attempted 7 days later (D15), but it was unsuccessful. Concerning ECMO, there were no complications, with good distal limb perfusion and pump flow maintained between 2.7-3L/min. The patient was then submitted to heart transplant (HT) 11 days later (D26), decannulated from ECMO at D28 and ICU discharged at D37.

Patient B, with relevant past medical history of smoking, alcohol consumption, dyslipidemia and excessive weight was admitted in the emergency department with complaints of retrosternal pain, excessive sweating, and nausea since the same day. He was hemodynamically stable, with preserved urine output and normal lactate, without vasopressors or inotropes. Electrocardiogram showed ST-segment elevation in the inferior leads and echocardiogram detected a posterior-inferior mid-basal akinesia plus a mid-apical VSR with left-to-right shunt (máximum gradient of 60mmHg). Biventricular function was preserved. Right coronary artery occlusion was treated with stenting during primary percutaneous intervention and IABP insertion was done at the same time. The patient was then transferred to our hospital cardiology ward, needing IABP for circulatory support, and transitory non-invasive ventilation because of pulmonary congestion. On day 5, he got complicated with cardiac tamponade and emergent VSR repair was done (similar surgical approach to that described to patient A). No other mechanical support besides IABP was needed. Mild biventricular dysfunction was documented, but no apparent residual shunt was present after the procedure. Ventriculography 7 days after surgery showed a large septal defect with left-to-right shunt, so it was decided a re-intervention with apparent resolution of septal defect (D20). During the procedure the patient evolved with CS refractory to vasopressors and inotropes needing femoral-femoral VA-ECMO. Pump flow was maintained stable between 2.7-3.2L/min and no problems related with distal limb perfusion emerged. Favourable clinical evolution allowed decannulation from VA-ECMO 9 days later (D28). Percutaneous decannulation was complicated in the ICU so it was removed in the operating room. No other complications occurred. Unfortunately, on D32 ventricular shunt reappeared causing left ventricular dysfunction to develop. Because ventricular dysfunction progressed despite medical therapy, the patient was submitted to HT (D40). He was discharged from ICU 8 days later.

Table 1 **Comparative data of patients A and B**

Patient	A	B
Sex	male	male
Age	46y	58y
Medical history	smoking; alcohol consumption	smoking; alcohol consumption; dyslipidemia; excessive weight
Symptoms on admission	dyspnoea, sweating, asthenia	retrosternal pain; sweating; nausea
Initial evaluation	normotensive, tachycardic, congestive; new holosystolic murmur	normotensive, normocardic;
Electrocardiogram	Q waves, ST-segment elevation in inferior leads	ST-segment elevation in inferior leads
Echocardiogram	Septal and inferior wall akinesia; biventricular dysfunction	Posterior-inferior mid-basal akinesia; preserved biventricular function
Ventricular septal defect	Mid portion; maximum gradient of 55mmHg	Mid-apical portion; maximum gradient of 60mmHg
Coronary artery occluded Intervention	Right coronary CABG	Right coronary Angioplasty with stenting
IABP insertion	Day 1	Day 1
VA-ECMO cannulation	Day 1; femoral-femoral	Day 20; femoral-femoral
VSR correction	Day 8; ventricular approach; closure of access and defect with bovine pericardium patch	Day 5; ventricular approach; closure of access and defect with bovine pericardium patch
Post-operative complications	None	Intraoperative bleeding; cardiac tamponade in the first 24h submitted to emergency re-sternotomy
VSR relapse	First 24h after surgery (D9)	7 days after surgery (D12)
Heart transplant	Day 26	Day 40
ECMO decannulation	Day 28	Day 28
ICU Discharge	Day 37	Day 48

DISCUSSION

Until surgical intervention can be done, the cornerstone management of these patients resides in lowering left ventricular afterload to decrease the interventricular shunt. When possible, an association of IABP and vasodilators is used to stabilise the patient. Inotropes are also often necessary to maintain cardiac output[2]. If it is not enough, other MCS devices may be necessary for hemodynamic stabilisation and optimization of the patient status before surgery[2]. The use of different devices such as ECMO, Impella, TandemHeart, ventricular assist devices (VAD) and total artificial heart have been described in different reports[1,2,4]. The hemodynamic changes induced by MCS in the presence of a VSR depend on the configuration or association of different devices. Each one has potential benefits, but also downsides we must be aware of.

IABP allows afterload reduction, favours left ventricle (LV) contractility, while reducing septal shunt and increasing CO. However, it often provides limited support. In these situations requiring more support, peripheral VA-ECMO may be a good option since it provides biventricular support and a higher flux. Nevertheless, although enabling right ventricle (RV) unloading, it increases both LV afterload and left-to-right shunt. Paradoxically, this may simultaneously reduce aortic valve opening and blood ejection, reducing CO, and lead to RV overload. Association of IABP is interesting because it may counteract these effects. Impella has also been described in some reports, but despite unloading the LV/RV, the presence of VSR has been considered a contraindication because of the risk of aspiration of debris and embolization, besides the risk of shunt inversion. Another device described is Tandemheart which promotes LV unloading through left atrial drainage, without the risk

of aspiration of debris. The problem may arise if excess drainage occurs causing inversion of the shunt and hypoxic brain injury. Other types of MCS are VADs which also provide unloading of the ventricle, but are usually used after initial haemodynamic stabilisation. Similarly to Impella, depending on the localization of the inflow cannula, there is the risk of aspiration and, therefore, embolization of debris. If the unloading is too much, it may also cause inversion of the shunt with the risk of hypoxic brain injury. Besides, after a MI, myocardium tissue is fragile and the high flux generated by the device causes stress over fresh sutures and may lead to patch dehiscence.

If pharmacologic and MCS fail, surgical intervention may be required urgently. There is no certainty of the optimal timing for surgery, however early surgery is associated with a high mortality rate, reported as high as 80%[1,6]. Arnaoutakis et al.[7], found an overall in-hospital or 30-day mortality of 42.9%, and a significant decrease in mortality with delay in surgical repair: 54.1% with repair within 7 days from MI versus 18.4% after 7 days. Other published series also show a decline in mortality with delay in VSR repair[2,4,7,8]. Reported surgery delay since VSR diagnosis varies between 2 to 19 days depending on the series[1,2,4,8].

In the situations presented, Patient A had both IABP and VA-ECMO introduced on the same day of VSR diagnosis, was submitted to VSR repair at D8 and had a total duration of ECMO of 28 days; Patient B was first supported at diagnosis with only IABP. VA-ECMO was introduced at D20 and kept for 16 days. Unfortunately, both VSR repair failed no matter the surgery delay and the use of ECMO. Nonetheless, ECMO did allow hemodynamic stabilisation and, more importantly, supported them enough time and became a bridge for successful heart transplant.

More evidence is needed to understand what type or association of MCS is best and which are the targets of optimal surgery delay in these populations. Still, these examples highlight the different role that MCS, specifically VA-ECMO, may have: stabilisation to avoid shock after MI, time to allow tissue healing and surgery planning, circulatory support during the delicate peri-operative período and after the surgery, or as a bridge to other alternatives such as heart transplant.

REFERENCES

1. Zbikowska K, Wróbel K. Mechanical Circulatory Support in Delayed Surgery of Post-Infarction Ventricular Septal Rupture in Patients in Cardiogenic Shock - A Review. JCM. 2022 Aug 12;11(16):4728.
2. Ronco D, Matteucci M, Ravaux JM, Marra S, Torchio F, Corazzari C, et al. Mechanical Circulatory Support as a Bridge to Definitive Treatment in Post-Infarction Ventricular Septal Rupture. JACC: Cardiovascular Interventions. 2021 May;14(10):1053–66.
3. O'Gara PT, Kushner FG, Ascheim DD, Casey DE, Chung MK, De Lemos JA, et al. 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction: Executive Summary. Journal of the American College of Cardiology. 2013 Jan;61(4):485–510.
4. Shibasaki I, Otani N, Saito S, Ogawa H, Masawa T, Tsuchiya G, et al. Overview of mechanical circulatory support for the management of post-myocardial infarction ventricular septal rupture. Journal of Cardiology. 2023 May;81(5):491–7.
5. Schlotter F, Huber K, Hassager C, Halvorsen S, Vranckx P, Pöss J, et al. Ventricular septal defect complicating acute myocardial infarction: diagnosis and management. A Clinical Consensus Statement of the Association for Acute CardioVascular Care (ACVC) of the ESC, the European Association of Percutaneous Cardiovascular Interventions (EAPCI) of the ESC and the ESC Working Group on Cardiovascular Surgery. European Heart Journal. 21 de julho de 2024;45(28):2478–92.
6. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with STsegment elevation. European Heart Journal. 2018 Jan 7;39(2):119–77.
7. 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. The Annals of Thoracic Surgery. 2012 Aug;94(2):436–44.
8. Morimura H, Tabata M. Delayed surgery after mechanical circulatory support for ventricular septal rupture with cardiogenic shock. Interactive CardioVascular and Thoracic Surgery. 2020 Dec 7;31(6):868–73.