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CASE REPORT

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Intraoperative ventricular septal rupture: A case report

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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 patch repair and postoperative extracorporeal membrane oxygenation (ECMO) support. This case highlights the risk factors, patient presentation, and management recommendations for this potentially lethal pathology.

KEYWORDS

cardiothoracic surgery, cardiovascular disorders

1 | INTRODUCTION

Ventricular septal rupture (VSR) is a mechanical complication of myocardial infarction that classically presents within 1 week of the initial insult as coagulation necrosis occurs within the infarcted tissue.[1,2] Prior to the widespread use of reperfusion therapy, VSR was estimated to complicate between 1 and 3% of acute myocardial infarctions,[3–5] whereas more recent estimates quote an incidence of 0.17–0.34%.[6–8] While uncommon, VSR carries a high risk of mortality when it occurs, estimated between 19 and 54% even with optimal treatment.[4,9–12]

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).

2 | CASE HISTORY

A 65-year-old male patient presented to our institution with a 3-day history of 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 (MI) and was taken for emergent cardiac catheterization. Coronary angiography demonstrated a 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 (DES) placement to the RCA, and cardiac surgery was consulted for CABG given the presence of multi-vessel disease.

Perioperatively, the patient was admitted to the cardiovascular progressive care unit for further monitoring. He was continued on antiplatelet therapy with aspirin for his DES, but ticagrelor was held in preparation for upcoming CABG. Of note, the patient developed recurrent angina on post-procedure day one, corresponding to an increased troponin of 2.05 from 0.74 immediately post-procedure.

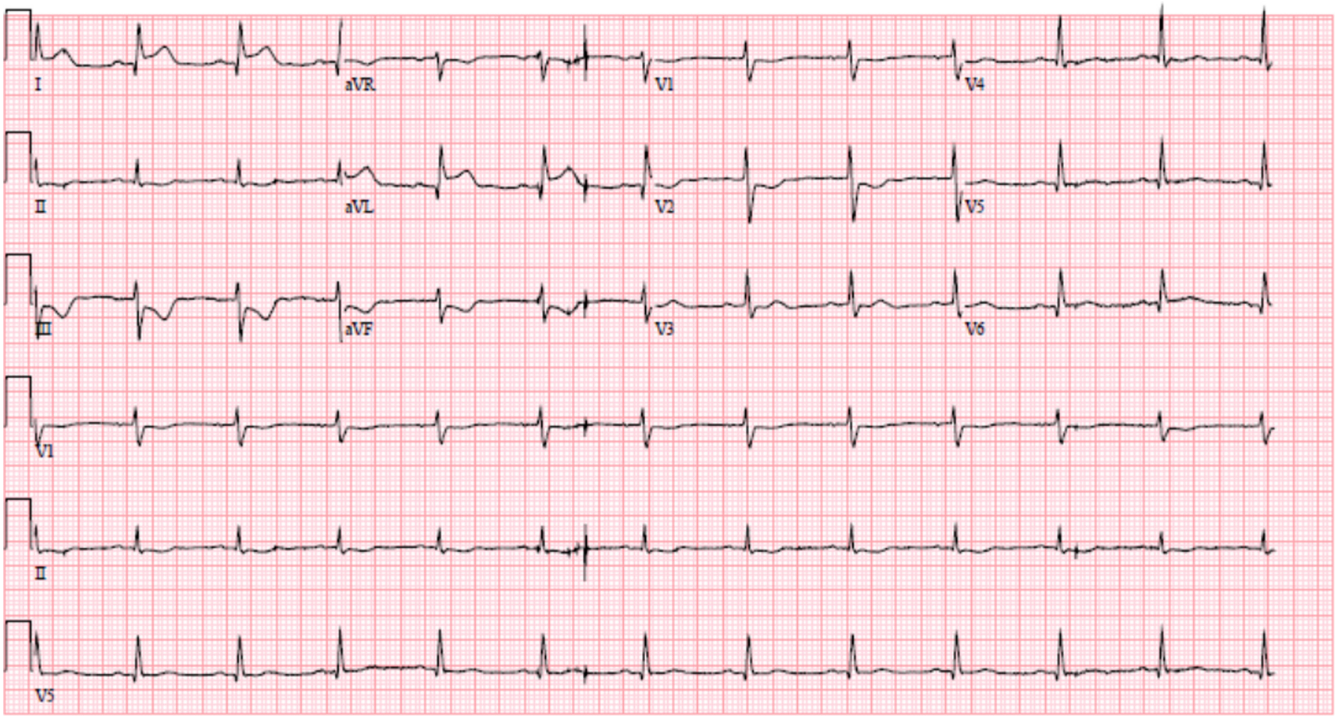

FIGURE 1 EKG on initial presentation with ST elevation in leads I and aVL and ST depression in leads II, III, and aVF.

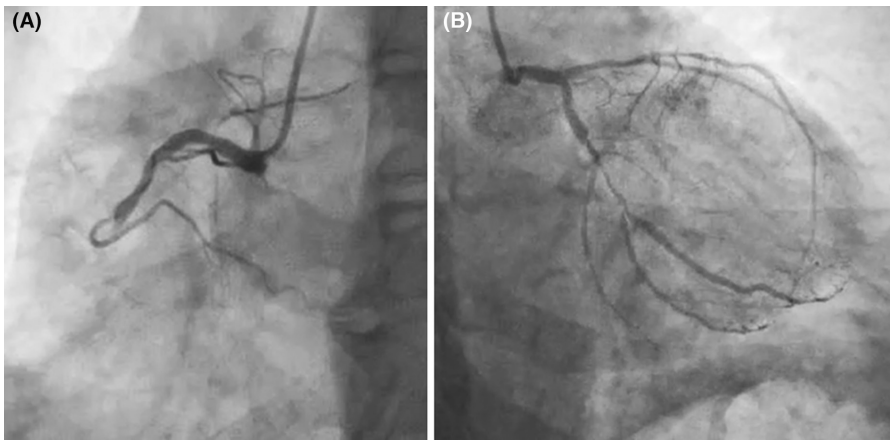

FIGURE 2 Coronary angiography with (A) complete thrombotic mid-RCA occlusion, (B) partial occlusion of the mid-LAD (80%) and mid-circumflex/proximal OM2 (80–90%).

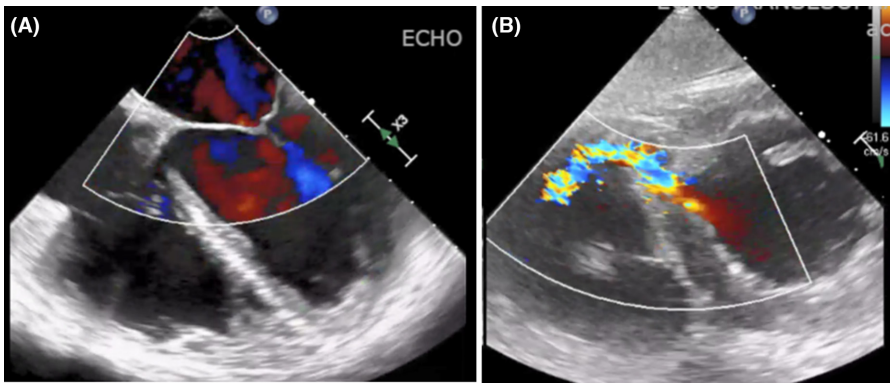

FIGURE 3 Intraoperative echocardiography demonstrating (A) no left to right shunting on initiation of bypass and (B) a large VSR with left to right shunting on attempted weaning of cardiopulmonary bypass.

His angina resolved with administration of sublingual nitroglycerin and intravenous morphine, and he was subsequently started on a cangrelor drip, which was continued until the time of surgery.

3 | 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.

4 | 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.

5 | DISCUSSION

In this report, we describe a patient who presented with an inferolateral wall MI 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 the risk factors and tenets of management for VSR.

Given that VSR occurs as a consequence of progressive myocardial ischemia and compromised tissue integrity, the most important risk factors for VSR are those that permit ongoing ischemia, such as delayed reperfusion, incomplete coronary revascularization, and lack of collateralization of coronary blood flow.[9,13,14] Particularly for the surgeon, who does not often encounter such a problem intraoperatively, it is important to be aware of this potential complication when the patient has one or more of these characteristics. In this case, the patient presented after several days of symptoms, indicating a prolonged period of ischemia that most likely resulted in the ultimate VSR. Another possible explanation for this complication is stent thrombosis of the RCA, given the patient's recurrent anginal symptoms and rising troponin on post-procedure day one in the setting of transiently paused dual antiplatelet therapy (DAPT).

The delayed patient presentation seen here is worth discussing in light of the ongoing impact of the COVID-19 pandemic, as patients may be more likely to delay seeking care to avoid exposure to the virus in the healthcare setting.[15] 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.[16–18] Exemplifying this fact, we were surprised to encounter a similar case report by Kok et al. published in 2021.[19] 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 variation in management between these two cases,

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. Although the societal impact of COVID-19 has lessened since the onset of the pandemic, this pattern of healthcare avoidance could recur with the introduction of new variants, and is therefore important to bear in mind in the interest of preventing potentially lethal sequelae such as VSR. In a report released by the Center for Disease Control in the height of the pandemic, individuals most at risk of delayed or avoided care included unpaid caregivers for adults, individuals with underlying medical conditions or disabilities, and minority populations[15]; these individuals should be among the first targeted in messages encouraging timely presentation.

Although the most likely cause of VSR in this case was the delay in initial presentation, the possibility of stent thrombosis also highlights the importance of perioperative antiplatelet therapy following percutaneous coronary intervention (PCI) to reduce the risk of reinfarction and development of mechanical complications such as VSR. The standard perioperative management of DAPT is to withhold P2Y12 inhibitors such as ticagrelor or clopidogrel for 5–7 days before CABG to minimize intraoperative and postoperative bleeding complications from platelet inhibition,[20,21] but in patients at high risk of stent thrombosis (such as those with recent DES placement), the risk of bleeding from ongoing platelet inhibition must be weighed against the risk of occlusion.[22,23] In cases such as this, perioperative bridging with glycoprotein IIb/IIIa inhibitors or cangrelor, which have shorter half-lives, is a viable option to reduce the risk of perioperative stent thrombosis without increasing perioperative blood loss.[24–27] Our patient was managed with a cangrelor bridge for the majority of the perioperative period, but there was a transient period of time without DAPT. Particularly in cases where DAPT is held without use of a bridging agent, the clinician should be aware of the risk for stent thrombosis and reinfarction, and should closely monitor for any evidence of recurrent ischemia in the interest of preventing mechanical complications such as VSR.

An additional consideration highlighted by this case is the decision to pursue immediate surgical repair, particularly in the rare circumstance of VSR development in the intraoperative setting. Even in the far more common case of VSR outside of the operating room, the literature is divided in terms of the optimal timing of repair. While several studies have reported lower operative mortality in patients with delayed surgical repair,[10,28–30] these studies may have a significant component of selection bias, given that patients with smaller defects and preserved RV and 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[10,29]; however, without prompt repair many patients will develop progressive heart failure and will not survive the delay. In the case reported by Kok et al., surgical repair was delayed for 10 days, and the patient was supported with ECMO in the interim. We believe the key difference between these two cases lies in the overall hemodynamic stability of the patient and the degree of RV dysfunction present. In cases where delayed repair is likely to result in irrecoverable heart failure and severe organ dysfunction, immediate surgical repair may be the best option to prevent progressive RV overload and dysfunction. Conversely, if the patient is able to be stabilized with pharmacologic and mechanical RV and LV unloading techniques, delayed repair may be the preferred approach to improve the durability of patch repair. 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.

Other than the timing of repair, the case by Kok et al. differs only in the preoperative management of the culprit lesion. While our case required immediate PCI to restore coronary perfusion, the patient described by Kok et al. experienced spontaneous reperfusion and had no additional interventions performed prior to CABG. The major risk of patients managed with delayed PCI or CABG following spontaneous reperfusion is that of reinfarction, which, although reportedly rare,[31,32] was likely the source of VSR in the case reported by Kok et al. Regardless, these two cases collectively emphasize the risks of VSR following incomplete or delayed reperfusion, the patient-specific factors when considering optimal timing of repair, and the utility of ECMO in perioperative management.

Indeed, as both the number of ECMO cases and the number of ECMO centers have seen tremendous growth in recent years,[33] this is an important adjunctive therapy to consider for patient optimization, regardless of the timing of surgical repair. Although no large prospective or retrospective studies have evaluated the efficacy of ECMO specifically in the setting of VSR, several case reports and case series have reported good results when using ECMO in the perioperative period.[34–38] 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 our 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, the surgeon should

be aware of the potential for intraoperative development of this complication, particularly in urgent or emergent cases with delayed or incomplete revascularization, or in patients at high risk for stent thrombosis and reinfarction. 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 circumstances where immediate repair is 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.

6 | CONCLUSION

Ventricular septal rupture should be suspected in cases of delayed or incomplete revascularization when an abrupt change in hemodynamics or RV overload is observed. The mainstay of VSR management is with surgical repair, which can be done on an emergent or delayed basis depending on the stability of the patient and the size of the defect.

AUTHOR CONTRIBUTIONS

Hannah Rando: Data curation; formal analysis; methodology; writing – original draft; writing – review and editing. **Ahmet Kilic:** Conceptualization; data curation; methodology; supervision; writing – review and editing.

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CONFLICT OF INTEREST

All authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

DATA AVAILABILITY STATEMENT

Data sharing not applicable - no new data generated.

CONSENT TO PUBLISH

Written consent for publication was obtained from the next of kin.

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