Clinical vignette

A 82-year-old man with a past medical history of gastroesophageal reflux with Barrett’s esophagus, hypertension, and dyslipidemia presented with an acute ischemic ventricular septal defect (VSD). He presented to a peripheral hospital with ST-elevation myocardial infarction (STEMI) of the anteroseptal territory. He was treated with intravenous thrombolytics. He had persistent chest pain and was transferred to our hospital for coronary angiography, which revealed recanalization of the culprit lesion in the left anterior descending artery but with residual stenosis. A decision was made to treat with a drug eluting stent. Urgent transthoracic echocardiography demonstrated a large VSD with an enlarging left-to-right shunt. His left ventricular ejection fraction was estimated at 45–50% with reasonable right ventricular function. Serial laboratory investigations revealed rising lactate and serum creatinine, and he had become anuric. We discussed with the patient the high-risk nature of potential intervention and presented the options of ongoing medical therapy alone, extracorporeal membrane oxygenation, and definitive repair. He wished to proceed with emergent repair of the post-myocardial infarction VSD.

Surgical technique

Cannulation strategy

Through midline sternotomy, the patient was centrally cannulated in the usual fashion in the proximal aortic arch and right atrial appendage. In order to facilitate the prophylactic left ventricular assist device (LVAD), we routinely cannulate the right superior pulmonary vein with a 24-Fr metal tipped, angled cannula, externalized through a stab wound at the bottom of the sternal incision. This cannula is initially used for venting the left ventricle (LV), and later converted to the LVAD inflow cannula. Two pledget-reinforced prolene sutures are used for the cardioplegia cannulation site in the anterior ascending aorta, which is later converted to a 20-Fr straight arterial cannula externalized through the bottom of the incision and used for the LVAD outflow cannula. We feel that the prophylactic insertion of CentriMag LVAD support can greatly help to facilitate gradual loading of the acutely infarcted and repaired LV, provide superior hemodynamics and minimize the risk of crash extracorporeal membrane oxygenation, which is generally associated with very poor outcomes (1).

Ventricular septal defect repair

On preoperative imaging the ventricular defect appeared to have an anteroapical orientation. After cardioplegic arrest, we performed a left ventriculotomy 1.5–2 cm parallel to the left anterior descending artery starting distally and extending proximally, to the point that we could visualize the extent of the LV infarct. The VSD was easily identified through this incision and the infarcted tissue extended high up on the septum, extending posteriorly to the base of the papillary muscle and all the way to the apex. We cleared the
edges of the VSD of any loose tissue that might embolize, but we do not routinely debride around it. We then tailored a large polytetrafluoroethylene (PTFE) outflow patch to the shape of the planned area of LV exclusion, much larger than the area of the VSD. We started with the suturing of the deep superior and posterior margins of the patch to the healthy endocardium with multiple interrupted pledgeted prolene sutures to then parachute the patch down onto the most difficult portions to sew. We ran a second layer of running 3-0 prolene, working from deep to superficial on either side of the patch. The anterior portion of the patch is folded in to exclude the infarcted LV and sewn within the ventriculotomy closure line along with felt strip reinforcement in multiple layers of large prolene stitches. Added reinforcement sutures were placed in any areas of concern, particularly around the base of the papillary muscle that was juxtaposed to the infarct. We did not hesitate to oversee the distal left anterior descending artery within the ventriculotomy closure as the distal myocardium had no viability.

Transition to prophylactic LVAD

We completed de-airing maneuvers, removed the root vent/cardioplegia line and switched it for a 20-Fr straight cannula, externalized through a stab wound at the bottom of the incision. The tip of the cannula was placed 4–5 cm deep in the aorta to minimize risk of accidental dislodgement and secured with Rummel tourniquets. Then, the right superior pulmonary vein cannula was disconnected from the pump and attached as the inflow for the CentriMag LVAD and LVAD support was initiated as cardio-pulmonary bypass support was weaned.

Post-operative course and management

In the intensive care unit (ICU) the patient stabilized, and end organ function recovered. During his ICU stay, the patient was extubated and ambulated as much as tolerated. He required minimal inotropic support and had his left ventricular support gradually weaned over five days with daily transthoracic echocardiographic guidance. Transesophageal echocardiography revealed adequate bi-ventricular function to support independent circulation, thus the patient was decannulated successfully in the operating room.

He continues to recover in the hospital but has not required re-escalation of ventricular support. We have used this strategy systematically for emergency repair of post-myocardial infarction complications including papillary muscle rupture, free wall rupture and VSD.

Comment

Advances in percutaneous management of acute coronary events have greatly reduced the incidence of mechanical complications after ischemic events (2). However, mechanical complications still occur in around 1% of patients after a myocardial infarction and perhaps may be more frequent during the Covid-19 pandemic due to delays in patient presentation and care (3). Medical management is usually not sufficient and is associated with a high mortality (above 90% at 30 days) (4). In high volume centers, survival after surgical VSD repair has acceptable hospital and long-term mortality (31.2% mortality during hospitalization and 51% actuarial survival at 10 years) (5).

The choice of surgical technique will depend on several factors including type of VSD (anterior vs. posterior), extent of infarcted area, degree of cardiogenic shock, patient context and surgeon experience. There are many modalities and timing of mechanical support use during post infarction VSD repair (6). We routinely use central CentriMag left ventricular support at the completion of surgery regardless of post-operative ventricular function. This permits a period of hemodynamic stability in which the patient can recover end organ function and heal myocardium without concern for low cardiac output. Our strategy is reproducible and does not require expensive devices.

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Footnote

Conflicts of Interest: MWAC has received speakers honoraria from Medtronic, Edwards Lifesciences, Abbott Vascular, Terumo Aortic and Cryolife. The other authors have no conflicts of interest to declare.

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References
