An Update Review on Postinfarction Ventricular Septal Rupture

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When ventricular septal rupture (VSR) occurs following acute myocardial infarction (AMI), the mortality is still high. To save the patient, prompt diagnosis, proper management and satisfactory surgical closure are mandatory. This discussion of VSR management offers some information on the epidemiology, pathogenesis, diagnosis, management, and repair procedures from with a surgeon’s point of view. Although definitive surgery remains the treatment of choice, it remains a challenge carrying high early mortality. Percutaneous closure may provide an option for patients whose conditions preclude surgical repair, and may be useful to close a recurrent shunt. Prompt diagnosis of VSR with referral to an experienced heart team is important for the improvement of clinical outcomes.

KEY WORDS: acute myocardial infarction, cardiogenic shock, mechanical complication, surgical closure, ventricular septal rupture

I. Incidence

Before the introduction of reperfusion therapies, VSR occurred in 1 to 3 percent of AMI cases (1-6). In the era of reperfusion therapy, Crenshaw et al. reported the VSR incidence in the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries trial (GUSTO-I) at 0.2 percent, which indicated a reduction of the incidence of VSR since the introduction of reperfusion therapies (7). In spite of this reported reduction the real-world incidence may not have continued to improve as much as one might hope. Moreyra et al. found from the MIDAS database that the rate of VSR had not changed from 1990 to 2007, and that mortality had remained high and relatively constant (8). Annual reports from The Japanese Association for Thoracic Surgery, including data from almost all the centers nationwide demonstrated fairly consistent numbers of VSR repairs, ranging from 275 to 296 cases annually from 2010 to 2017 (9-14).

VSR is more probable after anterior myocardial infarction than after inferior infarction (2, 6-7). Risk factors for VSR in the pre-reperfusion era included hypertension (15, 16), advanced age (60 to 69 years) (17), female sex (15, 17), and no history of angina or myocardial infarction (1, 2, 18-20). Angina or myocardial infarction may stimulate the growth of coronary collaterals, which may reduce the development of rupture (20). In the reperfusion era, advanced age, female sex, and the absence of smoking are often associated with an increased risk of developing VSR (7), but the absence of previous angina has not been associated with an increased risk (27).

II. Pathogenesis

After acute myocardial infarction with ST segment elevation (STEMI), VSR may develop anywhere in the septum. Anterior infarction is more likely to cause anterior VSR, typically apical VSR, and inferior infarction is more likely to cause posterior VSR, particularly in the basal septum. Anterior VSR is generally simple (Fig. 1) and posterior VSR is often complex (Fig. 2), occasionally involving the ventricular free wall or papillary muscles (6). Wherever the septum ruptures, a left-to-right shunt also develops and clinical presentations range from relatively stable hemodynamics to apparent cardiogenic shock depending on the size of VSR and hence the amount of left-to-right shunting flow. Sudden unpredictable deterioration of hemodynamic condition is common in the days and weeks after VSR occurs, because the margin of a VSR is fragile and the hole tends to enlarge. Long-term survival is extremely rare without surgical closure of that hole.

Septal rupture generally occurs within the first week after STEMI without reperfusion therapy (3, 5, 22, 23). There are two peak distributions of VSR, with a high incidence on the first day and on days 3 through 5. It rarely develops more than two weeks after infarction. The time from the onset of symptoms of STEMI to VSR is generally 24 hours or less in patients who are receiving thrombolysis (24). The median time from the onset of STEMI to VSR was 1 day (range, 0 to 47 days; 94 percent of cases were diagnosed within 1 week) in the GUSTO-I trial (7) and 16
hours in the Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock (SHOCK) trial\textsuperscript{25}. Although thrombolytic therapy reduces the size of the infarct, it may promote hemorrhagic dissection in the myocardium in some cases, accelerating the onset of septal rupture\textsuperscript{6}.

III. Diagnosis

It is crucial that all patients with acute myocardial infarction should be briefly examined for mechanical complications of AMI prior to primary percutaneous coronary intervention (PCI)\textsuperscript{25}. A harsh, systolic precordial murmur is characteristic of VSR and mitral regurgitation caused by papillary muscle rupture, and an accentuated pulmonary component of the second heart sound and rales may be audible, but clinical manifestations of VSR vary with the status of each patient, from having no symptoms, through chest discomfort, palpitation and progressive dyspnea. Since an apparently stable condition usually deteriorates unexpectedly, it is advisable for clinicians to perform echocardiography for any subtle clinical changes in STEMI patients. If a Swan-Ganz catheter is in place, high oxygen saturation of the mixed venous blood, sampled from the tip of the catheter, is characteristic of a newly developed left-to-right shunt to differentiate from papillary muscle rupture\textsuperscript{41}.

Transthoracic echocardiography is usually adequate to confirm a diagnosis of VSR. The septal defect should be visible by 2D image, and shunt flow through the septum is seen by color Doppler. It may also be advisable to assess the anatomical size of the defect. When the patient has poor acoustic windows due to endotracheal intubation, a transesophageal echo should be considered.

When VSR is found prior to emergency catheterization, primary PCI strategy no longer takes priority, and dual-antiplatelet medications should not be administered, because these medications potentially increase the risk of bleeding in an emergency surgical VSR closure. Emergency consultation with an experienced cardiac surgeon is crucial at this point, and heart team decision-making is strongly recommended for each patient strategy.

IV. Medical management

Prolonged medical management is futile with rare exception. Definitive surgical closure remains the treatment of choice\textsuperscript{20}. Therefore medical management of VSR is a bridge and is directed to reducing left ventricle (LV) afterload, to increase effective LV stroke volume by reducing left-to-right shunting. Sodium nitroprusside may be an ideal short half-life intravenous agent, but its use is limited in Japan. Intra-aortic balloon counterpulsation (IABP) provides the most effective support, at least temporarily, for VSR patients in reducing afterload and augmenting cardiac output. As mentioned above, this temporary stabilization does not guarantee any reduction of sudden deterioration.

Some reports describe extra-corporeal membrane oxygenators (ECMO) and left ventricular assist devices used as a bridge to surgery\textsuperscript{27, 28}, but details are limited to case reports. Another recent device strategy is the Impella as a bridge to surgery with 30 day mortality of 40\%\textsuperscript{29}. These reports are interesting, but still limited in scope.

V. Surgical VSR closure

Although definitive surgery remains the treatment of choice, it has remained a challenging operation carrying high early mortality\textsuperscript{20}. The first surgical repair of VSR was reported in 1957 by Cooley\textsuperscript{20}. The patient temporarily recovered but died on postoperative day 45, suggesting recurrence of the shunt flow. Daggett
used infarctectomy and septal reconstruction through LV incision, reporting 52% operative mortality when repair was attempted within 21 days, but only 7% when done after 3 weeks\textsuperscript{31}. David introduced a single pericardial patch infarct exclusion without infarctectomy through a left ventriculotomy in 1990\textsuperscript{32}; it seemed conceptually simple, and became a standard technique. Nonetheless, operative mortality is higher when VSR repair is attempted soon after myocardial infarction. Arnaoutakis et al. reported on the largest recent database of The Society of Thoracic Surgeons (STS) in 2012\textsuperscript{33}. Overall operative mortality was 42.9%, but if repair was attempted within 7 days from MI, operative mortality was 54.1%. These more urgent patients tend to be in worse general conditions, with more cardiogenic shock, preoperative IABP, preoperative intubation and multi-organ dysfunction. The preoperative cardiogenic shock itself carries a high mortality. They concluded that VSR remained a devastating complication in spite of numerous surgical attempts. Annual reports from The Japanese Association for Thoracic Surgery (JATS), with data from 2010 to 2017, demonstrated hospital mortality ranging from 26.7% to 39.7% when VSR repair was attempted within 14 days after MI, and from 8.8% to 23.5% when more than 14 days after\textsuperscript{9-14}.

Predictors for poor outcome include cardiogenic shock, the need for repair within 7 days after AML, posterior VSR and shunt recurrence. It is impossible to differentiate freshly infarcted tissue from viable myocardium intraoperatively in acute VSR. If one fixes a patch with tiny partial-thickness bites and running sutures on the freshly infarcted wall, one risks myocardial tearing, cut-through, patch dehiscence and shunt recurrence. Major shunt recurrence can be fatal. If a patient survives long enough after VSR, the tissue around the defect becomes stable, and the surgical mortality will be low\textsuperscript{31}. However, many patients with rapid deterioration into cardiogenic shock need emergency surgery. Although surgical mortality has remained high, non-surgical mortality would certainly be higher.

Recent development of a novel surgical procedure by the author may eliminate that persistent dilemma\textsuperscript{34-36}. We have proposed the “extended sandwich patch technique” (Fig. 3), which is particularly immune from shunt recurrence in the acute phase. A Dacron patch is cut to size to overlap the edges of the defect by about 2 cm; it is introduced through a right ventricle (RV) and through the septal defect into the left ventricle. Extremely secure fixation is achieved by placing large mattress sutures transseptally or transmurally around the defect. This simple tech-
nique can be applied to both anterior and posterior VSR. Shunt recurrence is mostly eliminated even with very acute VSR. We believe that if surgical risks of mortality and shunt recurrence are sufficiently reduced, there is no need to delay surgery. If surgical repair can be done both early and with minimal risk, the sort of unexpected deterioration described above is no longer a problem. Currently our results show that 30 day mortality has been 10%, with shunt recurrence at 6.7%, among 30 consecutive cases including 90% acute VSR (< 7 days). This promising surgical procedure is gradually being adopted among many cardiac surgeons.

VI. Percutaneous VSR closure

Percutaneous closure of VSR is another option in patients at significant risk from surgical closure. It is directed either for definitive closure or for a bridge to surgical VSR closure, as well as closure of a residual defect after surgery. Several small series have been reported.

Despite less invasive technique and high procedural success, percutaneous intervention mortality remains higher than open surgical mortality in experienced centers. Dislocation of the device, residual and recurrent shunt, LV rupture and RV rupture have been reported. Similar to surgical reports, outcomes tend to improve as patients progress from acute to chronic phase. Anatomically the defect margin is fragile and in ongoing necrosis in acute VSR. Attempting to pass a guide-wire and closure device through a VSR may increase the aperture. It is challenging as the defects frequently lack an adequate tissue rim to secure the closure device. Most authors suggest that the small sized defect (<15 mm) is optimal.

VII. Conclusion

Although mortality due to AMI has fallen dramatically in the past thirty years, the mortality of VSR remains high. Prompt diagnosis, a component of cautious management of each STEMI patient, is crucial. Once the diagnosis of VSR is confirmed, medical management to reduce afterload and to institute IABP should be considered immediately. Prior to primary PCI, emergency heart team decision-making including consultation with an experienced cardiac surgeon, is highly advisable. Long-term survival is extremely rare without surgical closure. Complete surgical closure before cardiogenic shock can develop is the goal in this lethal disease. Percutaneous closure may provide an option for patients whose conditions preclude surgical repair, and may be useful to close a residual shunt. The management of VSR requires critical care with sophisticated surgical and other interventional expertise. It is recommended that those patients be transferred to reference centers with experience if such resources are not locally available.

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Conflict of Interest Statement

The author has no conflict of interest.

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