Commentary: Left ventricular free wall rupture: Patch and unload?

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Central Message

Use of a hemostatic patch is an effective method to manage oozing-type LV free wall rupture. Mechanical complications in the early postoperative period are common and careful surveillance is required.

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Left ventricular free wall rupture (LVFWR), is a rare event in the era of percutaneous coronary intervention and thrombolysis—0.2% to 0.5% in modern series—because most full-thickness myocardial infarctions are prevented by timely presentation to hospital and revascularization^{1,2}

William Harvey first described LVFWR at autopsy in the mid 17th century.³ Hatcher and colleagues⁴ described the first long-term outcomes of 3 patients who underwent surgery between 1968 and 1971 using horizontal mattress sutures through the surrounding myocardium with large polytetrafluoroethylene felt strips. Two of their 3 patients survived beyond 1 year. Núñez and colleagues⁵ described the use of a large polytetrafluoroethylene patch over the rupture secured by continuous 2-0 polypropylene suture in the surrounding normal myocardium on cardiopulmonary bypass (CPB) between 1980 and 1981.⁵ Padró and colleagues⁶ first described the use of a sutureless technique in 13 patients operated on between 1986 and 1991 without using CPB. They placed a large 5 cm × 8 cm polytetrafluoroethylene patch over the defect and fixed it with topical tissue adhesive. They noted no mortality in their series.

Okamura and colleagues⁷ describe what must be among the largest surgical series of LVFWR to date. Between 2001 and 2016, the group applied a hemostatic collagen patch, often with fibrin glue, in 35 patients without CPB. Fifty-three percent of LVFWRs were in lateral or inferior wall territories; the authors used an off-pump coronary artery bypass stabilizer to optimize exposure without hemodynamic compromise. Rerupture occurred in 6 of 35 patients (17%) and was an independent predictor of in-hospital and midterm mortality. Rerupture occurred away from the original area of rupture. Three patients required subsequent mitral valve surgery and another developed a ventricular septal defect that was surgically repaired. Twenty-nine of 35 patients (83%) survived to discharge. Of 6 patients who died, 3 had rerupture. Another 6 patients died during the first year of follow-up, highlighting the poor

prognosis of this condition even after successful surgical repair.

Okamura and colleagues⁷ are to be congratulated on their results in this high-risk patient group that support the use of this technique in the oozing form of LVFWR. The technique is advantageous because it can be performed in all territories of the heart without the need for CPB and heparinization and is not particularly technically demanding. Meticulous monitoring of patients during the postoperative period is required for early detection of subsequent mechanical complications. Clearly, in those with more aggressive forms of LVFWR, a more traditional surgical intervention of resection and patch repair is required.

Further thought should be directed toward preventing the mechanical complications that occurred postoperatively in approximately 26% of patients—2 ruptured papillary muscles, 1 ventricular septal defect, and 6 reruptures. Unloading the ventricle postoperatively may benefit with intra-aortic balloon pump, the Impella device (Abiomed, Danvers, Mass), and/or left ventricular assist device. An Impella 5.0 may be preferable because it can be placed via an axillary artery to allow early mobilization and can completely unload the left ventricle.⁸⁻¹¹ This may prevent complications during the vulnerable period of coagulative necrosis when the muscle is weakest, but has the downside of low-level heparinization.

Although rare, continued improvement in techniques is required for the management of this lethal disease. We encourage longer-term follow-up of survivors of LVFWR to determine the durability of this novel technique.

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