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Video Presentation

Repair of post-infarction multiple anterior muscular ventricular septal defects by endocardial Dacron patch and left ventricular aneurysmectomy: a video presentation

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Introduction

Mechanical complications resulting from myocardial infarction, namely ruptured mitral valve papillary muscle, ruptured interventricular septum and left ventricular aneurysm, occur in 10-20% of patients after acute myocardial infarction [1-5]. Post-infarction ventricular septal defect is usually located in the anterior or apical portion of the ventricular septum (about 60% of cases) as a result of transmural, anterior myocardial infarction [6]. These patients usually do not have well-developed coronary collateral circulations [7].

The clinical course is usually one of fulminant pulmonary edema and cardiogenic shock. Only about 75% survive the first 24 hours and only 50% in the first week [8,9]. Surgical techniques have gradually evolved since the first successful operation for the post-infarct ventricular septal defect was performed by Denton Cooley in 1957, 11 weeks after myocardial infarction [10-14].

Hospital mortality after repair of the post-infarct ventricular septal defect is approximately 30-40% [15-18]. The actuarial 5-year survival has ranged from 44-57% and 10-year survival from 29% to 36% [15-18]. The hazard function for death after surgery has not only a high and rapidly declining early phase, but also an appreciable constant phase that is five times greater than after isolated coronary artery bypass grafting [15-18].

We present here-in a 66-year-old male patient diagnosed to have single-vessel coronary artery disease (left anterior descending artery-100% occlusion) and post-infarct multiple anterior ventricular septal defects with unstable hemodynamics. The patient underwent single vessel aortocoronary saphenous vein bypass grafting, Dacron patch closure of the ventricular septal defect and linear Teflon felt-supported left ventricular repair. Postoperative recovery was uneventful. Perioperative intra-aortic balloon counterpulsation was required for 96 hours.

Surgical techniques

The great saphenous vein is harvested from the left leg for the purpose of coronary revascularization. The left internal mammary artery is not harvested for extreme sternal osteoporosity and concomitant severe chronic obstructive pulmonary disease. Following median sternotomy, the thymus is subtotally excised taking care not to expose the brachiocephalic vein. The pericardium is opened in the midline

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in between stay sutures using scissors, and not cautery to avoid inadvertent cautery-induced ventricular fibrillation on a distended, poorly contractile cardiac chambers.

The operation is performed with moderately hypothermic cardiopulmonary bypass using angled venous cannulas into superior and inferior caval veins and aortic cannulation. The main pulmonary artery is being vented to facilitate cardiac decompression and prevent intraoperative soiling of blood through the coronary arteriotomy. The right pleural cavity is widely opened for later dislocation of the cardiac mass to facilitate exposure and prevent compression injury to the necrotic ventricular mass.

After aortic cross-clamping, myocardial preservation is achieved by using combined antegrade and retrograde cold hyperkalemic blood cardioplegia (integrated myocardial protection of Buckberg) and topical ice-cold saline. The reversed saphenous vein is anastomosed to the left anterior interventricular coronary artery using 7-0 polypropylene sutures (Johnson and Johnson Ltd., Ethicon, LLC, San Lorenzo, USA).

Additional graft cardioplegia is used on completion of the distal anastomosis for perfusion of the distal coronary and septal perforator vessels and for checking hemostasis. A laparotomy pack is placed behind the heart with the pulmonary artery vent on suction to facilitate exposure. The apex and diaphragmatic surface of the heart are lifted. The outlines of the ventricular infarct and ventricular aneurysm are identified.

The bulging necrotic myocardium overlying the apex and anterolateral surface of the left ventricle are identified and opened in between pledget supported stay sutures without causing injury to the adjacent coronary arteries. The loose red clot located within the trabeculae of the left ventricle is carefully removed using Russian forceps. The left ventricular cavity is thoroughly irrigated using cold saline. Through the left ventriculotomy, multiple muscular ventricular septal defects (one large and two small) involving almost one-third of the ventricular septum are identified and sized.

A slightly redundant over-sized Dacron polyester patch (Bard® Savage® filamentous knitted polyester fabric, Bard Peripheral Vascular Inc., Tempe, AZ, USA) is sutured to close the ventricular septal defects using 4-0 polypropylene sutures buttressed by medium-sized Teflon felt pledgets. The single patch is used to close all three defects. Extreme precautions are taken while tying the sutures, ensuring perfect apposition and hemostasis. Myocardial perfusion is restored through retrograde coronary sinus perfusion cannula at a rate of 150-200 mL/min, maintaining the coronary sinus pressure of 25-30 mmHg.

Two Teflon strips are placed on two sides of the ventriculotomy, just to the right of the left anterior interventricular coronary artery. Multiple interrupted horizontal mattress sutures of 3-0 polypropylene (Johnson and Johnson Ltd., Ethicon, LLC, San Lorenzo, USA) are used for repair of the defects. The sutures are passed through the Teflon felt, the parietal wall of the left ventricle and out through the opposite ventricular wall and Teflon felt strip ensuring no injury to the coronary arteries. Another long Teflon strip is placed over the raw edges of the ventriculotomy scar and sutured over the same using the 3-0 polypropylene sutures.

Care is taken to avoid injury to the papillary muscles and the adjacent coronary arteries. After completing the repair and releasing the aortic cross-clamp, ensuring perfect hemostasis, proximal coronary anastomosis is performed on the ascending aorta using 6-0 polypropylene suture.

After coming off bypass, oximetry is performed between the right atrium and pulmonary artery to exclude residual ventricular septal defect. Additionally, transesophageal echocardiography demonstrated severe ventricular dysfunction (left ventricular ejection fraction 0.20) and no residual ventricular septal defect. Postoperatively, the patient had moderate left ventricular dysfunction, left ventricular ejection fraction (0.50) on dopamine 7.5 µg/kg/min and Adrenaline 0.1 µg/kg/min. The patient required perioperative intra-aortic balloon counterpulsation for 96 hours.

Conclusion

We conclude that pre- and perioperative hemodynamic monitoring, judicious use of vasodilators and antiarrhythmic drugs, appropriate timing of surgical intervention, improved methods of myocardial preservation and perioperative use of intra-aortic balloon counterpulsation are the major determining factors for improved surgical outcome.

Conflict of interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of the article.

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