**Institutional report - Coronary**

**Beating-heart approach and moderately delayed surgical management of postinfarction ventricular septal rupture: an advisable approach to improve results?**

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**Abstract**

Early repair of postinfarction ventricular septal rupture is usually associated with significant mortality and morbidity. Assessing conditions to moderately delay the surgical intervention and reduce cardioplegic arrest may be an interesting approach to improve hospital results. Results of five non-selected patients in whom surgery was moderately delayed and performed according a policy of reducing the cardioplegic-induced ischemia were reviewed. Surgical options are discussed as well as observed advantages.

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**Keywords:** Postinfarction ventricular septal rupture; IABP; Beating heart; Right atrial approach

1. Introduction

Ventricular septal defect (VSD) is a severe complication of acute myocardial infarction (MI). Spontaneous evolution is dismal within 3 months [1]. However, with a mortality of 10 to 52% [1–3], surgical treatment remains a challenge and postoperative outcome is influenced by the age [2], the preoperative clinical status [4], the surgical technique of VSD [1,2,5], the time interval between infarction and operation [3,6] and the importance of ventricular impairment. This latter feature depends on the severity of infarction with its subsequent stunned myocardium but also from acute hemodynamic modifications generated by the left-to-right intraventricular shunt, leading to impair the right ventricle with a risk of biventricular failure.

Therefore, surgeons painstakingly developed strategies to improve results [6,7]. Since duration of cardioplegic arrest is known to produce detrimental effects on freshly infarcted myocardium and consequently increases difficulties during attempts to wean the patient from CPB, introduction of the beating heart concept in surgical treatment of VSD may be an interesting approach for reducing cardioplegic-induced ischemia. In order to validate factors influencing hospital results, we report our experience of a moderately delayed repair of postinfarction VSD using systematic preoperative intraaortic balloon and beating heart techniques in five patients. This concept was utilized either for myocardial revascularization or as a whole technique to repair apical VSD.

2. Materials and methods

From May 2001 to March 2004, 5 non-selected patients underwent surgical repair of postinfarction VSD. Preoperative patient characteristics are summarized in Table 1. Diagnosis of septal rupture was assessed by echocardiography. Anatomic and flow patterns of VSD are given in Table 2. Intraaortic balloon pump (IABP) was systematically considered for all patients and inserted once admitted in ICU. Then, surgery was moderately delayed to optimize hemodynamic conditions. Mean delay between IABP insertion and surgery was 12.2 ± 2.59 days. Cardiogenic shocks and low cardiac output syndromes were stabilized and coronaryography was performed in all patients. MI was attributable to left anterior descending (LAD) artery in 4 cases and right coronary artery in 1 case. Additional significant stenosis involved circumflex artery in two patients and diagonal branch in one. Predicted number of coronary bypass was 1.2/patient. In order to decrease duration of cardioplegic-induced ischemia, complete or partial beating heart approach was considered.

Therefore, patients with apical VSD were assigned for a complete clampless procedure without any ventriculotomy including myocardial revascularization under support of CPB whereas three patients were assigned for a beating heart myocardial revascularization and repair of VSD under cardioplegic arrest.
Shunt fraction and IABPs were removed 24 h after chest closure. The pulmonary bypass time was 75 min. Weaning from CPB was uneventful.

Prior history of coronary artery disease: 3 patients had a history of coronary artery disease.
- Angina: 2 patients
- Previous MI: 1 patient
- Diabetes: 2 patients
- HTA: 3 patients
- COPD: 2 patients
- Anterior: 1 patient
- Inferior: 1 patient

Coronary angiography:
- Single vessel disease: 2 patients
- Double vessel disease: 3 patients
- Triple vessel disease: 0 patients

Cardiogenic shock before IABP insertion: 3 patients

Operations were performed through a median sternotomy.

2.1.1. Beating heart myocardial revascularization
Following institution of a clampless normothermic CPB with bicaval venous drainage, coronary bypasses were performed with the Octopus® stabilizer (Medtronic) prior to VSD closure. Intracoronary shunts were systematically used. LIMA was utilized in five cases (LAD: n=3; OM: n=2) and saphenous veins in three cases (diagonal: n=1; OM: n=2).

2.1.2. Repair of apical VSD (n=2)
A clampless normothermic circulatory assistance with no ventriculotomy was established. The venous drainage created a biventricular deflation with an epicardial apical depressed area clearly delimitating borders of the septal rupture (Fig. 1A). Then, septal repair was performed by an external apical exclusion using a ‘sandwiched’ felt-buttressed technique with transventricular interrupted sutures (Fig. 1B,C). CPB decreased myocardial stress and allowed an adequate placement of transventricular sutures without any mechanical tension. No ventriculotomy was required. Completeness of closure was assessed by transesophageal echography. This technique was used twice. In one patient, coronary bypass was associated (Fig. 1D). Mean cardiopulmonary bypass time was 75±14 min whereas aortic cross clamp time was 0 min. Weaning from CPB was uneventful and IABPs were removed 24 h after chest closure.

2.1.3. Repair of VSD located on midportion of septum (n=2)
Following beating heart revascularization, aorta was clamped and cardioplegic arrest was achieved by means of cold blood cardioplegia delivered in an antegrade manner through the ascending aorta and the aortocoronary venous bypass. Posterior pericardial stitches were placed to improve cardiac exposure and maintain anterior wall enucleated from the pericardial cavity. Surgical access to septal defect was addressed through a left ventriculotomy at the level of infarcted area. Exposure was improved by stay sutures and septal rupture was identified. After a moderate debridement, interrupted sutures with pledges were placed from the right ventricle through the septum, all around the border of the defect. A patch was then anchored, and left ventriculotomy was closed with interrupted felt-buttressed sutures, reinforced by a continuous prolene suture. This approach was used twice. Mean cardiopulmonary bypass and aortic cross clamp times were respectively 97±17 min and 57±7 min. Weaning from CPB was uneventful and required low doses of dobutamine. In one case, IABP was removed immediately after chest closure. In the second case, IABP could be removed at the second postoperative day.

2.1.4. Repair of inferobasal VSD (n=1)
Following beating heart revascularization, aorta was clamped and cardioplegic arrest was achieved with cold cardioplegic bypass, undernormothermic CPB. A: CBP-induced epicardial apical depression (D) delineating borders of VSD. B: External apical amputation (no ventriculotomy). C: Final aspect. D: Final aspect with LAD/LIMA bypass.

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### Table 1
Baseline characteristics of patients and risk factors

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/Female</td>
<td>1/4</td>
</tr>
<tr>
<td>Mean age (ys) ± S.D.</td>
<td>75.6±5.3</td>
</tr>
<tr>
<td>HTA</td>
<td>3</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2</td>
</tr>
<tr>
<td>COPD</td>
<td>2</td>
</tr>
<tr>
<td>Prior history of coronary artery disease</td>
<td>3</td>
</tr>
<tr>
<td>- Angina</td>
<td>2</td>
</tr>
<tr>
<td>- Previous MI</td>
<td>1</td>
</tr>
<tr>
<td>Site of MI</td>
<td></td>
</tr>
<tr>
<td>- Anterior</td>
<td>3</td>
</tr>
<tr>
<td>- Lateral</td>
<td>1</td>
</tr>
<tr>
<td>- Inferior</td>
<td>1</td>
</tr>
<tr>
<td>Extension to the right ventricle</td>
<td>1</td>
</tr>
<tr>
<td>Site of VSD</td>
<td></td>
</tr>
<tr>
<td>- Distal apex</td>
<td>2</td>
</tr>
<tr>
<td>- Midpart</td>
<td>2</td>
</tr>
<tr>
<td>- Inferobasal</td>
<td>1</td>
</tr>
<tr>
<td>Coronary angiography</td>
<td></td>
</tr>
<tr>
<td>- Single vessel disease</td>
<td>2</td>
</tr>
<tr>
<td>- Double vessel disease</td>
<td>3</td>
</tr>
<tr>
<td>- Triple vessel disease</td>
<td>0</td>
</tr>
<tr>
<td>Cardiogenic shock before IABP insertion</td>
<td>3 (1 pt intubated)</td>
</tr>
<tr>
<td>Low cardiac output</td>
<td>2</td>
</tr>
<tr>
<td>Preoperative IABP</td>
<td>5</td>
</tr>
<tr>
<td>Preoperative cardiogenic shock</td>
<td>0</td>
</tr>
<tr>
<td>Preoperative RV failure</td>
<td>1</td>
</tr>
<tr>
<td>Mean LVEF (%) ± S.D.</td>
<td>26.8±6.6</td>
</tr>
<tr>
<td>Mean delay (days) IABP insertion/surgery ± S.D.</td>
<td>12.2±2.5</td>
</tr>
</tbody>
</table>

COPD: chronic obstructive pulmonay disease; CAD: coronary artery disease.
blood cardioplegia. The right atrium was then obliquely incised from the inferior vena cava towards right appendage and stay sutures were used to improve exposure. After placement of self-retaining blades of a Cosgrove retractor, anterior leaflet of tricuspid valve was retracted, and posterior leaflet detached. The edges of VSD were located amid right ventricular trabeculations by means of injections of saline solution through the left ventricular vent. After a moderate debridement of necrotic tissues, closure of septal rupture was commenced by placing interrupted pledgets sutures from the left ventricle through the septum and anchoring a patch. Posterior leaflet was reattached by means of GORE-TEX® stitches. Cardiopulmonary bypass and aortic cross clamp times were respectively 88 min and 64 min. Weaning from CPB was uneventful with low doses of dobutamine. Removal of IABP was implemented 36 h after chest closure. Postoperative echocardiography assessed competency of tricuspid valve with no residual shunt.

3. Results

There was no hospital death. Six beating heart coronary bypasses were performed (1.6 byp/pt). Mean overall CPB and aortic crossclamp times were respectively 86 ± 16 and 36 ± 33 min. Nevertheless, considering the exclusive three cases requiring ventriculotomy and right atriotomy, mean aortic cross clamp time was 59 ± 6 min. Mean time of IABP removal after chest closure was 26.6 ± 17.4 h. No complication related to a prolonged insertion of IABP was reported. The patient with inferobasal shunt had to be reoperated on at the fourth postoperative month, because of a 4 mm residual hemolytic shunt disclosed at the 2nd postoperative month. Surgical repair was achieved through the right atrium, however, with bioprosthetic replacement of tricuspid valve, as a second repair was hazardous. Postoperative outcome was characterized by transitory renal insufficiency and pulmonary complications related to a severe chronic obstructive pulmonary disease. Patients were followed-up for between 13 and 44 months and none of them was lost for follow-up. One patient is in NYHA functional class I and four are in class II.

4. Discussion

Postinfarction VSD is a severe complication estimated to occur in 1 to 3% of acute myocardial infarction [1–4]. Increasing aggressiveness in the diagnosis and primary PTCA may reduce its incidence [8]. Nevertheless, influence of thrombolysis is controversial as it might promote occurrence of VSD [8,9]. Spontaneous prognosis is dismal with a huge mortality within 3 months unless surgical repair is rapidly entertained [1,4,6] and only 7% survival at 1 year [1]. Despite several advances, surgical repair remains a high-risk procedure with a mortality of 10 to 52% [2–6], influenced by preoperative cardiogenic shock, interval between MI and surgery, infarct extension and severity of a concomitant right ventricular infarction [10]. Clinical right ventricular dysfunction is found in up to 95% of patients with an inferior MI [10,11]. Surgeons face to the challenges of assessing the optimal timing of operation, exposing the defect, and choosing the suitable techniques. If ability to moderately delay the surgical step depends on the extent of infarction, however, importance of right ventricular function remains crucial. Therefore, delaying surgery involves to focus on signs of right ventricular failure and if they become patent despite IABP and aggressive medical treatment, then surgery is mandatory. As reported by literature [2–4,6,11] our usual experience with early repair was disappointing. In addition, surgical difficulties were increased by necessity to repair septal wall amid necrotic tissue.

Therefore, attempt for optimizing the hemodynamic profile was preferred instead of immediate surgery [4,6,11,12]. Conversely, this delayed approach necessitates an aggressive medical management with a systematic IABP inserted as early as possible in the course for all patients who develop a postinfarction VSD since improved survival has been observed [12,13]. Early IABP insertion may accelerate resuscitation of stunned and ischemic myocardium by decreasing postload and volume of transventricular shunt, increasing cardiac output, improving coronary perfusion and tolerance of right ventricle with the left-to-right shunt. In our experience, systematic IABP was essential to assign stabilized patients for coronary angiography as three patients presented with cardiogenic shock. Furthermore, delaying surgery between 10 days and 3 weeks facilitated early fibrous healing process and improved solidity of surgical repair.

Furthermore, IABP support enables to evaluate myocardial reserve and consequently, if no hemodynamic stabilization can be set up, emergency surgery is mandatory. Contradictory debates exist about necessity to associate myocardial revascularization at the time of VSD repair, since it may increase operative time [14]. These controversies reinforce the necessity to reduce or suppress cardioplegic-induced ischemia. As previously reported [15], survival after VSD correction is improved by coronary artery bypassing. Clampless CABG performed on-pump prevented from the temptation to achieve incomplete revascularization and the risk to compromise the hemodynamic balance during cardiac mobilization. Additionally, despite a theoretical possibility to repair a distal apical VSD without pump, use of cardiopulmonary support decreases right and left ventricular pressure and cardiac cavity CPB-induced depression allows to visualize the apical septal defect and perform an external exclusion without any mechanical tension. Echographic follow-up of both patients undergoing this specific beating-heart approach demonstrated no significant impact on ventricular geometry as the excluded area was limited and akinetic.

Additional mitral insufficiency is likewise frequently disclosed and if severe, should theoretically be considered for surgical repair. However, severity of mitral regurgitation may be difficult to assess immediately after MI since ventricular shape is modified and remodelling process is known to influence the outcome of ischemic mitral disease. Considering the preoperative status of patients, until recently, surgeons were not particularly aggressive to associate mitral surgery with VSD repair and coronary bypass. Therefore, clampless CABG under CPB may be advisable prior to VSD and mitral repairs.
Repair of posterior VSD through a transinfarct ventriculotomy remains a challenge as cautious considerations are required regarding the access and exposure, the position of the posteromedial papillary muscle and the troublesome sutureline bleeding during closure of a friable ventriculotomy. To provide against these difficulties and avoid additional ventricular damages, exposure of the septum through the right atrium is an attractive option. However, this approach never gained popularity since concerns about exposure have been expressed and consequently, many surgeons still advocate repairing the defect through a ventriculotomy. Furthermore, with the right atrial route, surgeons still advocate repairing the defect through a transatrial approach never gained popularity since concerns about technical support.

Additional coronary bypasses 2 pts
Operative mortality 2
Survival 2
Residual shunt 1

VSD is a relatively uncommon complication and surgical training remains limited with discouraging results. Therefore, in order to optimize hospital results, preoperative medical management as well as surgical procedures have to be accurately discussed to moderately delay surgery. We believe that this strategy emphasized by attempt to reduce or suppress cardioplegic-induced ischemia is advisable as poor results may reflect the critical status of the patient but also an inadequate timing of surgery, usually leading to operate patients immediately after diagnosis of VSD.

This policy significantly changed clinical and surgical features of this postinfarction complication. Over the same period, four patients underwent early surgery with cardioplegic arrest. Table 3 summarizes preoperative characteristics. Despite similar preoperative characteristics, hospital results are disappointing and may reflect the difference of management.

In conclusion, although limited to allow strong legitimate conclusions, this experience confirmed that operative mortality might be directly related to the interval between infarction and surgical repair with a higher mortality if associated with a shorter delay. Reducing or even suppressing the cardioplegic arrest with a beating-heart approach may facilitate achievement of a complete surgical procedure including multivessel revascularization with VSD repair and if necessary mitral surgery.

Acknowledgments

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References