Institutional report - Cardiac general

Iatrogenic type A aortic dissection during cardiac surgery

Ho Young Hwanga, Dong Seop Jeongb, Kyung-Hwan Kimc, Ki-Bong Kimc, Hyuk Ahnc,*

aDepartment of Thoracic and Cardiovascular Surgery, Seoul National University Hospital, Seoul National University College of Medicine, 28 Yeongeon-dong, Jongno-gu, Seoul 110-744, South Korea
bDepartment of Thoracic and Cardiovascular Surgery, Seoul Metropolitan Government Seoul National University Boramae Medical Center, Seoul National University College of Medicine, Seoul, South Korea

cDepartment of Thoracic and Cardiovascular Surgery, Seoul National University Hospital, Seoul National University College of Medicine, Seoul, South Korea

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Abstract

We reviewed our experience of intraoperative type A aortic dissection during cardiovascular surgery. From January 1998 to May 2009, intraoperative aortic dissection occurred in 10 of 3421 cardiac surgical patients (M:F=4:6, 62.4±8.0 years). Preoperative diagnoses were valvular heart disease (n=6), ischemic heart disease (n=2), combined disease (n=1) and aortic aneurysm (n=1). All underwent total circulatory arrest (TCA) with retrograde cerebral perfusion and the torn aorta was replaced (n=8) or repaired (n=2). Iatrogenic type A dissection occurred in 0.29% of patients. It was related with cannulation of ascending aorta (n=4), axillary artery (n=2), aortic root (n=2), and femoral artery (n=1) and aortotomy repair (n=1). Mortality rate was 40% (4/10). After adoption of routine intraoperative transesophageal echocardiography, mortality rate decreased from 75% (3/4) to 17% (1/6) (P=0.190). We initiated TCA before achieving deep hypothermia in three of four non-survivors. There was a trend of increased mortality when the disease extended beyond aortic arch (67%, 4/6 vs. 0%, 0/4; P=0.076). Although intraoperative aortic dissection occurred in <0.3% of our patient population, mortality was high, especially when it extended beyond the arch vessels. Better results were expected when early recognition and proper treatment under deep hypothermic circulatory arrest could be performed.

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1. Introduction

Intraoperative type A aortic dissection is related to the procedures disrupting the wall of the aorta or its branch. It is a very rare but fatal complication. After the first report of successful treatment for the iatrogenic dissection [1, 2], several authors reported the incidence ranged from 0.12 to 0.23% and operative mortality from 24 to 43% [3–7].

In this study, we evaluated 1) the proportion of patients who experienced intraoperative aortic dissection during cardiac surgery and 2) surgical results and associated factors in the treatment of this devastating complication.

2. Materials and methods

2.1. Patients characteristics

From January 1998 to May 2009, 3421 patients including 605 cases of cardiac redo-operation underwent cardiac or aortic surgery under cardiopulmonary bypass (CPB) by cannulating aorta or its branch. Among them, intraoperative aortic dissection occurred in 10 patients (male:female = 4:6, 62.4±8.0 years). The most common co-morbidity was hypertension (n=3) followed by diabetes mellitus (n=2).

Preoperative diagnoses were valvular heart disease (n=6), ischemic heart disease (n=2), combined valvular and ischemic disease (n=1) and aortic aneurysm (n=1) (Table 1). There were two cases of cardiac redo-operation. The study protocol was reviewed by the Institutional Review Board and approved as a minimal risk retrospective study (approval number: H-0911-005-299) that did not require individual consent based on the institutional guidelines for waiving consent.

2.2. Operative techniques

The aortic cannulation technique consisted of double concentric subadventitial pursestring sutures using 2-0 multifilament for ascending aortic cannulation and 4-0 monofilament diamond shaped suture after freeing adventitial fat tissue for cardioplegic root cannulation. Occasionally the aortotomy site was predilatated with a curved dilator when there was some difficulty in inserting arterial cannula. Commercially available 8 mm vascular graft was used as a side graft for axillary cannulation. Femoral cannulation was performed with transverse arteriotomy. Fogarty Hydragrip clamp (V. Mueller, Baxter, Deerfield, IL, USA) was used for aortic cross-clamping in all cases. To prevent aortic injury, bypass flow was lowered to <1 l/min when the ascending aorta was clamped and declamped.
2.3. Treatment of intraoperative dissection

When the aortic dissection was identified by inspection or intraoperative transesophageal echocardiography (TEE), we rapidly changed the arterial cannula to another site, usually the femoral artery. Patients were cooled to achieve total circulatory arrest (TCA) under deep hypothermia. With the retrograde cerebral perfusion (RCP) and transcutaneous monitoring of cerebral oxygen saturation (INVOS cerebral/somatic oximeter, Somanetics Corporation, Troy, MI, USA), the dissected ascending aorta was explored and usually replaced with a vascular graft. When the tear site was clearly identified and dissection did not extend proximally to the coronary artery ostia and it did not interfere with brain perfusion, the tear site was repaired within the lumen using multiple transverse mattress sutures reinforced with Teflon felt.

2.4. Statistical analysis

Statistical analysis was performed with the SPSS software package (version 12.0, SPSS Inc, Chicago, IL, USA). Data are expressed as mean±standard deviation (S.D.) or as proportions. Categorical and continuous variables were analyzed with non-parametric methods like Fisher’s exact test and the Mann–Whitney U-test, respectively. A P<0.05 was considered to be statistically significant.

3. Results

3.1. Preoperative and intraoperative findings

Study patients were older than the other patient population who underwent major cardiovascular surgery during the study period (62.4±8.0 years vs. 53.4±14.8 years, P=0.006). Body surface area (BSA) and body mass index were larger than total patient population (Table 2). In seven patients, the ascending aortic diameter was measured with preoperative computed tomography (CT). All were >30 mm (31 mm–55 mm). The aortic tear was related with cannulation of the ascending aorta (n=4), axillary artery (n=2), aortic root (n=2), and femoral artery (n=1) and aortotomy repair (n=1) (Table 3). The dissection was extended proximally to the coronary artery ostia level in two patients, and distally over the aortic arch in six patients. Retrograde dissection occurred due to femoral arterial wall disruption during direct cannulation of the femoral artery in one case. Although we used a side graft in the axillary cannulation, aortic dissection occurred in two cases. In one case, TEE before conducting CPB revealed atheromatous plaque at the bifurcation site between the right carotid artery and the right subclavian artery and this might have lead to an intimal tear after initiating CPB. However, the cause of the aortic dissection was unclear in the other case. Eight patients underwent ascending aorta replacement including the tear site with a commercially available vascular graft. The tear site was repaired in two patients. All procedures were performed under TCA with RCP. In one patient who was diagnosed as an aortic arch aneurysm and underwent a total arch replacement under axillary cannulation, TCA was performed with RCP initially, because the dissection occurred as the result of an axillary artery cannulation. After incising the aortic arch, the innominate artery and the right common carotid artery were cannulated directly and antegrade cerebral perfusion was started. TCA time was 48±23 min and rectal temperature during TCA ranged from 18 to 32 °C.

3.2. Early results

Iatrogenic type A aortic dissection occurred in 0.29% of patients. It was similar between primary operation (0.28%, 8/2816) and cardiac redo-surgery (0.33%, 2/605) (P=0.693). Operative mortality was 40% (4/10). Three patients died due to intractable metabolic acidosis. One patient died of severe hypoxic brain damage. TCA was initiated before achieving deep hypothermia in three of four non-surviving patients. In one patient, aortic tear site bleeding was uncontrollable even after changing aortic inflow site. We should initiate circulatory arrest under moderate hypothermia. In another patient, we assumed that primary repair was possible and started TCA under moderate hypothermia. However, because the tear was more extensive than expected, we cooled the patient further after closing...
the aortotomy, and replaced the ascending aorta. In the other patient who underwent total arch replacement with auxiliary cannulation as described above, immediate brain perfusion was needed because sustained brain hypoperfusion was suspected due to arch vessel dissection. We incised the aortic arch with TCA and RCP under mild hypothermia. Then, the arch vessels were directly perfused, distal anastomosis was performed and the distal body was perfused. In this patient, hypoxic brain damage occurred due to cerebral malperfusion before identifying the occurrence of iatrogenic dissection. Deep hypothermic TCA was achieved in all of the survivors. There was a trend of increased mortality when the dissection extended to the arch level (67%, 4/6 vs. 0%, 0/4; \( P = 0.076 \)). Mortality rate has decreased recently, although it is not statistically significant (\( \leq 2002 \text{ vs.} \geq 2003 = 75\%, \ 3/4 \text{ vs.} \ 17\%, \ 1/6, \ P = 0.190 \)). Other variables including age, sex and coronary artery ostial involvement were not related with early mortality (Table 4).

Pathological findings were available in five recent patients among eight patients who underwent graft replacement of a torn aorta. Myxoid degeneration was found in all patients. Dystrophic calcification was found in three patients. Diffuse atherosclerotic change of the ascending aorta and aortic arch was found in the patient who underwent a total arch replacement due to aortic dissection which occurred after axillary cannulation with no definite reason.

Compartment syndrome occurred in one patient at the ipsilateral lower leg where the femoral arterial cannula was inserted.

3.3. Late results

Six survivors have been followed-up for 51 (7–119) months. A CT-angiogram was performed in four (66%) patients. Two patients who underwent a primary tear site repair had no residual aortic lesion on the CT-angiogram performed at one and nine years after surgery. Residual aortic dissection remained in two patients three years after surgery without a change in diameter. Late death occurred in one patient. This patient underwent a mitral valve replacement and aortic tear site repair and died suddenly, 46 months after surgery.

4. Discussion

This study demonstrated two main findings. First, over a period of 11 years, intraoperative type A aortic dissection occurred in 0.29% of the patients who underwent cardiovascular surgery using CPB with cannulation of the aorta or its branch. Second, mortality was predicted when we failed to achieve a deep hypothermic TCA or when the arch vessels were involved.

Intraoperative type A aortic dissection is related to the procedures disrupting the wall of the aorta or its branch. It is a very rare but fatal complication. Several authors reported that the incidence ranged from 0.12 to 0.23% and operative mortality ranged from 24 to 43% [3–7]. In our cases, the proportion of intraoperative aortic dissection was 0.29%. It was comparable with previous reports. Reported causes of aortic dissection were multifactorial and included ascending aortic cannulation, antegrade cardioplegic cannulation, axillary artery cannulation, partial or cross-clamping of the ascending aorta, aortotomy for proximal anastomosis in coronary artery bypass grafting (CABG) surgery and injury from bypass flow on aortic arch or descending thoracic aorta [2, 4, 6, 8–11]. In the present study, most of the dissection was associated with cannulation of the ascending aorta (n = 4), its branch (n = 3) and aortic root (n = 2). In one patient, it occurred after aortotomy repair. Previous reports demonstrated that aortic dissection occurred or was detected at the postoperative period [4, 12]. However, we have no experience of postoperative type A aortic dissection. In the present study, the mortality rate was 40%. Three patients died of multi-organ failure with intractable acidosis. One patient died due to hypoxic brain damage. Achievement of deep hypothermic TCA and the extent of aortic dissection were related with operative mortality. The extension of dissection beyond the aortic arch vessels might reflect decreased cerebral blood flow during CPB and recognition of dissection late after its occurrence. Early mortality rate was 17% (1/6) in the latter study period (\( \geq 2003 \)). It was lower than that of early study period (75%, 3/4 patients), although it was not statistically significant. This might be due to the routine application of intraoperative TEE in the latter period. Intraoperative evaluation of the cardiac and aortic status was performed after inducing general anesthesia. It was repeated after beginning CPB, during CPB and early after discontinuation of CPB. This might lead to early recognition and prompt management of intraoperative aortic dissection.

Atherosclerosis is a known predisposing factor for the occurrence of aortic dissection [5]. However, it is difficult to find any significant predictors for the occurrence of intraoperative aortic dissection, because the incidence is very low. Kodolitsch et al. [13] found that dilated ascending aorta, known atherosclerosis, previous CABG surgery, older age and blood pressure at the time of dissection were risk factors for iatrogenic dissection. Still and his colleagues [5] suggested that angina, left ventricular dysfunction, hypertension, previous myocardial infarction and intra-aortic balloon counterpulsation were related to intraoperative aortic dissection. In the present study, patients who experienced intraoperative aortic dissection were older than total patient population during study period. In addition, 70% (7/10) of the study patients underwent preoperative CT and all had dilated ascending aorta with atherosclerosis.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-survivors</th>
<th>Survivors</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>63.5 ± 5.8</td>
<td>61.7 ± 9.7</td>
<td>1.000</td>
</tr>
<tr>
<td>Female sex</td>
<td>2 (50%)</td>
<td>4 (66%)</td>
<td>1.000</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.9 ± 0.15</td>
<td>1.9 ± 0.31</td>
<td>0.929</td>
</tr>
<tr>
<td>TCA under deep hypothermia</td>
<td>1 (25%)</td>
<td>6 (100%)</td>
<td>0.033</td>
</tr>
<tr>
<td>Aortic arch involvement</td>
<td>4 (100%)</td>
<td>2 (33%)</td>
<td>0.076</td>
</tr>
<tr>
<td>Coronary artery involvement</td>
<td>2 (50%)</td>
<td>0 (0%)</td>
<td>0.133</td>
</tr>
<tr>
<td>Primary repair of tear site</td>
<td>0 (0%)</td>
<td>2 (33%)</td>
<td>1.000</td>
</tr>
<tr>
<td>Year of operation (before 2003)</td>
<td>3 (75%)</td>
<td>1 (17%)</td>
<td>0.190</td>
</tr>
</tbody>
</table>

BSA, body surface area; TCA, total circulatory arrest.
However, we could not perform multivariate analysis, because data were incomplete for total patient population.

Ruchat et al. [4] demonstrated that two of seven patients with intraoperative dissection died due to hypoxic brain injury. Although no statistical analysis was performed in their study, they concluded that cerebral perfusion defines the outcome. In our non-parametric analysis, variables including type of the repair, coronary artery involvement and age were not related with early death. Fleck and his colleagues [6] showed that graft interposition should be a treatment of choice because the dissection was not localized to a small part of the aorta. We could not find any evidence, however, that graft replacement yielded a better outcome than primary repair including follow-up results when it was applied in selected patients according to the extent of the disease and site of the tear.

There are limitations to the present study that must be recognized. First, the present study was not performed in a prospective manner. Second, because of the limited number of patients, it was difficult to make definite conclusions. Although non-parametric analysis was performed, it was hard to find statistical significance due to the small sample size.

In conclusion, intraoperative type A aortic dissection occurred rarely, but the results were fatal. Care should be taken especially in old patients with dilated, atherosclerotic aorta to avoid this complication. Routine application of TEE might be helpful in identifying iatrogenic aortic dissection and better results might be expected when early recognition and proper treatment under deep hypothermic circulatory arrest could be performed.

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References