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Clinical research

Early and late outcomes of acute type A aortic dissection: analysis of risk factors in 487 consecutive patients

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KEYWORDS
Aortic dissection;
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Surgery

Aims The purpose of this retrospective study was to assess the risk factors for the early and late outcome of the surgical treatment of acute type A aortic dissection, in terms of mortality and morbidity.
Methods and results From 1976 to 2003, 487 patients with acute type A aortic dissection treated surgically were enrolled. Twenty-five pre-operative and intra-operative variables were analysed to identify conditions influencing early and late morbidity and early mortality. The in-hospital mortality rate including operative death was 22% (107 patients). Multivariable analysis indicated that pre-existing cardiac disease (RR = 3.7, 95% CI = 1.8–7.4) and cardiopulmonary resuscitation (RR = 6.8, 95% CI = 2.3–20.2) were independent predictors of in-hospital death. The causes of in-hospital mortality were low cardiac output in 32 patients (6.6%), major brain damage in 24 patients (5.9%), haemorrhage in 11 patients (2.2%), sepsis in nine patients (1.8%), visceral ischaemia in eight patients (1.6%), multiple organ failure in seven patients (1.4%), rupture of the thoracic aorta in six patients (1.2%), respiratory failure in six patients (1.2%), and four intra-operative deaths. The follow-up was 100% complete. The actuarial survival was 94.9 $\pm$ 1.2% and 88.1 $\pm$ 2.6%, at 5 and 10 years, respectively.
Conclusions Patients' pre-operative co-morbidities and dissection-related complications significantly affect early and late survival and morbidity after surgical treatment of acute type A aortic dissection.

Introduction

Acute Stanford type A aortic dissection is a condition needing emergency operation to prevent sudden death. Although in the last decade early referral of the patient for surgery, improved surgical experience, and intra-operative management have contributed to a better operative outcome of this lethal aortic disease, it continues to be associated with severe post-operative complications and a high mortality rate. Surgical mortality rates have been estimated to range from 9 to 36% with a 5 year survival rate of 50–80%.[1–6]

In this study, we retrospectively reviewed 487 consecutive patients in order to identify possible pre- and/or

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intra-operative variables influencing early and late mortality as well as morbidity after emergent repair of acute type A aortic dissection.

Methods

A total of 487 consecutive patients with acute type A aortic dissection were treated surgically at the department of cardiothoracic surgery of the St Antonius Hospital ($n = 315$), Nieuwegein, The Netherlands and the department of cardiovascular surgery of the University of Bologna ($n = 172$), Italy, over three decades (1976–2003). Type A aortic dissection was defined, according to the Stanford University classification, as involving the ascending aorta and/or aortic arch, progressing distally towards the descending thoracic aorta, differing from type B, involving only the descending thoracic aorta. Aortic dissection was defined as acute if chest pain or other related symptoms were present less than 14 days before operation.[7] All subacute (between 2 and 6 weeks) and chronic (>6 weeks) dissections were excluded.

Dissection involving the ascending aorta was classified Stanford type A according to previously published criteria.[8] *Table 1* summarizes the demographic and clinical characteristics of the patients. Hypertension was defined as diastolic pressure >90 mmHg and systolic >160 mmHg. Diabetes was type II under medical treatment (oral drugs or insulin). Hypercholesterolaemia was defined when the blood value was >200 mg/dL. Previous cardiac operations were aortic valve replacement and coronary artery bypass surgery. Pre-existing cardiac disease was any valve disease or angina. The most common presenting symptom was acute and severe anterior chest or back pain. The diagnosis was confirmed in the early experience with aortography (10.5%, $n = 51$), currently with computed tomography (45.2%, $n = 220$), magnetic resonance imaging (6.8%, $n = 33$), and transoesophageal echocardiography (TEE) (71.5%, $n = 348$), or a combination. TEE is our current diagnostic method of choice. All patient data were obtained by retrospective review of hospital records and follow-up via written or telephone contact, or both.

In hospital, mortality was defined as death occurring within 30 days of the operation or during the initial hospital stay. No patient was lost to follow-up during this period.

Table 1 Demographic and clinical characteristics of the population

Patients (n)	487
Age (years)	
Mean ± SD	58.34 ± 12.5
Range	17–82
Gender	
Male	322 (66.1)
Female	165 (33.9)
Hypertension	259 (53.2)
Diabetes	14 (2.9)
Hypercholesterolaemia	51 (10.5)
Marfan syndrome	32 (6.6)
Chronic obstructive pulmonary disease	38 (7.8)
Aortic valve regurgitation	309 (63.4)
LVEF <50	24 (4.9)
Cardiac tamponade	100 (20.5)
Mesenteric ischaemia	4 (0.8)
Shock	50 (10.3)
AMI	20 (4.1)
Previous cardiac operation	43 (8.9)
Cardiopulmonary resuscitation	19 (3.9)
Rupture of ascending aorta	25 (5.1)
Primary tear	
Proximal ascending aorta	324 (66.5)
Middle ascending aorta	66 (13.6)
Distal ascending aorta	32 (6.6)
Aortic arch	51 (10.5)
Proximal descending aorta	14 (2.9)
Bicuspid aortic valve	18 (3.7)
Focal neurological damage	41 (8.4)
Renal failure	6 (1.2)

LVEF, left ventricular ejection fraction. Data are n (%) unless otherwise stated.

Operative technique

During these 27 years, different surgical techniques were used; however, the following reflects our general operative approach. Induction of anaesthesia was obtained with propofol 2 mg/kg, fentanyl 2 μg/kg, and pancuronium 0.1 mg/kg. Anaesthesia was maintained with propofol and fentanyl. For all patients, pH balance control was carried out using the α-stat method.

After confirmation of diagnosis by TEE, patients routinely underwent median sternotomy, cannulation of the femoral artery for the arterial line of the cardiopulmonary machine, providing oxygenated blood to the patient and the right atrium, for the venous, non-oxygenated blood return from the patient to the pump. The left ventricle was vented through the right superior pulmonary vein to prevent the ventricular distension. Myocardial protection was achieved with cold crystalloid cardioplegia and continuous topical hypothermia. Total cardiopulmonary bypass (CPB) was conducted using moderate systemic hypothermia. Temperature was continuously measured in the nasopharynx, oesophagus, and rectum. The distal ascending aorta was cross-clamped at 25–28°C or earlier in most cases ($n = 443$) following induction of ventricular fibrillation and the proximal aorta was opened to locate the intimal tear and then reconstructed.

When the intimal tear extended distally, the quality of the aorta at the site of distal anastomosis was unfavourable or the surgeon found it more convenient to perform an open distal anastomosis, the patient was further cooled down, while performing the proximal aortic reconstruction. During the deep hypothermic circulatory arrest (DHCA), which was used in almost 40%, the arch was inspected to check the presence of an intimal tear and the arch was totally or partially replaced, in the latter as a hemiarch procedure. If the arch appeared normal at the inspection without any lucid entry tear, it was considered unnecessary to replace the aortic arch and an open distal aorto-prosthesis anastomosis was performed at the base of the innominate artery, after which cardiopulmonary bypass was re-instituted with antegrade aortic perfusion.

Since 1990, antegrade selective cerebral perfusion (ASCP) has progressively been introduced in our institutions.[9]

In effect, we first cool the patient down to a nasopharyngeal temperature of 25°C at which the CPB is discontinued and the ascending aorta is opened. Under visual control and with the patient in the Trendelenburg position, the cannulae for bilateral ASCP are inserted into the innominate and left common carotid artery. In addition, the left subclavian artery is either clamped or occluded with a Fogarthy catheter. Subsequently, we protect the heart by cold crystalloid antegrade cardio-

plegia through both coronary ostia. Nasopharyngeal and blood temperatures are kept at 25°C, blood is perfused into both carotid arteries at a rate of 10 mL/kg/min, and cerebral perfusion pressure is adjusted to maintain a right radial artery pressure of 40–70 mmHg. The eventual arch replacement and distal aorto-prosthesis anastomosis are performed under total circulatory arrest. Finally, when this procedure is completed, ASCP is stopped and antegrade perfusion is re-instituted though the prosthesis. Re-warming is achieved at 1°C per 3 min, not exceeding a 10°C gradient between blood temperature and nasopharyngeal or rectal temperature. The proximal anastomosis was performed during re-warming. Attempts were made to preserve the aortic valve by re-suspension of the commissures if the dissecting process had caused regurgitation, but it was always replaced in cases of annulo-aortic ectasia or if the cusps showed a severe morphological alteration. *Table 2* shows the operative data. *Figures 1–4* show the surgical techniques.

Table 2 Operative data

Arterial cannulation	
Femoral artery	478 (98.2)
Ascending aorta	3 (0.6)
Aortic arch	2 (0.4)
Axillary artery	4 (0.8)
Venous cannulation	
Right atrium	428 (88.1)
Bicaval	35 (7.2)
Femoral vein	23 (4.7)
Perfusion techniques	
ECC	110 (22.7)
ECC + DHCA	191 (39.4)
ECC + DHCA + ASCP	75 (15.5)
ECC + ASCP	87 (17.9)
ECC + DHCA + RSCP	22 (4.5)
Operative procedure	
Ascending aorta replacement	254 (52.7)
Ascending aorta and hemiarch replacement	78 (16.1)
Ascending aorta and arch replacement	26 (5.4)
Ascending aorta, arch and ET	7 (1.4)
Bentall	54 (11.2)
Bentall + hemiarch	17 (3.5)
Bentall + arch	3 (0.6)
Bentall + arch + ET	2 (0.4)
AVR + ascending aorta replacement	22 (4.5)
AVR + ascending aorta replacement + hemiarch	3 (0.6)
AVR + ascending aorta replacement + arch	2 (0.4)
End-to-end anastomosis	15 (3.1)
CPB time	
Mean	185.3 ± 61.2
Range	57–523
Cross-clamp time	
Mean	108.9 ± 44.9
Range	20–324
ASCP time	
Mean	56.1 ± 36.8
Range	19–315

AVR, aortic valve replacement; ECC, extracorporeal circulation; ET, elephant trunk; RSCP, retrograde selective cerebral perfusion. Data are *n* (%) unless otherwise stated.

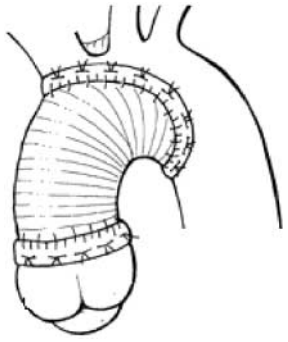

Figure 1 The ascending aorta and hemi-arch replacement.

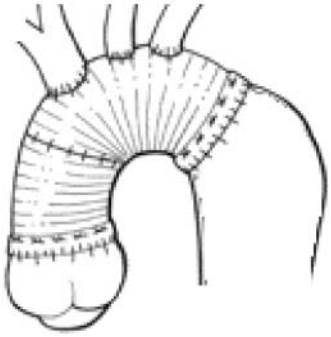

Figure 2 The total arch replacement.

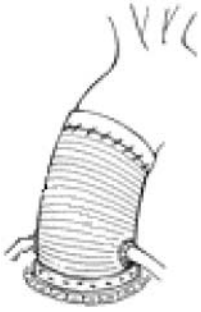

Figure 3 The aortic valve and ascending aorta replacement according to the Bentall procedure with re-implantation of the coronary ostia.

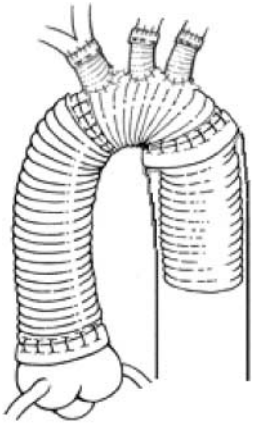

Figure 4 The ascending aorta and arch replacement associated with the elephant trunk in the proximal descending aorta.

Statistical analysis

Statistical analysis was performed using SPSS 11.5 statistical software (SPSS Inc, Chicago, IL, USA) and STATA 7.0 statistical package (Stata Corporation, College Station, TX, USA).

The continuous data in this study are expressed as the mean $\pm$ standard deviation and categorical data as percentages. The unpaired Student's *t*-test and χ^2-test or Fisher's exact test were used for continuous and categorical variables, respectively (two-sided tests). A *P*-value <0.05 was considered statistically significant. The risk of type I error was 0.05 therefore for each individual comparison. We used a split-sample approach: we split data randomly into two data sets, and then an independent analysis was conducted within each data set in order to obtain the results. In order to assess the predictive risk factors for mortality and morbidity, a Cox proportional hazards analysis was performed, using the forward stepwise procedure. Each variable that was considered significant at univariate analysis was selected for multivariable analysis (method: likelihood ratio). The variables were added one at time to the model if they met the selection criterion based on the *P*-value for the score statistic. The default value for inclusion was 0.05. The default value for removal from the model was a *P*-value of 0.10 for the maximum partial likelihood estimates criterion. The proportional hazard assumptions of the model were assessed by plotting the scaled Schoenfeld residuals against time to provide relatively precise information about the time dependence of the covariate effects. This graphical procedure was performed for each selected variable in the models. In order to verify the accuracy of the models, Cox–Snell residuals have been calculated: the plots of cumulative hazard function (starting from the Kaplan–Meier survival estimation of Cox–Snell residuals as survival time) on Cox–Snell residuals for two obtained models are reported (*Figures 5 and 6*). Kaplan–Meier cumulative survival plots were constructed for display. A binary variable was also created in order to distinguish the surgical treatments of the patients before and after 1990, when we introduced the use of ASCP; another binary variable was created to specify each department where the patients were operated on.

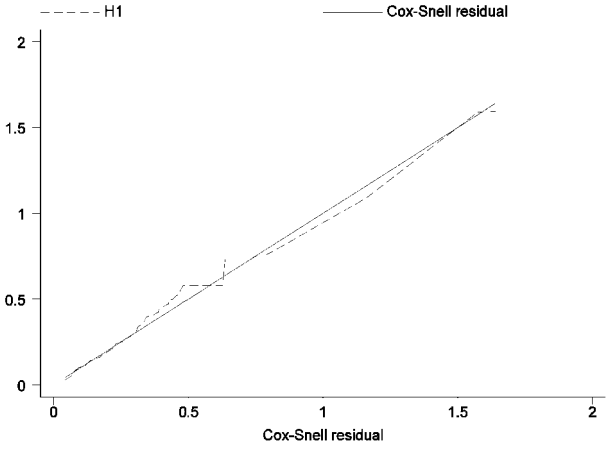

Figure 5 The plot of cumulative hazard function on Cox–Snell residuals (early mortality).

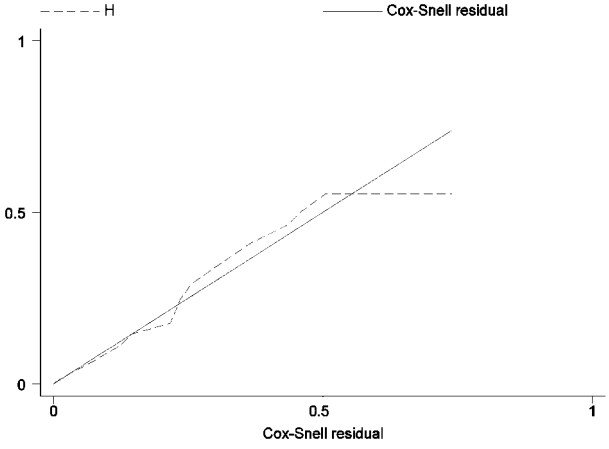

Figure 6 The plot of cumulative hazard function on Cox–Snell residuals (late mortality).

Results

In-hospital mortality

The in-hospital mortality rate, including operative deaths, was 22% (107/487 patients). Four patients could not be weaned from CPB and they died in the operating room. The causes of in-hospital mortality were low cardiac output in 32 patients (6.6%), major brain damage in 24 patients (5.9%), haemorrhage in 11 patients (2.2%), sepsis in nine patients (1.8%), visceral ischaemia in eight patients (1.6%), multiple organ failure (MOF) in seven patients (1.4%), rupture of the thoracic aorta in six patients (1.2%), and respiratory failure in six patients (1.2%).

Table 3 shows the results of univariate analysis of 14 pre-operative and intra-operative risk factors.

Multivariable analysis identified only two variables as independent predictors of in-hospital death: pre-existing cardiac disease ($P < 0.001$) and cardiopulmonary resuscitation ($P = 0.001$).

Table 3 Statistically significant risk factors for in-hospital mortality (univariate analysis)

Risk factors	*P*-value	HR	95% CI
Pre-operative central neurologic deficit	<0.001	2.71	1.66–4.43
Pre-operative anuria	<0.001	1.62	1.37–5.02
Pre-operative shock	<0.001	1.42	1.28–1.62
Cardiopulmonary resuscitation	<0.001	2.17	1.43–3.29
Cardiac tamponade	0.001	2.38	1.15–3.87
Pre-operative creatinine (>1.2 mg/dL)	0.001	1.08	1.03–1.26
ECC time (>180 min)	0.002	1.22	1.08–4.12
Aortic valve regurgitation	0.008	2.05	1.19–3.65
LVEF <50%	0.014	1.97	1.12–3.47
Hypercholesterolaemia	0.022	1.14	1.08–3.75
Pre-existing cardiac disease	0.028	1.94	1.21–4.90
Visceral ischaemia	0.031	3.85	1.12–13.71
Chronic obstructive pulmonary disease	0.038	1.18	1.02–3.11
Arch involvement	0.043	1.02	1.01–1.06

Late survival

The temporal interval of the study was 27 years; during this period the 380 patients who survived the operation and were discharged from the hospital were followed up. *Figure 7* shows the actuarial survival curve estimated by the Kaplan–Meier method. The actuarial survival rate of these patients was $94.9 \pm 1.2\%$ and $88.1 \pm 2.6\%$, at 5 and 10 years after the operation, respectively. Thirty-one patients died during this period. The causes of death were stroke in 12 patients, acute myocardial infarction in four patients, heart failure in three patients, and malignancy in five patients. Seven patients died during aortic re-operation: they underwent re-operation because of re-dissection or progressive dilation of the false lumen of the aorta. Pre-operative type II diabetes was a statistically significant predictor of late death at follow-up ($P = 0.008$).

Figure 8 shows the freedom from re-operation curve: at follow-up, freedom from re-operation was $92.2 \pm 2.8\%$ at 25 years.

Early morbidity

We considered the events occurring within the 30 days following surgery as 'early'. The mean ICU stay was 48 ± 12 h with no statistically significant difference according to the time periods. The mean length of stay was 8 ± 3 days. Post-operative complications included re-operation for bleeding in 115 patients (23.8%), respiratory failure requiring prolonged mechanical ventilation, defined as more than 72 h and/or tracheostomy in 31 patients (6.4%), renal failure with oliguria in 15 patients (3.1%), a major neurological injury (stroke) in 88 patients (18.2%), the mediastinitis in seven patients (1.5%). Multivariable analysis revealed that the primary tear in the proximal ascending aorta significantly predicted a prolonged post-operative stay in the ICU (>48 h) (RR = 1.41; 95% CI = 1.02; 1.96); a pre-operative cardiac disease was a significant risk factor for post-operative tracheotomy (RR = 4.3; 95% CI = 1.9; 9.7). *Tables 4 and 5* describe the risk factors for post-operative re-operation for bleeding and for major central neurological injury, respectively.

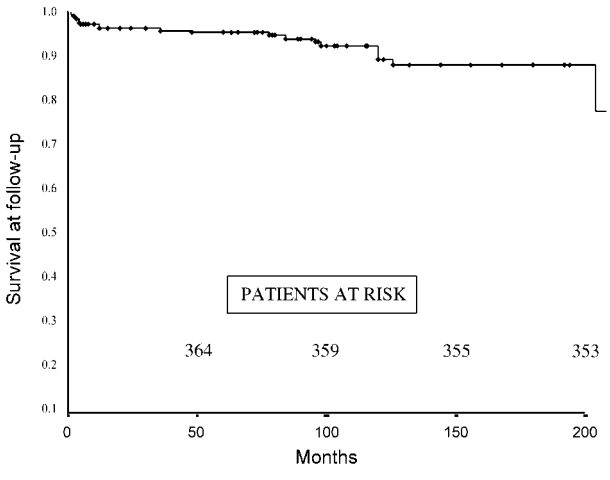

Figure 7 Kaplan–Meier curve for long-term survival at follow-up, among hospital survivors.

Morbidity at follow-up

We also performed a multivariable analysis in order to estimate the weight of pre-operative, intra-operative and post-operative variables in the late onset of the same complications. Severe post-operative bleeding was a risk factor for onset of a stroke at follow-up

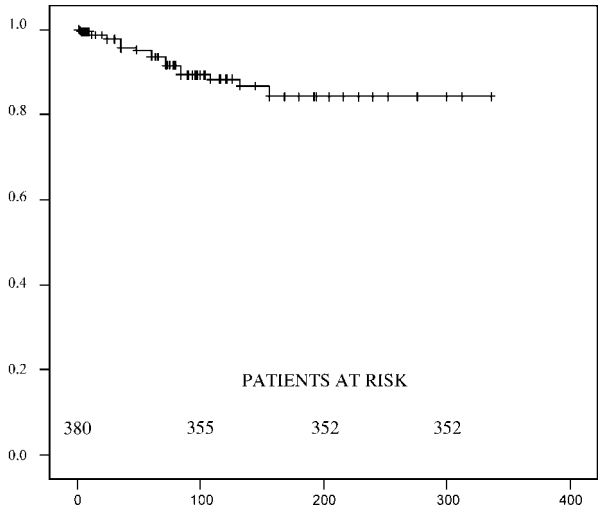

Figure 8 Freedom from re-operation curve at follow-up, among hospital survivors.

Table 4 Cox proportional hazards analysis of re-operation for bleeding (multivariable analysis)

Risk factors	*P*-value	RR	95% CI
Aortic arch	<0.001	1.4	1.2–1.6
Cardiac tamponade	<0.001	3.9	2.3–6.7
Age <70 years	0.01	1.9	1.14–3.4
Pre-existing cardiac disease	0.01	2.3	1.2–4.4
Cardiopulmonary resuscitation	0.028	4.6	1.2–18.5

Table 5 Cox proportional hazards analysis of major central neurological injury (multivariable analysis)

Risk factors	*P*-value	RR	95% CI
Pre-existing cardiac disease	0.01	2.2	1.2–4.2
Pre-operative transient central neurological deficit	<0.001	5.6	2.7–11.5
Aortic valve regurgitation	0.004	0.7	0.6–0.9
Perfusion technique (DHCA)	0.005	0.7	0.6–0.9

(RR = 4.2; 95% CI = 1.3–13.5). The pre-operative oliguria significantly increased the risk to need dialysis in the post-operative period (RR = 6.2; 95% CI = 1.3; 28.7). Fourteen patients experienced stroke, acute myocardial infarction occurred in six patients, four patients developed heart failure; statistical analysis did not show any significant predictor.

Discussion

Although hospital mortality for emergency repair of acute type A aortic dissection has decreased over the last decade,[10,11] a great disparity in the operative mortality rates still exists among the recently reported series, ranging between 9 and 36%.[1,2]

The number of high risk patients operated on might explain the differences reported in mortality rates among institutions, even if a rather standard surgical approach is usually applied.

We retrospectively analysed our 487 patients operated on in two departments, sharing the same surgical strategies, in order to evaluate the predictive risk factors influencing current surgical outcome, early and late after operation.

Although the surgical treatment of patients with this emergency situation has evolved in the past decades with excellent results,[12,13] in this study population we could not find the operative date as a significant risk factor for operative death as well as the centre where the patients were operated on. This means that, although the treatment of these patients clearly evolved with time, improving surgical expertise and operating times, mortality remained comparatively stable throughout the entire 27 year period, even if the pre-operative haemodynamic conditions of the most recently operated on patients (after 1990: 280/487) were a little more unstable: among the latter, in effect 30 patients had a re-operation, 15 patients underwent cardiopulmonary resuscitation before surgery, 20 patients had a LVEF $<50\%$, and 70 patients presented a cardiac tamponade, pre-operatively.

In the present study, the hospital mortality rate (22%) and the 5-year survival ($88.1 \pm 2.6\%$) match statistics reported previously.[1,2]

Previous reports have indicated that pre-operative myocardial ischaemia,[14] pre-operative resuscitation,[15] peripheral ischaemia,[15,16] visceral ischaemia,[15–17] haemopericardium,[4,15,16] renal failure,[4,12,15] older age,[4,5,15,16,18] pre-operative haemodynamic instability,[5,14,18] and coma,[5,14] are risk factors for hospital death.

The current study demonstrated a pre-existing cardiac disease ($P = 0.000$) and pre-operative resuscitation ($P = 0.001$) to be the leading predictors of hospital death: the same was reported by the International Registry of Acute Aortic Dissection (IRAD), in which 'hypotension/shock/tamponade' were demonstrated to be the independent predictors of mortality, that is 26% after surgical therapy compared with 58% for medical management alone.[19]

These findings also add evidence to pre-operative haemodynamic stability being the key predictor of operative success, emphasizing the need for urgent surgical repair in patients with acute type A aortic dissection before the onset of haemodynamic instability. With advances in diagnostic methods and quicker referral to cardiothoracic centres with a wide experience in aortic surgery, it should be possible to avoid delay and situations that need cardiopulmonary resuscitation.

Contrary to previously reported findings,[4,5,17,18] older age (>70 years) was itself not a risk factor for early death and any post-operative complication: the in-hospital mortality rates were 25% (23/92) for patients aged ≥ 70 years and 21.3% (84/395) for patients aged <70 years, without any statistically significant difference ($P = 0.25$). Because older patients are likely to have a decreased life expectancy, the influence of age on medium-term survival may be partially explained as a reflection of the natural life expectancy. On the other hand we noticed a statistically significant difference ($P = 0.005$) in terms of survival at follow-up between the two groups, obviously due to the shorter life expectancy of the oldest patients. Although aortic arch repair increased the risk of operative mortality in previous reports,[3,12,20,21] there was no significant difference ($P = 0.4$) in mortality between patients with (8.2%) or without (91.8%) total arch replacement in our series. Our current indications for the total arch replacement are the same as those suggested by Dr Kazui: (i) acute aortic arch dissection with intimal tear in the aortic arch; (ii) acute aortic dissection with the intimal tear in the descending aorta; (iii) rupture or massive false lumen of the aortic arch; (iv) compromise of the arch vessels; (v) co-existing aortic arch aneurysm; and (vi) young Marfan's patients without serious pre-operative complications such as shock or renal/visceral ischaemia.[22]

In our analysis, we also considered potential risk factors for post-operative morbidity. A primary tear located in the ascending aorta was found to be a predictor of prolonged (>48 h) post-operative ICU stay ($P = 0.03$), while pre-operative cardiac disease was a risk factor for post-operative respiratory failure and tracheotomy ($P < 0.001$). Furthermore, pre-operative cardiac disease ($P < 0.001$), cardiac tamponade ($P < 0.001$), aortic arch dissection ($P < 0.001$), age <70 years ($P = 0.01$), and cardiopulmonary resuscitation ($P = 0.028$) were predictors of re-operation for bleeding.

Another issue involving the acute repair of the dissected aorta is preventing the need for re-operation in the future, as this is usually also associated with a higher mortality, due to the progression of the disease because of the pathophysiology of the weakened aortic tissue.

The re-operation-free survival at follow-up was $92.2 \pm 2.8\%$ at 25 years, which is much more than the 60–80% reported in previous studies.[3,23–25]

We believe that these results are due to our aggressive surgical management of aortic repair: the intimal tear should always be resected and if it is not found immediately after the opening of the dissected aorta, one should look for it in the intima.

In our current operative strategy we always try to identify the intimal tear. Therefore inspection of the distal ascending aorta or the aortic arch should follow with the use of DHCA; if the aortic valve is not morphologically normal or if the annulus is dilated, a Bentall procedure might be recommended. If an aortic clamp is applied, the clamping area should always be excised and the ascending aorta should be replaced.

In conclusion, our experience indicates that the patients with an acute type A aortic dissection should have early diagnosis and repair in order to avoid pre-operative situations such as cardiogenic shock and the need for cardiopulmonary resuscitation to improve survival. Furthermore, age, in our study, does not contraindicate surgery for acute type A aortic dissection.

Appendix

Pre- and peri-operative variables assessed for hospital and late mortality

Age, gender, hypertension, diabetes, hypercholesterolaemia, Marfan syndrome, chronic obstructive pulmonary disease, aortic valve regurgitation, LVEF, cardiac tamponade, mesenteric ischaemia, shock, AMI, previous cardiac disease, cardiopulmonary resuscitation, site of primary tear, extension of dissection, bicuspid aortic valve, surgery before 1990, pre-operative neurological deficit, perfusion techniques, operative procedures, CPB time, cross-clamp time, ASCP time and major neurological injury.

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