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Fate of False Lumen after Repair of Acute Type A Aortic Dissection

Thesis

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ABSTRACT

OBJECTIVE: The aim of the study was to investigate the impact of ascending aortic repair of type A acute aortic dissection (TAAAD) on the obliteration of remaining false lumen of the descending aorta and detect the state of thoracic aorta wall distal to surgical repair in early ,mid and long term postoperative period.

METHODS: Between January 2000 and December 2012 , a total of 100 patients who underwent TAAAD surgery were evaluated by contrast-enhanced computed tomography (CT) patency and width of false lumen and maximal diameter of the aorta were compared between early and last follow-up images. Changes were analyzed separately in the thoracic segments and factors influencing faster growth were analyzed. Distal reoperations and patient survival were examined. Follow-up time averaged 5 years.

RESULTS: The false lumen was patent in 69 patients (70%). In 41 patients (41%), the descending aorta dilated by 1 cm or more. Dilatation occurred more frequently in the thoracic aorta and in patients with patent or wide false lumens, larger aortic diameter, Marfan syndrome, younger age, and male sex. Meanwhile, shrinkage of thoracic false lumen occurred in 31 patients(30%). Such shrinkage occurred in 18 of 19patients (95%) who had thrombosed and narrow false lumens in the thoracic aorta. The patients who did not require reoperation at 1, 5 and 10 years of follow-up were , 98%, 96% and 84%respectivly. Overall survival estimates after surgery (in-hospital mortality excluded) were 96% ± 1.3% at 1 year, 90.1± % ±2.2% at 5 years, and 80.0%±2.0% at 10 years

CONCLUSION: Despite its seriousness, surgery for acute aortic dissection produces good early and long term results. we confirmed the high incidence of descending aortic dilatation after repair of TAAAD. However, we also found that the descending aortic lesion changed favourably in some patients. Early postoperative characteristics of the false lumen were helpful for predicting both favourable and unfavourable changes in thoracic aorta distal to TAAAD repair during later follow-up e.g. dilation and regression. Our data show not only a high incidence of descending aortic dilatation after repair of TAAAD, but also shrinkage of thoracic false lumen in some patients. Our results can be used as a control data for determining the benefit and indication of a more extensive or newer approach such as frozen elephant trunk.

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List of Abbreviations

TAAAD	:	Type A acute aortic dissection
AD	:	Aortic dissection
BAV	:	Bicuspid aortic valve
CT	:	Computed tomography
CXR	:	Chest X ray
ECG	:	Electrocardiography
EDS	:	Ehler-Danlos syndrome
ED	:	Emergency department
FBN-1	:	Fibrillin-1
IMH	:	Intramural hematoma
IRAD	:	The International Registry of Acute Aortic Dissections
IV	:	Intravenous
LCCA	:	Left common carotid artery
LSA	:	Left subclavian artery
MRI	:	Magnetic resonance imaging
MRA	:	Magnetic resonance angiography
OR	:	Operating room
PAU	:	Penetrating atherosclerotic ulcer
TEE	:	Trans-esophageal echocardiography

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INTRODUCTION

Type A acute aortic dissection (TAAAD) represents a cardiovascular emergency event and remains one of the most challenging diseases the cardiothoracic surgeon faces. The course of an untreated TAAAD has the potential to rapidly progress into a catastrophic state. The known factors that contribute to increasing the mortality rate **(Mehta RH, et al, 2002)** include the development of pericardial tamponade from aortic rupture, acute myocardial ischemia or infarction due to involvement of the coronary arteries, extension of dissection into the branch vessels leading to compromised distal organ perfusion, aortic rupture into the pleural space, and aortic valve involvement resulting in acute onset heart failure **(Ramanath VS, et al 2009).** In the largest review of untreated patients with TAAAD in 1972, the estimated mortality without surgical treatment for TAAAD was 1% to 2% per hour; the mortality rate was about 50% within the first 48 hours 58% with less than 10% surviving 3 days (**Hagan PG, et al 2000)**. In a population-based series, the incidence of TAAAD was 3.5 per 100,000 person-years, with an overall 5-year survival rate of 32% and a median survival of 3 days, there is a general agreement that the operation must be performed as soon as possible, before complications occur or their effects become irreversible (**Clouse WD, et al 2004**).

In-hospital mortality after surgical treatment of TAAAD is 14% to 32.5% in published series **(Halstead JC, et al 2007)**. Expeditious surgical treatment is therefore essential in successful management of patients with TAAAD.

A report from the IRAD Investigators disclosed a high rate in-hospital mortality (23.9%) **(Rampoldi V,et al 2007)** (range;15% to 30%) and survival rates of 51-75% at 5 years have been reported **(Ramanath VS, et al 2009).** Late deaths are mainly due to chronic dissection rupture, end-stage heart failure, stroke, and reoperations **(Ramanath VS, et al 2009).**

The most important goal of the initial surgical management of TAAAD thought to be immediate survival by recover the continuity of the aortic wall and to reduce the acute phase deaths **(Heinemann M, 1990)**. Therefore, many surgeons favor a simple and less-invasive operative procedure for such patients by performing replacement of proximal segments of ascending aorta, including the concavity of the aortic arch (hemiarch repair)**,** remains sufficient for the acute situation in the majority of patients, conflicting opinions exist about the optimal extent of aortic correction. These operations are unable to prevent the need for distal reoperation in the future. **(Rampoldi V, et al 2007)**.

Several studies showed that a more radical and extensive operation, such as total arch replacement, regardless of the location of the entry site or extent of pathologic process, did not increase operative morbidity and mortality, and might even improve long-term outcome by decreasing the incidence of residual **(Hirotani T, et al 2003)** patent false lumen **(Urbanski PP, et al 2003).**

Although TAAAD remains a challenging condition to manage, the results from emergency surgical treatment are continuing to improve for the last decade and have reduced the immediate postoperative mortality risks of surgery **(Rampoldi V, et al 2007)**.

As the result of significant advances in diagnosis, the pre-operative work- up, the methods of cardiopulmonary bypass, surgical methods, the post-operative care. Those operative techniques including hypothermic circulatory arrest, retrograde cerebral perfusion, and selective cerebral perfusion which enable complete elimination of the primary tear and open distal anastomosis **(Pacini D, et al 2006)**, extended total arch replacement **(Watanuki H, et al 2007)** has been advocated as an aggressive approach with good clinical outcomes instead of ascending and hemiarch replacements **(Kazui T, et al, 2000)**.

Unfortunately, recent several studies showed that because of the extent of the disease to the entire aorta, the long-term prognosis after TAAAD surgery remains disappointing surviving the initial operation is not guarantee freedom from subsequent aortic events because much of the descending thoracic aorta remains dissected and at risk of aneurysm formation. The postoperative fate and long-term behavior of the distal aorta after TAAAD and false lumen is still unclear as patency of the descending aortic false lumen remains little affected by the surgical outcome of the primary proximal repair **(Park KH, et al 2009)** leading to the long-term prognosis after TAAAD surgery remains disappointing..

The false lumen can undergo thrombosis or remain patent postoperatively most surviving patients continue to have a residual patent distal false lumen in the downstream aorta **(Heinemann M, et al, 2002)**, after conventional surgery TAAAD **(Park K-H, et al 2009)** ,residual patent false lumen has recently been reported as a potential risk factor for distal aortic enlargement progressively, aneurismal evolution and its complications, especially the rupture, that is the first cause of late

death **(De Bakey ME, et al, 1982)** and poor long-term outcomes **(Halstead JC,et al 2007)** e.g. reoperation, or both. In association with this, as one of the efforts which has been made to obliterate a false lumen,

However, the effect of partial thrombosis of the false lumen after TAAAD repair on long term outcomes has not been elucidated. **(Tsai TT, et al 2007)** showed that partial thrombosis in patients with acute type B aortic dissection predicts poor survival. Others hypothesized that the postoperative status of distal false lumens would be identical to that of acute type B aortic dissection

Although early and late results of repair of TAAAD have been widely analyzed (**Ruvolo G, et al 2002**), few studies have investigated the evolution of the false lumen and the incidence of descending aortic related events. As experience grows, the awareness of late dilatation in primarily untreated segments requiring secondary surgical intervention in downstream segments increases**(Fleck T et al, 2002).**

AIM OF WORK

This study was undertaken to assess the early, mid, and long-term outcomes after TAAAD repair, we evaluated the false lumen changes in the descending aorta significant risk factors for patency of the false lumen and studied the relationship between the aortic diameter and the false lumen patency status in these patients, distal aortic segmental growth rates, dissecting aneurysm evolution, distal aortic reoperations, late survival and deaths.

ANATOMY OF AORTA

THORACIC AORTA:

The thoracic aorta is divided into 4 parts: the aortic root (which includes the aortic valve annulus, the aortic valve cusps, and the sinuses of Valsalva); the ascending aorta (which includes the tubular portion of the ascending aorta beginning at the sino-tubular junction and extending to the brachio-cephalic artery origin); the aortic arch (which begins at the origin of the brachio-cephalic artery, and is the origin of the head and neck arteries; and the descending aorta (which begins at the isthmus between the origin of the left subclavian artery and the ligamentum arteriosum and courses anterior to the vertebral column, and then through the diaphragm into the abdomen) **(Standring, 2008).**

Ascending Aorta

Course:

The ascending aorta is 5 cm long, begins at the base of the left ventricle at the level of the lower border of the third left costal cartilage; it ascends obliquely, curving forwards and right, behind the left half of the sternum to the level of the upper border of the second left costal cartilage **(Hiratzka et al., 2010).**

Aortic Arch

Course:

The aortic arch continues from the ascending aorta. Its origin, slightly to the right, is at the level of the upper border of the second right sterno-costal joint.

The arch first ascends diagonally back and to the left over the anterior surface of the trachea, then back across its left side and finally

descends to the left of the fourth thoracic vertebral body, continuing as the descending thoracic aorta. It ends level with the sternal end of the second, left costal cartilage. Thus the aortic arch lies wholly in the superior mediastinum. Its diameter at the origin is the same as in the ascending aorta about 28 mm, but it is reduced to 20 mm at the end, after the issue of its large branches. At the border with the thoracic aorta, a small stricture (aortic isthmus), followed by a dilatation, can be recognized. In fetal life the isthmus lies between the origin of the left subclavian artery and the opening of the ductus arteriosus **(Standring, 2008).**

Branches:

Three branches arise from the convex aspect of the arch: the brachio-cephalic trunk, left common carotid and left subclavian arteries. They may branch from the beginning of the arch or the upper part of the ascending aorta. The distance between these origins varies, the most frequent being approximation of the left common carotid artery to the brachio-cephalic trunk.

Variations of the branches:

An analysis of variation in branches from 1000 aortic arches showed the usual pattern in 65%; a left common carotid shared the brachio-cephalic trunk in 27%, and the four large arteries branched separately in 2.5%. The remaining 5% showed a great variety of patterns, the most common (1.2%) being symmetric right and left brachio-cephalic trunks **(Standring, 2008).**