Large Area of the False Lumen Favors Secondary Dilatation of the Aorta After Acute Type A Aortic Dissection

Franz F. Immer, MD; Eva Krähenbühl, MD; Urs Hagen, MD; Mario Stalder, MD; Pascal A. Berdat, MD; Friedrich S. Eckstein, MD; Jürg Schmidli, MD; Thierry P. Carrel, MD

Background—Since 1994 patients with acute aortic dissection type A (AADA) are followed-up in our outpatient clinic. Early diagnosis of secondary dilatation of the diseased aorta is crucial to reduce late mortality in these patients. Aim of the present study is to assess the impact of a large volume in the false lumen of the diseased downstream aorta on secondary dilatation.

Methods and Results—134 patients of 264 patients who underwent surgery for AADA (between January 1994 and June 2003) are followed-up at our outpatient clinic since 1994. 84 patients (62.7%) fulfilled the inclusion criteria. Areas of the true and false lumens of the aorta were analyzed and a logistic regression was calculated at 5 levels of the aorta for each patient. Patients were divided in 3 groups: group 1 included 34 patients (40.5%) without progression, group 2 had 34 patients (40.5%) with slight progression, and group 3 had 16 patients (19.0%) with important progression, requiring surgery in all patients. In 87.5% of the patients the area of the original lumen was <0% in group 3, compared with 11.8% in group 2 and 8.8% in group 1 in relation to the total area of the aorta 6 months after surgery (P<0.001).

Conclusion—A large false lumen, with an area of the true lumen <30% 6 months after surgery, is the strongest predictor for secondary dilatation of the diseased downstream aorta. 

Key Words: aorta • dissection • follow-up studies • risk factors • type A

In patients with acute type A aortic dissection (AADA), surgical repair is in the majority of patients limited to the ascending aorta and, in some cases, additionally to the aortic arch. In dissections De Bakey type I, the aortic arch, as well as the downstream aorta remain dissected and are at risk for secondary dilatation in the close future.1-4

We have recently shown that the extent of destruction, especially in younger patients, involving the arch vessels and/or combined with malperfusion syndrome favor secondary dilatation.5 Patency of the false lumen has been shown to be the most important risk factor for aortic enlargement in patients with chronic type B aortic dissection (De Bakey type III).6,7 Several studies focused on the growth rate of aortic aneurysms.2-4 However, to our knowledge, there have been no reports on the influence of the area of a patent false lumen on the growth rate on dissected aortic arch and downstream aorta after surgery for AADA.

The purpose of this study was to evaluate the impact of the area of the patent false lumen on the growth rate of De Bakey type I double barrel aortic dissection with repeated computed tomography (CT) or magnetic resonance imaging (MRI) examinations.

Materials and Methods

Between January 1994 and June 2004, 134 of 264 patients who underwent surgery for AADA have been followed-up at our outpatient clinic. Patients with De Bakey type I dissection and at least 3 postoperative CT scans or MRI were included in the present study. 84 patients (62.7%) fulfilled the inclusion criteria. Because of the fact that patients are referred to our hospital from all over Switzerland, 116 patients have been followed-up elsewhere by their local cardiologists and/or general practitioners. 14 patients have been excluded from the present study, because dilatation of the aorta (≥5 cm) was already present at the time of diagnosis of AADA. Early re-operations were required in these patients (<6 months after surgery for AADA) and 6 (42.9%) had Marfan syndrome.

Analyses of the areas of the true and false lumens were performed and a linear logistic regression was calculated at 5 different levels of the aorta for each patient. Analyses were performed at the aortic arch, in the descending aorta at the level of the pulmonary artery bifurcation, supra-diaphragm and infra-diaphragm, as well as at the level of infrarenal abdominal aorta. According to the rapidity of subsequent dilatation, patients were divided into 3 groups: group 1 included 34 patients (40.5%) without progression of the diameter in the downstream aorta, group 2 included 34 patients (40.5%) with slight progression, and group 3 included 16 patients (19.0%) with important progression, requiring surgery in all patients (100%). Logistic regression curve from group 1 was defined as y=a+b0x10.00167, group 2, y>0.0167x−0.0167, and <0.0333x−0.0333; and group 3, y>0.0333x−0.0333. A slight progression (group 2) was defined as an increasing in size of the aortic arch and/or the descending aorta between 1 cm and 2 cm over an extrapolated time period of 60 months and an important secondary dilatation (group 3) was defined as expansion of ≥2 cm in a logistic regression model. Measurement of diameters of the aortic arch and/or descending aorta were performed computer based. The areas of the false and the true
lumens were calculated at the time of diagnosis of AADA and 6, 12, and 18 months after surgery. A quotient reflecting the area of the true lumen in relation to the total area of the cross section of the aorta at the different levels was calculated.

Statistical Analysis

Data are presented as absolute values or mean values ± their first standard deviation. A Mann–Whitney *U* test and χ² test were used for comparison between groups of continuous and nominal variables, respectively. Linear regression for the relationship between modification of the diameter of the aortic arch and/or descending aorta over time was analyzed. Cutoff values for repartition of the 3 groups were defined to improve the predictive value. *P* < 0.05 was considered significant. Data were analyzed using the StatView 4.1 statistical package (Abacus Concepts, Berkley, Calif).

### Results

84 patients fulfilled the inclusion criteria. Pre- and perioperative characteristics of these patients are summarized in the Table. Follow-up was similar in all 3 groups, with an average follow-up of 55 ± 13 months. 584 CT scans or MRI were analyzed. 86% of the dilatations were found at the level of the aortic arch and/or thoracic descending aorta. 20% of the dilatations were found at level 1 (aortic arch), 46% at level 2, 20% at level 3 (thoracic descending aorta), and at levels 4 and 5 only 7% at each level. The relation between the areas of the true and the false lumens at the time of diagnosis and 6 months after surgery is displayed.

### Figure 1

Quotient (Area true lumen/area false lumen) displayed for groups 1 to 3 at the time of diagnosis and 6 months after surgery for AADA. Probability value compares quotients from groups 1 and 2 with quotient from group 3 and quotient at the time of diagnosis and at follow-up (t=6 months) in group 3.
in Figure 1. A significant difference was found at the time of diagnosis between groups 1 and 2, compared to group 3, which was even more pronounced 6 months after surgery, with a quotient of 0.9±0.5 in group 1, 0.9±0.6 in group 2, and 0.3±0.1 in group 3. A quotient <0.3 was found in 87.5% of the patients of group 3 at the follow-up, compared to 8.8% in group 2 and 11.8% in group 1 (P<0.05). Follow-up was similar in all groups.

The calculated odds ratio for a quotient <0.3 was 4.3 at the time of diagnosis and 7.4 at the follow-up 6 months after surgery. The maximal diameter of the aorta at the time of diagnosis of AADA was 3.4±0.8 cm in group 1, 3.3±0.6 cm in group 2, and 3.2±0.8 cm in group 3 (P=NS) (Figure 2).

Discussion
A patent false lumen has been shown to be a risk factor for secondary dilatation in patients with chronic type B aortic dissection. However, this observation has been discussed controversial. Dilatations of the diseased downstream aorta have been found in 83.9% of patients with chronic type B aortic dissection in an average follow-up period of 49 months. In the same observation period, dilatations were found in 59.5% of our patients, mainly at the level of the aortic arch and the thoracic descending aorta in patients with De Bakey type I dissection.

Because no significant difference was found of the aortic diameter at the time of diagnosis, one may assume that this aspect, which has been shown to be a risk factor for secondary dilatation, is not primary responsible for the dilatation observed in the first 6 months after surgery in group 3. However, patients with dilatation of the aortic arch and/or the downstream aorta at the time of diagnosis of AADA have been excluded from the present study, because they required surgery in the early follow-up. Of 14 patients being excluded, because of primary dilatation of the aorta, 6 (42.9%) had Marfan syndrome, which is a well-known risk factor for aneurysmatic dilatation of the aorta.

Analyses of the area revealed that expansion of the aorta, observed 6 months after surgery in group 3, is mainly caused by a dilatation of the false lumen, because the volume of the true lumen remains quite stable over time. This is probably related to the instability of the dissected aortic wall, mainly at the site of the false lumen, where the quality of the aortic wall is impaired. The increase of the area of the false lumen leads to the observed reduction of the true lumen (Q<0.3) in 87.5% of our patients from group 3, 6 months after surgery (odds ratio, 7.4).

Additionally, dilatation is favored because of the blood flow in the patent false lumen, increasing wall tension. Only a few invasive blood pressure measurements are available in these patients up to now, revealing that in patients with a large area of the false lumen, blood pressure is higher in the false lumen, compared with the values in the true lumen. However, these are only preliminary data in 8 patients, not allowing us to draw conclusions based on these observations.

We are aware that group 3 is only a small group, including 16 patients with an important progression of the dissected aortic arch and/or the downstream aorta after surgery for AADA. However, despite the small number of patients in group 3, the results are highly significant compared with the data from patients of groups 1 and 2, which counterbalances the limitation of the present study.

We conclude that a large area of the patent false lumen (>70% of the total area of the total trans-sectional diameter of the aorta) is the strongest predictor for secondary dilatation in the diseased downstream aorta. A follow-up should be performed in all patients after surgery for AADA at least up to 24 months after surgery to prevent acute late complications of the downstream aorta.

References


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