Long-Term Outcome of Aortic Dissection with Patent False Lumen: 
Predictive Role of Entry Tear Size and Location

Running title: Evangelista et al.; Long-term outcome of aortic dissection

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Abstract:

**Background** - Patent false lumen in aortic dissection (AD) has been associated with poor prognosis. We aimed to assess the natural evolution of this condition and predictive factors.

**Methods and Results** - One hundred and eighty-four consecutive patients, 108 surgically treated type A and 76 medically-treated type B, were discharged after an acute AD with patent false lumen. Transesophageal echocardiography was performed prior to discharge, and computed tomography at 3 months and yearly thereafter. Median follow-up was 6.42 years (Quartile 1-Quartile 3 [Q1-Q3]: 3.31-10.49). Forty-nine patients died during follow-up (22 type A, 27 type B), 31 suddenly. Surgical or endovascular treatment was indicated in 10 type A and 25 type B cases. Survival free from sudden death and surgical-endovascular treatment was 0.90, 0.81 and 0.46 (95% confidence interval [CI]: 0.36-0.55) at 3, 5 and 10 years, respectively. Multivariable analysis identified baseline maximum descending aorta diameter (Hazard Ratio [HR]: 1.32 (1.10-1.59); p=0.003), proximal location [HR: 1.84 (1.06-3.19); p=0.03] and entry tear size [HR: 1.13 (1.08-1.2); p<0.001] as predictors of dissection-related adverse events, while mortality was predicted by baseline maximum descending aorta diameter [HR: 1.36 (1.08-1.70); p=0.008], entry tear size [HR: 1.1 (1.04-1.16); p=0.001] and Marfan syndrome [HR: 3.66 (1.65-8.13); p=0.001].

**Conclusions** - Aortic dissection with persistent patent false lumen carries a high risk of complications. In addition to Marfan syndrome and aorta diameter, a large entry tear located in the proximal part of the dissection identifies a high-risk subgroup of patients who may benefit from earlier and more aggressive therapy.

**Key words:** aorta; computed tomography; prognosis; transesophageal echocardiography
Introduction

The long-term outcome of patients with successful initial treatment of acute aortic dissection and persistent patent false lumen in the descending aorta is not well established. Several studies have reported long-term overall survival of 50-80% at 5 years and 30-60% at 10 years \(^1\)\(^-\)\(^3\), with no differences between Stanford type A and B dissections \(^3\). Persistent patent false lumen in the descending aorta is common in both types and has been strongly associated with poor prognosis \(^3\)\(^-\)\(^7\). However, the majority of series of type A and B dissections did not exclude cases with absence of residual dissection, total false lumen thrombosis or intramural hematomas \(^3\)\(^-\)\(^8\), which imply a different natural history \(^9\)\(^-\)\(^10\).

Advances in imaging techniques may provide significant information \(^11\)\(^-\)\(^13\) for identifying patients at higher risk of adverse events. The advent of thoracic endovascular aortic repair raised new expectations for the early management of complicated aortic dissection \(^14\)\(^-\)\(^16\) by occluding the intimal tear, restoring true lumen flow and inducing false lumen thrombosis. However, to date, no study has shown that elective endovascular treatment in subacute phase of aortic dissection reduces mortality. Recent INSTEAD trial \(^16\) results failed to show an improvement in 2-year survival and adverse event rates. Therefore, identification of clinical and imaging predictors of poor prognosis seems mandatory to select patients for whom more aggressive management may be beneficial.

The aim of the present study was to assess the long-term outcome of aortic dissection with persistent patent false lumen in the descending aorta and define the clinical and imaging variables obtained in subacute phase that could predict adverse events during follow-up.
Methods

Study population

From January of 1994 to December 2009, 316 consecutive patients were discharged after an initial episode of acute aortic syndrome: 222 aortic dissections and 94 intramural hematomas. Of the aortic dissection group, 38 were not included: 27 without residual patent false lumen (12%), 9 with complicated acute type B dissection requiring surgical/endovascular treatment (11%) and 2 for residing far from the hospital. Therefore, the study group comprised 184 patients, 108 surgically-treated type A and 76 medically treated type B dissections, discharged with patent false lumen in descending aorta confirmed by transesophageal echocardiographic study (TEE).

Follow-up protocol

Patients underwent a strict clinical and imaging protocol and no patient was lost to follow-up. All admitted patients underwent TEE prior to discharge, and computed tomography (CT) and/or magnetic resonance imaging (MRI) were performed during the first month of follow-up. The findings of these studies constituted the baseline information. Patients were discharged with beta-blocker treatment, if not contraindicated, combined or not with other antihypertensive drugs aimed at achieving systolic blood pressure < 130 mm Hg. Patients were followed by two clinical cardiologists (AS and AE) who checked for adequacy of blood pressure control and the presence of symptoms or complications in imaging tests (CT, TEE or MRI depending on medical criteria) at 3 and 6 months and annually thereafter.

Surgical or endovascular treatment during follow-up was indicated according to a preestablished protocol. Intervention was considered if any of the following were observed: a) dissection progression with signs of impending rupture; b) signs of impaired visceral or peripheral perfusion; c) retrograde extension of the dissection to the ascending aorta; and d)
aortic diameter > 60 mm. The study was approved by the institutional review committee and patients gave their written informed consent.

The primary clinical end-points during follow-up were overall mortality and a presumably dissection-related event: death clearly related to any aortic dissection complication, otherwise unexplained sudden death, or the need for surgical/endovascular treatment in the descending aorta owing to severe complications. Ascending aorta surgery related to suboptimal results in acute phase was not considered a major event during follow-up. Clinical and imaging follow-up information was obtained for all patients.

**Imaging techniques**

TEE was carried out with GE System V or Vivid 7 ultrasound equipment. The TEE transducer was 5 MHz, with the information assessed by two-dimensional echocardiography and color Doppler. CT was performed with helical CT (Mx 8000; Philips) generating axial images with contiguous 5-mm-thick sections from the top of the aortic arch to the abdominal aorta, with nonenhanced and enhanced CT in all patients. Since 2001, multidetector CT angiography has been performed with a 16-detector Siemens Sensation which produces 1-mm slices at 0.5-mm intervals (50% overlap). MRI studies were performed with a Siemens Magnetom 1.5 – T (Erlangen, Germany) device. A standardized protocol including ECGgated spin-echo, HASTE sequences and a breath-hold gadolinium-enhanced rapid MR angiographic technique was followed in all studies.

**Imaging variables**

The following imaging variables were pre-specified and carefully assessed at each examination:

a) maximum aorta diameter in ascending aorta, arch and descending aorta segments determined by CT. The largest short-axial diameter perpendicular to the outer contour of the aorta was
measured. In cases where the cross-sectional slice of the aorta had an elliptical shape, the smallest of the two diameters was considered; b) antegrade or retrograde false lumen flow was analyzed by contrast CT and/or MRI angiography; c) true lumen compression was considered to be present when true lumen diameter represented less than 25% of the overall aorta diameter in axial slices by imaging tests; at least along 2/3 of the length of the descending thoracic aorta; and d) entry tear or main proximal communication between true and false lumina was defined as the largest communication which caused maximum entry flow into the patent false lumen.

Therefore, in type A, in which a primary entry tear had been removed at surgery, the proximal maximum communication between both lumina which caused the principal entry flow into the false lumen was considered to be the residual entry tear. Location and size of the entry tear in subacute phase were assessed by 2D and color Doppler TEE (Figure 1). CT was used when the entry tear was not correctly visualized by TEE. In fact, entry tear was visualized by TEE in most cases: 164 (89%); in the remaining 20 cases, it was only visualized by CT: 16 with entry tear located in the upper part of the ascending aorta and 4 in the abdominal aorta. Entry tear was considered proximal when located in the upper part of the ascending aorta or arch in an operated type A dissection, or at the proximal part of a type B dissection. Entry tear size was quantified by the maximum diameter of the tear by TEE using longitudinal and transverse views (Figure 2). In cases where the entry tear was not visualized by TEE, it was measured by CT. Interobserver variability of maximum entry tear diameter was analyzed in 20 cases in which TEE was repeated with an interval shorter than one month by two different and blinded echocardiographers from our department. Intraclass correlation coefficient was 0.86, mean difference (SD) = 0.35 mm (2.8 mm), p: ns. Agreement between maximum entry tear diameter measured by TEE performed before discharge and CT at 3 months in 20 random cases was high:
intraclau correlation coefficient = 0.85, mean difference (SD) = 0.05 mm (2.7 mm), p: ns.

**Statistical analysis**

Descriptive data are presented as means (standard deviation) or medians (quartile 1-quartile 3 [Q1-Q3]) or proportions depending on the variable distribution. Comparisons of variables between patients with type A or B dissections were made by Student’s t-test, Pearson’s chi-square test or Fisher’s exact test where appropriate. Individual aortic growth rates were calculated by dividing the change in aorta diameter measured in millimeters by time measured in years. For analysis purposes, entry tear location was dichotomized in proximal versus medial and distal since the number of entry tears located in the distal part of the dissection was small.

Growth rates were compared using the Kruskal-Wallis chi-square rank test. Receiver operator characteristic (ROC) curves were used to analyze the ability of entry tear size to predict adverse aortic events. Survival rates were estimated by the Kaplan-Meier method. Relationships between clinical and imaging variables and follow-up adverse events were assessed using univariable proportional hazards regression analysis. For the overall mortality endpoint, patients experiencing a non-fatal dissection-related event (need for surgical/endovascular treatment) were censored. For the dissection-related event endpoint, patients dying from other causes were censored.

Determinants of overall mortality rate or dissection-related events were also assessed by Cox proportional hazards regression analysis. Potential predictive variables were selected on the basis of clinical plausibility and their significant association with outcome in univariable Cox proportional hazards regression and retained in the final model when the p value was <0.2 (using the backwards stepwise variable selection procedure). Since our goal was to develop a parsimonious prediction model from potential baseline clinical predictors, variables such as chest
pain and blood pressure control assessed during follow up were not considered for prediction purposes. First order interaction terms were tested. All p-values were two-sided and considered significant at the 0.05 threshold.

Results

Study population and baseline characteristics

Population characteristics and baseline imaging study results stratified by aortic dissection type are shown in Table 1. As expected, significantly-different clinical variables between type A and type B dissections were age, hypertension and dyslipidemia, which were more prevalent in type B, and Marfan syndrome, more common in type A. Concerning imaging variables, differences were observed between type A and B groups (Table 1). Maximum descending aorta diameter was similar in both types. Most entry tears were located in the proximal part of the dissection in both types (44.4 % vs 51.3 %). However, medial location was more frequent in type B (46.1% vs 30.6%) while distal location was observed more in type A (25.0% vs 2.6%, p<0.001). Entry tear size ranged from 3 mm to 27 mm (median: 7 mm, Q1-Q3: 5-12 mm). Entry tear size was larger in type B than in type A (10.4±5.2 mm vs 7.3±4.2 mm, p< 0.001). Entry tear diameter was ≥10mm in 45 (59.2%) of type B and in only 31 (40.8%) of operated type A dissections (p< 0.001). The frequency of true lumen compression was similar in both types.

Long-term outcome and mortality predictors

Median follow-up was 6.4y (range: 0.5-17y; Q1-Q3: 3.3-10.5 y). Type B dissections presented a higher enlargement rate of maximum aorta diameter than type A: 0.21 mm (0.09 – 0.76) vs 0.48mm (0.12 – 1.57), p=0.04, respectively (Supplemental Figure 1). Seven patients underwent surgical and 28 endovascular treatment, 10 type A and 25 type B, owing to severe aorta
dilatation, impending rupture and/or peripheral ischemia. Forty-nine patients died during follow-up (22 type A, 27 type B). Of these, 31 had died suddenly: 11 during readmission with signs of aortic rupture and 20 at home from unknown causes. Six hospitalized patients died during surgical (n:3) or endovascular (n: 3) treatment performed for chest pain and severe aortic dilatation. The remaining 12 patients died from causes not directly related to aortic dissection: neoplasia (7), respiratory infection (3) and multiorgan failure in the context of sepsis (2). The incidence rate (CI 95%) for overall mortality was 0.04 (0.03-0.06) and for dissection-related events 0.06 (0.04-0.07). Survival rates of the total population at 3, 5 and 10 years were 0.95 (95% CI: 0.90-0.97), 0.87 (95% CI: 0.80-0.91) and 0.52 (95% CI: 0.41-0.62), respectively (Figure 3A). Survival free from sudden death and surgical/endovascular treatment for the total population at 3, 5 and 10 years was 0.90 (95% CI: 0.84-0.94), 0.81 (95% CI: 0.75-0.87) and 0.46 (95% CI: 0.36-0.55), respectively (Figure 3B).

The optimal cut-off value of entry tear size for prediction of aortic complications during follow-up, identified by ROC analysis, was ≥ 10 mm with 85% sensitivity and 87% specificity (Supplemental Figure 2). Patients with entry tear size ≥ 10 mm presented a higher incidence of dissection-related events than those with entry tear < 10mm (HR=5.8 (3.3 – 10); p<0.001). The median growth rate in patients with entry tear ≥10 mm was significantly higher than in patients with an entry tear < 10 mm, (0.80 mm, Q1-Q3: 0.17 – 1.88 vs 0.16mm, Q1-Q3: 0.07 – 0.48, p< 0.001, respectively).

Univariable Cox regression analyses relating likelihood of death or an aortic dissection-related event to baseline clinical and imaging variables are shown in Table 2. Marfan syndrome was the only clinical variable associated with mortality. Concerning imaging variables, patients with outcome events had a larger baseline maximum diameter in descending aorta and a large
proximal entry tear.

Multivariable predictors of events are shown in Table 3. Marfan syndrome, baseline maximum descending aorta diameter and entry tear size predicted total mortality. Similarly, baseline descending aorta diameter and entry tear size and also proximal location predicted aortic events. The association between entry tear location and size and aorta-related events is shown in Figures 4A and 4B. As shown in figure 5, stratifying by entry tear size and location, those patients with proximal entry tear \( \geq 10 \) mm, trend to a higher rate of complications during follow up, although it did not reach statistical significance.

Discussion

This study shows that, in the long-term, aortic dissection with patent false lumen in the descending aorta presents a high risk of complications. In addition to the well known risk factors, (Marfan syndrome \(^1,2,22-23\) and maximum descending aorta diameter \(^1,7,23-25\)), this study identifies the presence of a large proximal entry tear as a further predictor of mortality and the need for surgical/endovascular treatment.

**Long-term evolution in type A and B dissections with patent false lumen**

Patent false lumen in descending aorta segments after surgical treatment of type A dissection is common (64%-90%) \(^{22,26-29}\). Suboptimal connection of the distal part of the graft implanted in the ascending aorta to the true lumen or presence of secondary tears may account for the persistence of flow into the distal residual false lumen after complete surgical resection of the primary entry tear \(^{13,20,22,26}\). On the other hand, most studies evaluating the long-term outcome of type B dissection usually include intramural hematoma \(^4-7\), a different entity with a different pathogenesis and evolution pattern \(^9,10\), along with instances of aortic dissection with total false
lumen thrombosis. No studies have compared the long-term evolution of types A and B dissections with persistent patent false lumen in a consecutive series treated with a preestablished protocol. In the present study the aortic growth rate was higher in type B than in type A dissections (0.48 vs 0.21 mm/y, respectively). This growth rate was lower than in other series, probably due to a longer follow-up period \(^{4,7,8,24,27,30}\). Nevertheless, 19% (9% type A and 33% type B) of our patients required surgical or endovascular therapy during follow-up with interventional mortality similar to those reported in other series \(^{6,8,25,31}\). Overall mortality in our study was 5% at 3 years, 13% at 5 years and 48% at 10 years (20% in type A and 36% in type B), similar to those of contemporary series from major aortic surgery institutions \(^{6,7,23,26-29}\).

**Predictors of complications**

Some clinical predictors of complications, such as Marfan syndrome \(^{1,2,28}\), age \(^{3,26,29}\) or atherosclerotic disease have been reported previously \(^{32}\). In our series, only Marfan Syndrome was a clinical predictor of complications, with a 70% mortality rate at 10 years. Our study confirms the predictive value of maximum descending aorta diameter in subacute phase \(^{4-7,24,25}\).

Other groups reported that true lumen compression or large false lumen diameter had significant prognostic implications \(^{13,28}\). Nevertheless, these variable have significant limitations since true and false lumen size may vary considerably in the different aorta segments depending on several local factors such as tortuositites, spiral flap distribution and intimal flap mobility. In the present series, true lumen compression was more frequent in the group of patients with major events although it was not a predictor of complications after the inclusion of entry tear size in the multivariate analysis.

In the present study, size and proximal location of the entry tear were predictors of complications. Phantom studies have demonstrated that the larger the intimal tear in the proximal
aorta, the greater the tendency to exacerbate true lumen collapse. Furthermore, Tsai et al. showed that systolic pressure in the false lumen, compared to the true lumen, falls with decreasing tear size. Therefore, increased pressure with greater tear size will result in higher wall stress and risk of dilatation. They additionally suggested that proximal location of the entry tear was shown to cause a rise in false lumen diastolic pressure, thereby implying a tendency towards dilatation. Other studies suggested the possible predictive role of a proximal entry tear. Quint et al. assessed entry tear size and location by spiral CT in 52 patients with chronic aortic dissection, obtaining similar results in this finding to those of our series; the most common location was the proximal descending aorta (44%) and most tears (68%) were small (< 10mm), mainly in type A dissections. Accordingly, the higher rate of complications in type B versus operated type A dissections observed in our study may be justified by the more frequent presence of large entry tears in type B dissections.

An interesting finding of the present study was that both mortality and the need for surgical/endovascular treatment increased from the third year of follow-up after acute aortic syndrome in patients with persistent patent false lumen, which suggests that structural and/or dynamic factors responsible for dissection complications require time to appear. This fact could explain the lack of benefit of endovascular treatment in the INSTEAD trial. In that randomized study, elective stent-graft placement in survivors of uncomplicated type B dissection failed to improve 2-year survival and adverse rates despite favorable aortic remodeling. Moreover, we believe that the hemodynamic implications of patent false lumen are probably too complex to be understood exclusively from baseline morphologic imaging variables. In the future, other more dynamic variables such as flow patterns in true and false lumina assessed by contrast echocardiography or MRI should be considered in the hemodynamic assessment of patent false
lumen.

**Limitations**

The present study had several limitations. The series was not large, since only patients with persistent patent false lumen following conventional treatment were included in an attempt to obtain a population as homogeneous as possible. Nevertheless, compliance with the protocol was excellent, and no patient was lost to follow-up. During the study period, advances in imaging techniques improved morphologic and functional assessment. Although maximum entry tear diameter may not be representative of entry tear size, this parameter showed good reproducibility both by TEE and CT. However, the predictive value of this parameter should be validated in other series. Finally, partial false lumen thrombosis in subacute phase has been considered a predictor of mortality in type B dissection32. In the present study, this variable was not analyzed since assessment of the entire aorta by CT before discharge was not performed in all cases and many patients with type A dissection were under anticoagulation treatment. Future studies should analyze the relationship of this finding with entry tear size and its predictive value.

**Clinical implications**

The optimal management of patients discharged with persistent patent false lumen after treatment for acute aortic dissection remains under debate23,36,37. Although the short term evolution is stable, the incidence of complications rises after three years of evolution, particularly in type B dissections. Surgical or endovascular entry tear closure promotes both thrombosis of the false lumen and remodeling of the entire aorta14-16. It has been suggested that treatment efficacy should be greater in subacute phase than in subsequent periods when the aorta is severely dilated and the intima less elastic16,36,37. Our findings may help to identify patients at higher risk of complications by imaging techniques performed in the subacute phase of aortic
dissection. In addition to dilated aorta, the clinical outcome is more likely to be poor in those with proximal and large entry tears. Thus, these patients may benefit from more aggressive surveillance and treatment. These results also emphasize the importance of resecting or closing any significant communication between true and false lumina in the distal ascending aorta or aortic arch in patients undergoing surgery for type A aortic dissection.

Conclusions

The long-term outcome of aortic dissection with patent false lumen shows a high risk of complications, sudden death and need for surgery, particularly from the third year of evolution. In addition to Marfan syndrome, maximum aorta diameter and the presence of a large, proximal entry tear imply a higher incidence of complications during follow-up. Information obtained by imaging techniques in the subacute phase can help to identify patients at greater risk of complications and facilitate the indication of more aggressive treatment, including surgery or endovascular treatment, to improve their long-term prognosis.

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Conflict of Interest Disclosures: None.

References:


### Table 1. Demographic, clinical, imaging and in-hospital complications according to aortic dissection type.

<table>
<thead>
<tr>
<th></th>
<th>Overall (n=184)</th>
<th>Type A (n=108)</th>
<th>Type B (n=76)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean±SD)</td>
<td>56±12.9</td>
<td>55.4±13.3</td>
<td>57±12.4</td>
<td>0.43</td>
</tr>
<tr>
<td>Men (n;%)</td>
<td>144 (78.3)</td>
<td>85 (78.7)</td>
<td>59 (77.6)</td>
<td>0.86</td>
</tr>
<tr>
<td>Hypertension (n;%)</td>
<td>141 (76.6)</td>
<td>77 (71.3)</td>
<td>64 (84.2)</td>
<td>0.04</td>
</tr>
<tr>
<td>Marfan (n;%)</td>
<td>26 (14.1)</td>
<td>20 (18.5)</td>
<td>6 (7.9)</td>
<td>0.04</td>
</tr>
<tr>
<td>Dyslipidemia (n;%)</td>
<td>42 (22.8)</td>
<td>22(20.4)</td>
<td>20(26.3)</td>
<td>0.34</td>
</tr>
<tr>
<td>Diabetes (n;%)</td>
<td>13 (7.1)</td>
<td>8(7.4)</td>
<td>5 (6.6)</td>
<td>0.83</td>
</tr>
<tr>
<td>Atherosclerosis (n;%)</td>
<td>24 (13)</td>
<td>14 (13)</td>
<td>10 (13.2)</td>
<td>0.97</td>
</tr>
<tr>
<td>Previous aortic disease (n;%)</td>
<td>30 (16.3)</td>
<td>13 (12)</td>
<td>17 (22.4)</td>
<td>0.06</td>
</tr>
<tr>
<td>COPD* (n;%)</td>
<td>35 (19)</td>
<td>19 (17.6)</td>
<td>16 (21.1)</td>
<td>0.56</td>
</tr>
<tr>
<td>Renal failure (n;%)</td>
<td>40 (21.7)</td>
<td>26 (24.1)</td>
<td>14 (18.4)</td>
<td>0.36</td>
</tr>
<tr>
<td>Arch involvement (n;%)</td>
<td>100 (54.4)</td>
<td>90 (83.3)</td>
<td>10 (13.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Baseline maximum diameter (mean±SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascending aorta (mm)</td>
<td>41.3±8</td>
<td>41.9±8</td>
<td>40.4±7</td>
<td>0.18</td>
</tr>
<tr>
<td>Arch (mm)</td>
<td>38.2±7</td>
<td>38.4±7</td>
<td>37.9±6</td>
<td>0.61</td>
</tr>
<tr>
<td>Descending aorta (mm)</td>
<td>39.5±7</td>
<td>37.7±7</td>
<td>42±8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Descending aorta &gt;45mm (n;%)</td>
<td>32 (17.6)</td>
<td>11 (10.4)</td>
<td>21 (27.6)</td>
<td>0.001</td>
</tr>
<tr>
<td>Entry tear location (n;%)</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Proximal location (n;%)</td>
<td>87 (47.3)</td>
<td>48 (44.4)</td>
<td>39 (51.3)</td>
<td></td>
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<tr>
<td>Medial location (n;%)</td>
<td>68 (37)</td>
<td>33 (30.6)</td>
<td>35 (46.1)</td>
<td></td>
</tr>
<tr>
<td>Distal location (n;%)</td>
<td>29 (15.8)</td>
<td>27 (25.0)</td>
<td>2 (2.6)</td>
<td></td>
</tr>
<tr>
<td>Entry tear size (mm) (mean±SD)</td>
<td>8.6±4.9</td>
<td>7.3±4.2</td>
<td>10.4±5.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Annual maximum aortic diameter; increment (mm/year); median (Q1-Q3)</td>
<td>0.27</td>
<td>0.21</td>
<td>0.48</td>
<td>0.043</td>
</tr>
<tr>
<td>True lumen compression (n;%)</td>
<td>42 (23.6)</td>
<td>27 (26.5)</td>
<td>15 (19.7)</td>
<td>0.3</td>
</tr>
</tbody>
</table>

*COPD: Chronic Obstructive Pulmonary Disease*
Table 2. Univariable Cox regression analysis predicting death and dissection-related events, based on demographic, clinical and imaging variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>HR Total mortality (95% CI)</th>
<th>p value</th>
<th>HR Dissection-related event (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical variables</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (for one-year increase)</td>
<td>1.01 (0.99 – 1.04)</td>
<td>0.39</td>
<td>0.99 (0.97 – 1.01)</td>
<td>0.34</td>
</tr>
<tr>
<td>Women (vs men)</td>
<td>1.19 (0.59 – 2.38)</td>
<td>0.63</td>
<td>1.26 (0.7 – 2.27)</td>
<td>0.45</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.16 (0.52 – 2.59)</td>
<td>0.72</td>
<td>0.81 (0.44 – 1.48)</td>
<td>0.49</td>
</tr>
<tr>
<td>Marfan Syndrome</td>
<td>2.39 (1.19 – 4.83)</td>
<td>0.02</td>
<td>1.93 (1.03 – 3.63)</td>
<td>0.04</td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td>1.43 (0.67 – 3.05)</td>
<td>0.36</td>
<td>0.74 (0.32 – 1.72)</td>
<td>0.49</td>
</tr>
<tr>
<td>Previous aortic disease</td>
<td>1.76 (0.9 – 3.45)</td>
<td>0.1</td>
<td>1.84 (1.04 – 3.29)</td>
<td>0.04</td>
</tr>
<tr>
<td>COPD*</td>
<td>1.08 (0.52 – 2.22)</td>
<td>0.84</td>
<td>1.38 (0.77 – 2.46)</td>
<td>0.28</td>
</tr>
<tr>
<td>Renal failure</td>
<td>0.38 (0.14 – 1.06)</td>
<td>0.07</td>
<td>0.86 (0.45 – 1.64)</td>
<td>0.64</td>
</tr>
<tr>
<td>Type of aortic dissection (B versus A)</td>
<td>1.74 (0.99 – 3.05)</td>
<td>0.06</td>
<td>2.6 (1.56 – 4.32)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Poor BP control during follow-up</td>
<td>1.29 (0.69 – 2.39)</td>
<td>0.43</td>
<td>1.36 (0.8 – 2.32)</td>
<td>0.26</td>
</tr>
<tr>
<td>Chest pain during follow-up</td>
<td>2.58 (1.44 – 4.62)</td>
<td>0.001</td>
<td>2.62 (1.58 – 4.34)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Imaging variables</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal vs medial or distal entry tear</td>
<td>1.71 (0.97 – 3.01)</td>
<td>0.07</td>
<td>2.6 (1.54 – 4.37)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Entry tear diameter (for 1-mm increase)</td>
<td>1.14 (1.08 – 1.2)</td>
<td>&lt;0.001</td>
<td>1.2 (1.15 – 1.26)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>True lumen compression</td>
<td>0.99 (0.51 – 1.94)</td>
<td>0.98</td>
<td>1.7 (1.02 – 2.85)</td>
<td>0.04</td>
</tr>
<tr>
<td>Baseline maximum descending aorta diameter</td>
<td>1.08 (1.04 – 1.13)</td>
<td>&lt;0.001</td>
<td>1.09 (1.05 – 1.13)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>(for each 5-mm increase)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*CopD: Chronic obstructive pulmonary disease; HR: Hazard ratio; BP: Blood pressure

Table 3. Predictors of total mortality and dissection-related events. Results of multivariable Cox models.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Adjusted HR (95% CI)</th>
<th>p value</th>
<th>Adjusted HR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total mortality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (for one-year increase)</td>
<td>1.02 (0.99 – 1.05)</td>
<td>0.17</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Type B (vs type A)</td>
<td>-</td>
<td>-</td>
<td>1.47 (0.84 – 2.56)</td>
<td>0.18</td>
</tr>
<tr>
<td>Marfan syndrome (vs rest)</td>
<td>3.66 (1.65 – 8.13)</td>
<td>0.001</td>
<td>1.79 (0.89 – 3.58)</td>
<td>0.1</td>
</tr>
<tr>
<td>Baseline maximum descending aorta diameter</td>
<td>1.36 (1.08 – 1.7)</td>
<td>0.008</td>
<td>1.32 (1.1 – 1.59)</td>
<td>0.003</td>
</tr>
<tr>
<td>(for each 5-mm increase)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Entry tear diameter (for 1-mm increase)</td>
<td>1.1 (1.04 – 1.16)</td>
<td>0.001</td>
<td>1.13 (1.08 – 1.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Proximal entry tear (vs rest)</td>
<td>-</td>
<td>-</td>
<td>1.84 (1.06 – 3.19)</td>
<td>0.03</td>
</tr>
</tbody>
</table>
Figure Legends:

**Figure 1.** Entry tear of aortic dissection visualized by 2D (left) and color Doppler (right) TEE: a) type B dissection with an entry tear located in the proximal part of the descending aorta (arrow) by tranverse view; b) type A dissection with an entry tear in the proximal part of the residual dissection (arrow) in the upper ascending aorta by longitudinal view.

**Figure 2.** Entry tear of type A dissection in the mid part of the descending thoracic aorta. Entry tear diameter was larger on transverse (a) than longitudinal (b) views, 13 mm vs 7 mm, respectively, and the former was considered to be the maximum entry tear diameter.

**Figure 3.** A) Cumulative survival of type A and type B dissections; B) cumulative survival free from sudden death and surgical/endovascular treatment of type A and type B dissections.

**Figure 4.** A) Cumulative survival free from sudden death and surgical/endovascular treatment by entry tear size; B) Cumulative survival free from sudden death and surgical/endovascular treatment by entry tear location.

**Figure 5.** Cumulative survival free from sudden death and surgical/endovascular treatment by entry tear pattern (size and location).
Long-Term Outcome of Aortic Dissection with Patent False Lumen: Predictive Role of Entry Tear Size and Location

Artur Evangelista, Armando Salas, Aida Ribera, Ignacio Ferreira-González, Hug Cuellar, Victor Pineda, Teresa González-Alujas, Bart Bijnens, Gaietà Permanyer-Miralda and David García-Dorado

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**FIGURE LEGENDS**

**Supplemental Figure 1.** Severe enlargement of the descending aorta in a type B dissection. Maximum descending aorta diameter was 49 mm in subacute phase (a) and 64 mm with partial false lumen thrombosis at 9 years of follow-up (b). A large entry tear of 27 mm was located in the proximal descending aorta (c).

**Supplemental Figure 2.** ROC curve for adverse aortic events and entry tear size.