European Journal of Radiology 73 (2010) 175–180

Contents lists available at ScienceDirect

European Journal of Radiology

journal homepage: www.elsevier.com/locate/ejrad

Endovascular repair or medical treatment of acute type B aortic dissection? A comparison

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ARTICLE INFO

Article history:
Received 21 April 2008
Received in revised form 21 August 2008
Accepted 23 September 2008

Keywords:
Type B aortic dissection
Stent-graft
Thoracic endovascular aortic repair
Complications
Medical treatment

ABSTRACT

Introduction: The aim of this retrospective study was to compare the outcome of thoracic endovascular aortic repair (TEVAR) to that of medical therapy in patients with acute type B aortic dissection (TBD).
Materials and methods: From July 1996 to April 2008, 88 patients presenting with acute TBD underwent either TEVAR (group A, $n = 38$) or medical therapy (group B, $n = 50$). Indications for TEVAR were intractable pain, aortic branch compromise resulting in end-organ ischemia, rapid aortic dilatation and rupture. Follow-up was performed postinterventionally, at 3, 6 and 12 months and yearly thereafter and included clinical examinations and computed tomography (CT), as well as aortic diameter measurements and assessment of thrombosis.
Results: Mean follow-up was 33 months in group A and 36 months in group B. The overall mortality rate was 23.7% in group A and 24% in group B, where 4 patients died of late aortic rupture. In group A, complications included 9 endoleaks and 4 retrograde type A dissections, 3 patients were converted to open surgery and 2 needed secondary intervention. None of the patients developed paraplegia. In group B, 4 patients were converted to open surgery and 2 to TEVAR. The maximal aortic diameter increased in both groups. Regarding the extent of thrombosis, our analyses showed slightly better overall results after TEVAR, but they also showed a tendency towards approximation between the two groups during follow-up.
Conclusion: TEVAR is a feasible treatment option in acute TBD. However, several serious complications may occur during and after TEVAR and it should therefore be reserved to patients with life-threatening symptoms.

1. Introduction

The treatment of choice for acute type B aortic dissection (TBD) remains a matter of debate in the scientific community. However, there is widespread consensus that in patients with uncomplicated acute TBD, medical therapy is superior to open surgery [1–3]. Persistent pain, aortic dilatation, drug-resistant hypertension or dissection-related complications, such as rupture, impending rupture and end-organ ischemia are clear indications for either surgical intervention or thoracic endovascular aortic repair (TEVAR). As surgery continues to result in high mortality rates [4–7], depending on the complexity of the aortic dissection, TEVAR has been emerging as a less invasive and safe alternative to conventional surgery in patients with aortic disease. For more than a decade now, many authors have documented their experience with TEVAR [8–11]. However, to the best of our knowledge, there is only one study comparing the outcome of TEVAR to that of conservative treatment of TBD [12].

We have recently reported promising mid-term results in patients with acute TBD treated by TEVAR [13] and we have also reported on the results of volumetric measurements in patients with acute TBD treated either by TEVAR or conservatively [14].

The aim of the present retrospective study was to compare the outcome of TEVAR to that of conservative medical therapy in patients with acute TBD, based on data acquired in these two studies. The results were analyzed taking into account the remarkable differences between the two groups in terms of pre-treatment health status.

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doi:10.1016/j.ejrad.2008.09.031

Table 1
Patientïs characteristics.

	Stent	Medical
Patients	38	50
Male	29	37
Female	9	13
Mean age (years)	64 (35–89)	65 (40–84)
ASA I	2	0
ASA II	10	32
ASA III	10	15
ASA IV	0	3
ASA V	16	0
Hypertension	33	36
Diabetes mellitus	3	4
Chronic obstructive pulmonary disease	9	3
Chronic renal failure	5	7
Cardiovascular disease	11	10
Malignancy	1	3
Adipositas	11	8
Smoker	2	6

2. Materials and methods

2.1. Patients

From July 1996 to April 2008, a total of 88 patients were referred to our department with acute TBD. In 38 patients (mean age 64 years, range 35–89), intractable pain ($n = 15$), aortic branch compromise resulting in end-organ ischemia ($n = 15$), rapid aortic dilatation ($n = 5$) and rupture ($n = 3$) necessitated immediate intervention (group A). The decision for TEVAR was made by a team of vascular and cardiovascular surgeons, anesthesiologists and interventional radiologists. Complete written informed consent was obtained from the patients or relatives if the patient was on mechanical ventilation ($n = 6$). In the other 50 patients (mean age 65 years, range 40–84), the team decided on medical treatment (group B). The study was approved by the local ethical review board. Patient data are given in Table 1.

2.2. Aortic branch compromise

The diagnosis of aortic branch compromise was made on the basis of CT scans, angiography and clinical examinations.

In group A, 15 patients presented with aortic branch compromise, 10 of them with dynamic compromise (undulating dissecting lamella obstructing the ostium of a branch vessel), one with static compromise only (static extension of the dissection flap into a visceral artery) and 4 with both, static and dynamic compromise. Static compromise affected the celiac artery in two patients, the celiac and superior mesenteric arteries in one and the renal artery in two patients.

In group B, 5 patients presented with static compromise. It involved the celiac artery and the superior mesenteric artery in one patient, the superior mesenteric artery and the right kidney in two patients, the celiac trunk, the superior mesenteric artery and the right kidney in one patient and the right kidney in another one.

2.3. Diagnostic work-up

The diagnosis of dissection was established on the basis of CT scans and/or angiography and clinical examination. In each patient, contrast-enhanced helical CT with three-dimensional (3D) vascular reconstruction from the apex of the thorax down to the groin was obtained immediately after admission and diagnostic angiography at the time of stent-graft insertion. They provided the needed information on length and diameter of the aortic lesion and anchoring sites, and about involvement of important thoracic and abdominal branches, as well as about the anatomy of the vessels used for access. Up to May 1999, CT examinations were performed using a single-slice SDCT scanner (CT/i, GE Medical Systems, Milwaukee, WI, USA) and from May 1999 to June 2006 using a 4-row multi-slice scanner (Light Speed QX/i, GE Medical Systems, Milwaukee, WI, USA). Since June 2006, data have been acquired from a 64 detector-row MDCT scanner (Light Speed VCT, GE Medical Systems, Milwaukee, WI, USA) using a slice thickness of 1.25 mm with pitch 0.98 and a reconstruction interval of 0.6 mm in the standard reconstruction kernel. Scans were obtained using 120–150 ml of a nonionic contrast agent (Ultravist, Schering, Berlin, Germany) administered at a concentration of 300–370 mg I/ml and a flow rate of 4 ml/sec. The raw data were transferred to an independent workstation (Sun Ultra 60, Sun Microsystems, Mountain View, CA, USA) running the Advantage Windows software (AW4.0, GE Medical Systems, Milwaukee, WI, USA) for calculating MIP, curved reformats (Fig. 1) or 3D reconstructions. We do not routinely use ECG-gating.

2.4. Stent-grafts

Dimensions of the stent-grafts (SGs) were determined on the basis of the findings on preinterventional contrast-enhanced helical CT scans and angiography.

In group A, 48 Talent SGs (Medtronic AVE, Sunrise, FL, USA; length 100–150 mm, diameter 34–46 mm), 5 Excluder SGs (WL Gore and Associates, Inc., Flagstaff, AZ, USA; length 150 mm, diameter 40 mm) and 13 TAG SGs (WL Gore and Associates, Inc., Flagstaff, AZ, USA; length 100–200 mm, diameter 31–37 mm) were implanted during primary interventions. 18 patients received one SG each, 14 patients 2 SGs each, 4 patients 3 SGs each, and 2 patients 4 SGs each. 2 Talent SGs were implanted during secondary interventions.

In group B, two patients had to be converted to TEVAR. They received 1 Excluder SG (length 150 mm, diameter 37 mm) and three Talent SGs (length 160 mm, diameter 36–40 mm) respectively.

All patients were treated with SGs from our emergency kit containing Talent, Excluder and TAG SGs of various sizes (Talent: 80–167 mm in length and 16–46 mm in diameter, Excluder: 150–200 mm in length and 31–40 in diameter and TAG: 150–200 mm in length and 31–37 mm in diameter). Oversizing was 10–20%.

2.5. Proximal landing zones

In 13 patients, the origin of the left subclavian artery had to be completely crossed with the covered portion of the SG. In 7 of them, the origin of the left carotid artery was crossed with the bare springs of the SG. In 16 patients, the origin of the left subclavian artery was crossed with the bare springs of the SG and in 9 patients, it remained uncovered.

2.6. Technique

Between October 1998 and April 2008, all procedures were performed in an angiographic suite (Integris BV 3000, Phillips, Eindhoven, Netherlands, Europe) under fluoroscopic guidance by a team of experienced radiologists, anesthesiologists, cardiovascular and vascular surgeons, using general anesthesia and intubation. Preinterventionally, each patient received antibiotic prophylaxis consisting of a single shot of a Cefuroxim (1.5 g Curocef per patient, Glaxo Smith Kline, GB). The technical details of SG placement were described previously [8]. They have remained unchanged except for balloon dilatation, which we now avoid, as we observed a strong association between this technique and the occurrence of retrograde type A dissections [13,15].

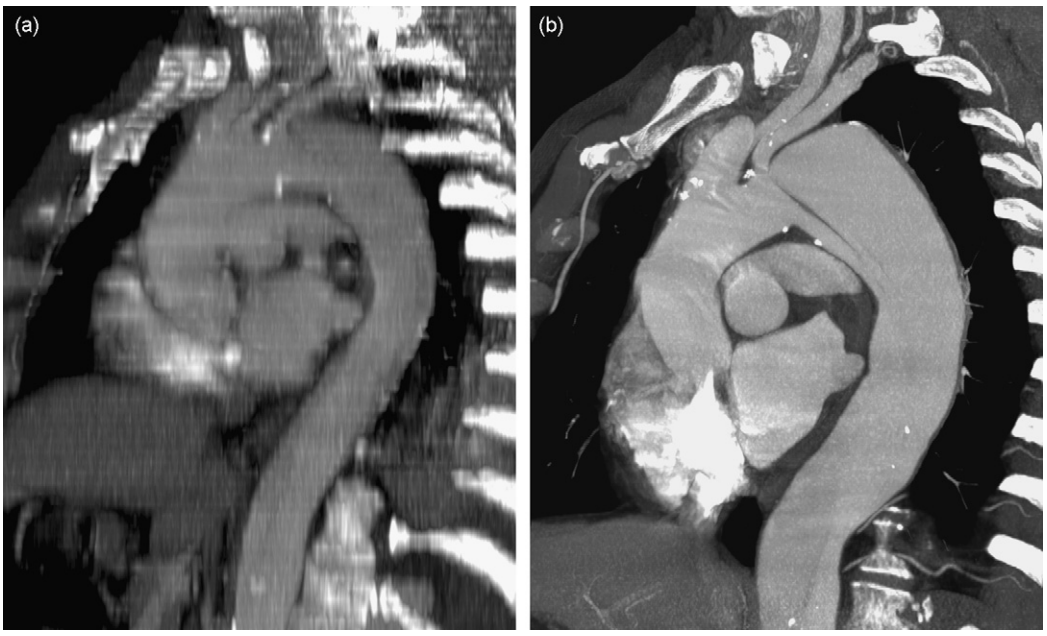

Fig. 1. (a) MIP shows a medically treated patient at the time of admission. (b) MIP shows the same medically treated patient 4 years after diagnosis of TBD. The descending thoracic aorta has enlarged over time.

We did not perform spinal cord protection, such as spinal fluid drainage or hypothermia. Neither did we perform preinterventional transposition of aortic branch vessels nor preinterventional examination of spinal cord vascularization. However, after TEVAR, all patients were transferred to the intermediate care unit, where neurologic examinations were performed.

2.7. Follow-up protocol

The follow-up protocol included clinical examination and enhanced spiral CT scans, which were performed postinterventionally, at 3, 6 and 12 months and yearly thereafter. Additional scans and/or digital subtraction angiography (DSA) were performed as needed to address specific problems.

2.8. Assessment of thrombosis and diameter measurements

The maximal aortic diameter was measured by hand on axial CT slices. To avoid overestimation of the aortic diameter due to tortuosity, diametric measurements were obtained perpendicular to the maximal aortic diameter.

Thrombus assessment was performed in both groups over the entire observation period. Missing thrombosis was identified by opacification of the FTL by contrast agent, partial thrombosis by the presence of contrast and thrombus in the FTL and complete thrombosis by the absence of contrast and complete occlusion of the FTL by a thrombus.

In both patient groups, aortic diameters and thrombus formation were evaluated by comparing the first available CT scan (the preinterventional scan in group A, and that obtained on admission in group B) to the last available scan in the respective patients, regardless of the length of follow-up.

2.9. Statistics

Cumulative overall survival rates of the two groups were evaluated using the Kaplan–Meier life table method and log-rank test. A p value of less than 0.05 was considered statistically significant. These statistics were conducted using SPSS 15.0.

3. Results

Mean follow-up in group A was 33 months (range, 0–97 months). Three patients were lost to follow-up, because they returned to their home-countries. Mean follow-up in group B was 36 months (range, 0–122 months). Four patients were lost to follow-up. All of them were in severely reduced medical condition and not able to come to the follow-up examinations.

3.1. Survival rates

In group A, survival rates were 81.5% at 1 year, 81.5% at two and 69.0% at 5 years. In group B, they were 88.5% at 1 year, 79.6% at two and 70.2% at 5 years (Fig. 2). The difference between the two groups was statistically not significant ($p = 0.86$). The overall mortality rate was 23.7% in group A and 24% in group B.

3.1.1. 30-Day mortality

In group A, the 30-day mortality rate was 13.1% (5 patients). Two patients died within 12 h after TEVAR of hemorrhagic shock due to aortic rupture, one within 24 h of multiorgan failure secondary to aortic rupture and another one within 6 days of thrombo-embolic cerebral infarction. The fifth patient died of sepsis 34 days after TEVAR.

In group B, the 30-day mortality rate was 6.0% (3 patients). One patient died of multiorgan failure due to static compromise of the celiac trunk and the superior mesenteric artery 3 days after admission. One patient died of cerebral ischemia 5 days and the other one of sepsis 12 days after admission.

3.1.2. Late mortality

In group A, four more deaths occurred during follow-up. One patient died of sepsis 15 days after conversion to open surgery and 51 months after TEVAR, one patient died of pneumonia 50 months

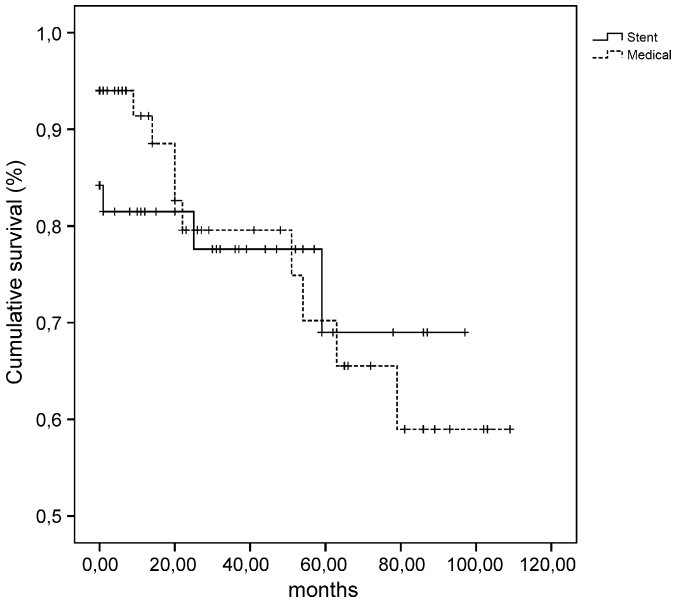

Fig. 2. Kaplan–Meier estimates of survival of patients treated for TBD, either by TEVAR or conservatively.

after TEVAR and 2 patients of myocardial infarction, one of them 25 months after TEVAR and the other one 5 months after emergency replacement of the aortic arch (dissection Stanford type A) and 65 months after TEVAR.

In group B, 9 more deaths occurred during follow-up. Four patients died of aortic rupture, 5, 6, 51 and 55 months after admission respectively, one patient died of retrograde Type A dissection and cardiac tamponade 9 months after diagnosis of TBD, one of cerebral ischemia 20 months after admission, two patients of cardiac failure 14 and 63 months after admission respectively and one of cerebral cancer 20 months after admission.

3.2. Complications in group A

A total of 4 patients developed retrograde type A dissection 3, 26, 28 days and 59 months after TEVAR respectively necessitating conversion to open surgery in 3 of them. In one patient, in whom a Talent SG was implanted, the dissection was detected after TEVAR on the postinterventional CT scan, and we assumed that the dissection was caused by forced balloon dilatation during the intervention to seal early endoleak type I. In another patient, the intraoperative situs revealed that a strut of the Excluder SG had prolapsed into the new entry tear. In the remaining two patients, the entry tear was located in the distal part of the ascending aorta and the intraoperative situs did not show any relationship between the struts of the Talent SG and the dissection 2 patients developed late type III endoleak 21 and 62 months after TEVAR respectively, requiring secondary TEVAR to seal the leaks. 3 Patients had early proximal type I endoleak and 2 patients late proximal type I endoleak 20 and 72 months after TEVAR, respectively. In 2 patients, postoperative CT studies showed type II endoleak via the left subclavian artery (Fig. 3).

Two of the patients with dynamic compromise needed additional fenestration of the abdominal intimal flap. The remaining patients including those with static compromise needed no further therapy, because serum lactate levels were normal. There were no patients with spinal cord ischemia.

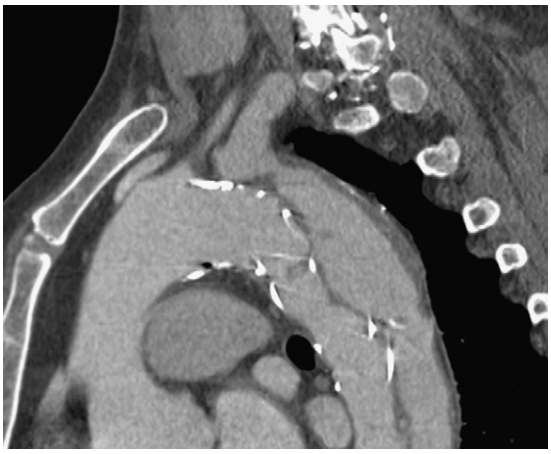

Fig. 3. MIP visualizes a type II endoleak via the left subclavian artery.

3.3. Complications in group B

In this group, 4 patients had to be converted to open surgery, 1, 20, 22 and 51 months after the first imaging respectively. In all of them, CT scans, which were performed because of sudden onset of intractable pain, revealed substantial aortic dilatation, especially in the distal part of the descending aorta, involving the origin of the celiac trunk and the superior mesenteric artery.

Two patients had to be converted to TEVAR due to rapid aortic dilatation, one of them 4 and the other one 11 months after the first imaging. As the patients were in severe clinical condition (ASA4), we preferred TEVAR to open surgery.

Two patients with static compromise were treated by adjunctive deployment of a SG within the true lumen of the affected vessel. One of them, who presented with static compromise involving the superior mesenteric artery and the right kidney, was treated by adjunctive deployment of a SG within the true lumen of the superior mesenteric artery.

In the other one, in whom the right kidney was affected, an additional SG was placed within the true lumen of the renal artery. In the remaining patients, no further therapy was necessary.

The patient, with involvement of the celiac trunk and the superior mesenteric artery died 3 days after admission because of multiorgan failure.

3.4. Thrombosis

In group A, thrombosis was assessed in 35 patients. Preinterventional CT scans showed complete thrombosis of the FTL in none of the patients and partial thrombosis in 13 patients (37.1%). In 22 patients (62.9%), thrombosis was completely missing. The last available CT scans showed complete thrombosis in 2 patients (5.7%), partial thrombosis in 30 patients (85.7%) and missing thrombosis in 3 patients (8.6%).

In group B, thrombosis was assessed in 41 patients. Evaluation of the CT scans performed on admission showed complete thrombosis of the FTL in none of the patients, partial thrombosis in 18 patients (43.9%) and missing thrombosis in 23 patients (56.1%). The last available CT scans showed complete thrombosis in 11 patients (26.8%), partial thrombosis in 18 patients (43.9%) and missing thrombosis in 12 patients (29.3%).

3.5. Maximal diameter of the thoracic aorta

In group A, the maximal diameter of the thoracic aorta was 42 mm (32–61 mm) preinterventionnally and 46 mm (31–84 mm)

on the last available CT scans. In group B, it was 41 mm (30–62 mm) on admission and 45 mm (28–71 mm) on the last available CT scans.

4. Discussion

There is widespread consensus that in patients with uncomplicated acute TBD, medical therapy is superior to open surgery. However, patients with life-threatening symptoms including rupture, impending rupture, end-organ ischemia, rapid aortic dilatation and refractory pain require immediate intervention. In these patients, TEVAR offers a less invasive and safe alternative to open surgical repair, which is still associated with high morbidity [4] and mortality rates [4–7]. However, various serious complications can occur during and after TEVAR. The most common complications include endoleaks, retrograde type A dissection, cerebral ischemia and paraplegia [16–19]. Therefore, recent reports have scrutinized indications for TEVAR of TBD [18,20,21].

In the literature, 30-day mortality rates after TEVAR of TBD are ranging from 3.2% to 13% [12,19,20,22]. A combined series including 62 patients with acute and 69 patients with chronic TBD, reported a 30-day mortality of 8.4% [23]. In medically treated patients with acute TBD, Hsu et al. and Hata et al. documented low in-hospital mortality rates (0% and 3.8% respectively) [2,24], whereas an IRAD study including 384 patients reported an in-hospital mortality of 9.6% [6]. Our results, which show a 30-day mortality rate of 13.1% in group A and 6.0% in group B, are comparable to those of other study groups. In our series, perioperative mortality was higher in group A than in group B, whereas late mortality was higher in group B than in group A. This can be explained by the remarkable difference between the two groups in terms of pre-treatment health status. The patients who were selected for TEVAR presented with life-threatening complications and most of them were in poor general condition, whereas the patients assigned to medical therapy had uncomplicated TBD and were in comparably better general condition.

Survival rates after TEVAR have been reported to range from 81% to 89.9% at 1 year and from 74% to 88.8% at 2 years [9,12,22,25–27]. Dialetto et al. reported a rate of 86.3% at 5 years [12]. In medically treated patients, survival rates range from 81.6% to 98.9% at 1 year and from 71% to 98.9% at 5 years [1,3,12,24,28]. In our series, similar rates were achieved at 1 and 2 years. At 5 years, our results are in agreement with international standards in group B (70.2%) but not in group A. Compared to the study of Dialetto et al., our results are less favorable (69.0%). However, our series included more patients (39 versus 28) and the mean age of our patients was higher (67 versus 60.8) [12]. A comparison of the survival rates at 5 years, showed no significant difference between groups A and B. However, relating to both, overall mortality and 5-year survival rates our results slightly favor the strategy of TEVAR, when considering the remarkable difference in terms of pre-treatment health status.

The most serious complications in patients with TBD are aortic rupture end-organ ischemia. However, as stated by Estrera et al., rupture is relatively rare at initial presentation [1], whereas end-organ ischemia due to static or dynamic compromise of branch vessels is a common cause of primary morbidity and/or mortality [1,29]. Dynamic compromise can be successfully treated by SG placement. In our study, it restored blood-flow to the compromised vessels in 12 of the 14 patients with dynamic compromise, the other two needed additional balloon fenestration of the dissection flap.

Static compromise can be treated by adjunctive deployment of a stent within the true lumen of the affected branch [9]. Two patients in group B were successfully treated by adjunctive deployment of a stent within the true lumen of the right renal artery and the superior mesenteric artery respectively. None of the other patients presenting with static compromise required placement of adjunctive SGs. Those with involvement of visceral arteries showed sufficient collateralization and none of them developed irreversible end-organ ischemia and those with involvement of the kidneys showed only slightly elevated lactate levels and renal function parameters.

Aortic rupture has been shown to be an important predictor of early mortality in aortic dissection [5]. In group A, three patients had aortic rupture at initial presentation. In all of them, SG placement was performed successfully. However, unfortunately, they all died within 12 and 24 h after TEVAR respectively due to hemorrhagic shock or multiorgan failure secondary to aortic rupture. As in all three patients the rupture was located in the descending aorta and as there was residual back-flow across a large abdominal reentry, the patients could not be stabilized. In group B, there were 4 late aortic ruptures, whereas no late aortic rupture occurred in group A, which shows that TEVAR can stabilize the thoracic aorta and prevent late aortic rupture.

Thrombosis of the false lumen has been reported to be a good indicator of a favorable long-term prognosis [30] and Bernard et al. observed that false lumen patency in the descending aorta is a predictive factor for late mortality [31]. In our study, most patients in group A, showed missing thrombosis on admission and partial thrombosis at the last follow-up, while most patients in group B, showed missing or partial thrombosis on admission and partial or complete thrombosis at the last follow-up. However, in 29.3% of the patients in group B, the FTL remained patent from the beginning of the observation period to the end. These results show that TEVAR promotes thrombus formation, but they also show a tendency towards approximation between the two groups during follow-up.

Diameter measurements are a widely used and valuable tool in the follow-up of aortic diseases [2,3,17,19,32,33]. Several authors reported that the maximal aortic diameter increased or remained stable in conservatively treated patients [2,3], while it decreased in patients treated by TEVAR [17,19]. Our results showed increase in thoracic aortic diameter in both patient groups. This may be due to the comparably long follow-up period in our study.

Several authors have documented a strong association between paraplegia and the length of SG coverage and Mitchell et al. stated that the incidence of paraplegia increases with the number of occluded intercostal arteries, ranging from 0% to 2.9% [34]. Herold et al. proposed a maximum SG coverage of 200 mm in order to minimize the risk of paraplegia [35]. Conversely, it is well known that long—distance grafting should be preferred, especially in cases of multiple entry points in the thoracic aorta [18,36]. None of our patients developed paraplegia, although SG coverage exceeded 200 mm in 13 of them. As the diameter of the descending aorta often differs from that of the aortic arch and as full deployment of the SG within the descending aorta can take some time, it may be possible that the vascularization of the spinal cord adapts to the new situation by collateralization.

We do not routinely use ECG-gating. Therefore, it may be possible that we missed entry points on the preinterventional CT scans. It is well known that multiple entry points in the intimal flap of the thoracic and/or in the abdominal region limit successful TEVAR, as false lumen patency and/or lack of aortic remodeling may occur [18,37,38]. However, as we routinely evaluate the outcome of TEVAR during the intervention, gaps in the intimal flap missed on the preinterventional CT scans will be detected during the procedure. In such cases, we adapt the length of SG coverage to the new situation.

Persistence of severe pain in medically treated patients may be an indicator of early progression to rupture [25]. Nevertheless, based on our growing experience, which has shown that serious complications may emerge a long time after the intervention, we

have changed our strategy and now prefer close monitoring of these patients to immediate TEVAR.

In their study comparing TEVAR of TBD to medical treatment, Dialetto et al. concluded that TEVAR is superior to conservative therapy and seems to favorably change the natural course of the disease [12]. Based on our results with a larger patient population (38 versus 28 patients treated by SG placement) and a longer follow-up (mean follow-up 33 months versus 18 months), we think that TEVAR is the treatment of choice in patients with life-threatening symptoms necessitating immediate intervention, such as rupture, impending rupture and aortic branch compromise. These complications can be treated successfully in most patients. However, considering the risk of various serious complications associated with TEVAR, it should be reserved to these patients only. However, in view of the small, non-randomized set of patients in this retrospective study and because of the heterogeneity of the two patient groups in terms of pre-treatment health status, further studies will be of great interest.

Conflict of interest

None.

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