Type B aortic dissections: treating the many to benefit the few?

Dittmar Böckler, Alexander Hyhlik-Dürr, Maani Hakimi, Tim Frederik Weber, Philipp Geisbüsch

Angaben zur Veröffentlichung / Publication details:

Böckler, Dittmar, Alexander Hyhlik-Dürr, Maani Hakimi, Tim Frederik Weber, and Philipp Geisbüsch. 2009. "Type B aortic dissections: treating the many to benefit the few?" *Journal of Endovascular Therapy* 16 (Supplement 1): 80–90. https://doi.org/10.1583/08-2611.1.

Type B Aortic Dissections: Treating the Many to Benefit the Few?

Dittmar Böckler, MD, PhD[1]; Alexander Hyhlik-Dürr, MD[1]; Maani Hakimi[1]; Tim Frederik Weber, MD[2]; and Philipp Geisbüsch, MD[1]

Departments of [1]Vascular and Endovascular Surgery and [2]Diagnostic and Interventional Radiology, Ruprecht-Karls University Heidelberg, Germany.

Purpose: It is now more than a decade since aortic stent-grafts were introduced clinically to provide a less invasive and potentially less harmful therapeutic option to treat type B aortic dissections. However, recent publications on best medical treatment and quality of life in patients with chronic type B dissection support conservative treatment due to the low incidence of aneurysm formation, rupture, and disease-related complications. Against this backdrop, we analyzed our experiences and now discuss whether the availability of endografts allowed us to change indications toward a more aggressive endovascular approach to acute and non-complex type B dissections, seeking to determine which patients we should treat and which ones we should observe.
Methods: Between 1997 and 2008 in our institution in Heidelberg, we treated 172 patients with acute and chronic type B dissections, most (n=118, 69%) conservatively. However, 54 patients (40 men; mean age 57 years, range 30–82) underwent endovascular repair; 43% (n=23) were emergency cases. Patients were followed periodically with computed tomographic angiography.
Results: Correct stent-graft deployment was achieved in 50 (93%) patients; the left subclavian artery was intentionally covered in 30 (55%) cases. Two carotid-subclavian bypass grafts were performed at the time of the endovascular repair due to partial coverage of the left common carotid artery. The perioperative complication rate was 19% (n=10), but there were no neurological sequelae. The 30-day mortality rate was 11% (n=6). Over a mean 32.1±25 months, 4 other patients died (18.5% overall mortality rate); survival estimates by Kaplan-Meier analysis were 80.4% and 66.1% after 1 and 5 years, respectively. Complete false lumen thrombosis was observed in 32 (60%) and a persisting completely patent false lumen in 3. The aortic expansion rate was 31% (17/54) overall. No difference was found between acute and chronic dissections in terms of survival (p=0.247).
Conclusion: Despite a minimally invasive approach, complication and mortality rates for endovascular therapy of type B aortic dissections are considerable. Endografting is limited to symptomatic patients and those with chronic large aneurysmal expansion. At this stage in stent-graft development, asymptomatic patients benefit more from conservative treatment.

Key words: thoracic aorta, dissection, thoracic endovascular aortic repair, stent-graft, outcome analysis, mortality

The authors have no commercial, proprietary, or financial interest in any products or companies described in this article.

Address for correspondence and reprints: Dittmar Böckler, MD, PhD, Department of Vascular and Endovascular Surgery, University of Heidelberg, Im Neuenheimer Feld 110, 69120 Heidelberg, Germany. E-mail: *dittmar.boeckler@med.uni-heidelberg.de*

The clinical spectrum of acute type B thoracic aortic dissection extends from asymptomatic, uncomplicated dissection to life-threatening organ ischemia and impending rupture. Therefore, type B aortic dissection is a condition burdened with high mortality and a dilemma concerning its correct management. According to the population-based longitudinal study by Meszaros et al.,[1] mortality of untreated acute aortic dissection (type A/B) is 1% to 3% per hour and 25% in the 24 hours after the initial presentation; after the first and second week, the rates are 70% and 80%, respectively. The International Registry of Acute Aortic Dissection (IRAD) found an overall 27.4% mortality of aortic dissections, confirming the poor natural history of this pathology.

Despite improving surgical techniques and accumulated experience, open surgical repair still is accompanied by high morbidity and mortality rates up to 27% in elective procedures, over 50% under emergency conditions.[2,3] The advent of endovascular prostheses to treat descending thoracic aortic lesions offered an alternative approach in patients with dissections and severe comorbidities who were poor candidates for open surgery.[4,5] It is now more than a decade since these aortic stent-grafts were introduced clinically to provide this less invasive and potentially less harmful therapeutic option to treat aortic dissections.[4,5] While the feasibility and technical success rate of thoracic endovascular aortic repair (TEVAR) in type B aortic dissection was confirmed in a meta-analysis published by Eggebrecht et al.[6] 2 years ago, this approach was associated with a complication rate of 11%. These results highlight the technical challenges of treating thoracic aortic dissection endovascularily, one of the unresolved issues in the field of endovascular techniques and technologies.

The development of new stent-grafts has expanded the spectrum of therapy for dissections. Both feasibility and safety have been reported by several centers, and initial enthusiasm has triggered a spreading, potentially unguarded preference for stent-grafting based on an 80% midterm survival compared to classic open surgery. Yet even today, we have no randomized study to confirm any consistent prognostic benefit of endovascular versus open treatment of acute type B dissection. Many investigators have discussed this ongoing conflict and questioned patient selection in the treatment of aortic type B dissections. A commentary by Nienaber et al.[7] brought the problem into focus: ''to stent or not to stent aortic dissection: good news for a chosen few, but who?''

Furthermore, recent publications on best medical treatment and quality of life in patients with chronic type B dissections support conservative treatment due to the low incidence of aneurysm formation, rupture, and disease-related complications.[8,9] Against this background, we analyze our own results and discuss whether the availability of endografts has allowed us to change indications toward a more aggressive endovascular approach to acute and non-complex type B dissections.[10] Finally, we try to answer the question: ''Which patients should and which should not be treated by endovascular means in order to benefit the most and to harm as few as possible?''

THE HEIDELBERG EXPERIENCE

Patient Population

Between January 1997 and October 2008, a total of 225 patients underwent TEVAR in our institution. In this time period, 172 consecutive patients presented with acute and chronic Stanford type B dissections. In all cases, diagnosis was confirmed with computed tomography (CT) or magnetic resonance angiography (MRA). Of these 172 patients, 118 (68.6%) were treated conservatively and were followed routinely by CT scan on an annual basis. Fifty-four patients (40 men; mean age 57 years, range 30–82) underwent TEVAR for acute or chronic type B dissection (patients with retrograde involvement of the arch and ascending aorta were excluded from this analysis)

More than one third of all the cases (n=23; 43%) were emergency interventions in acute patients who experienced some degree of chest pain as the first presenting symptom. Twenty-three patients developed visceral, renal, or leg ischemia with true lumen collapse; 4 patients suffered from rupture of

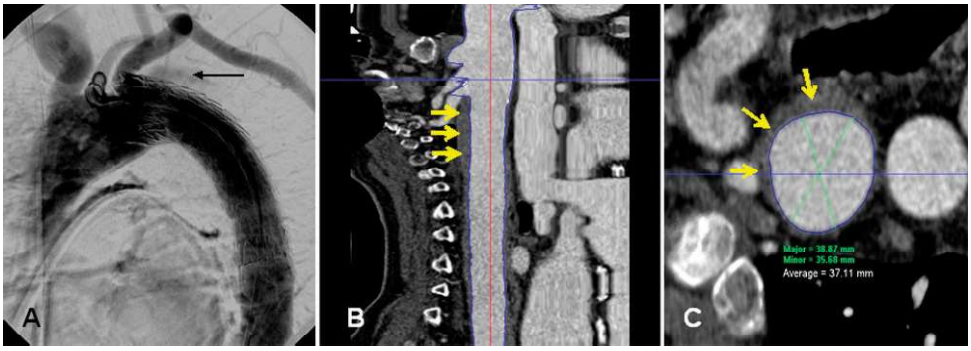

Figure 1 ◆ (**A**) Completion angiography after stent-graft deployment shows contrast enhancement at the outer curve (arrow). (**B**) Centerline postprocessing demonstrates wall thickening and intramural hematoma reaching the left subclavian artery (arrows). (**C**) This orthogonal view of a CTA reconstruction indicates aortic arch involvement representing retrograde dissection.

the false lumen (1 presenting with aortobronchial fistula 2 years after the initial uncomplicated dissection). All surgical procedures were performed in an operating theater equipped with fluoroscopic and angiographic capabilities (Series 9800; OEC Medical Systems, Inc., Salt Lake City, UT, USA) and a carbon fiber operating table. The exact procedure protocol has been published.[11] Eight patients received 2 stent-grafts and another received 3 devices for a total of 76 stent-grafts implanted: 63 Excluder Thoracic Endoprostheses (TAG; W.L. Gore & Associates, Flagstaff, AZ, USA), 11 Talent endoluminal stent-grafts (Medtronic Vascular, Santa Rosa, CA, USA), and 2 EndoFit Thoracic Endoprostheses (LeMaitre Vascular, Inc., Burlington, MA, USA) in lengths from 100 to 220 mm and diameters between 28 and 40 mm. The median length of aorta covered by the stent-graft was 169 mm (range 100–220).

Definitions for technical and clinical success were in accord with the reporting standards for endovascular aortic aneurysm repair.[12] Follow-up included postoperative CT angiography (CTA) before discharge; clinical examination, plain chest radiography, and CTA/MRA were performed at 6 and 12 months postoperatively and annually thereafter.

Outcomes

Technical success was achieved in 50 (93%) patients. Stent-grafts were deployed successfully in all but 1 case in which device tracking into the true lumen was difficult; the patient was successfully treated in a second operation 4 days later. The left subclavian artery (LSA) was overstented in 30 (55.5%) patients to secure adequate proximal fixation and device attachment. Two of these patients required an immediate carotid-subclavian bypass graft after inadvertent partial coverage of the left common carotid artery. Another patient suffered from relevant subclavian steal syndrome and received a bypass graft 2 days later. No primary conversion or other simultaneous intervention was needed.

In the 30-day perioperative period, the complication rate was 19% [2 cardiac, 3 pulmonary, 2 retrograde dissections (Fig. 1), 1 acute renal failure, 1 subclavian steal syndrome, and 1 infrarenal rupture on day 5]. No paraplegia or stroke was observed. Additional reinterventions were undertaken in 7 (13.0%): 6 (26.1%) acute and 1 (3.1%) chronic patients. Five of 6 patients with true lumen collapse and mesenteric ischemia showed a drop in lactate and liver enzymes 12 hours after endovascular entry occlusion; the values returned to normal in an average of 4.5 days after the intervention.

Six (11.1%) patients died in the 30-day period, all acute cases. Multiorgan failure (n=4), the leading cause of death, was observed predominantly in patients with continuing visceral ischemia. A 50-year-old man with true lumen collapse and a 2-day

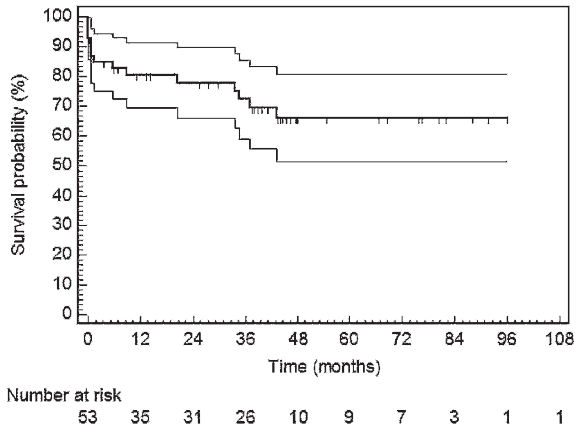

Figure 2 ◆ Kaplan-Meier estimate of actuarial survival in 53 patients (1 lost to follow-up) treated by TEVAR for acute and chronic type B aortic dissections. Gray lines represent the 95% confidence interval.

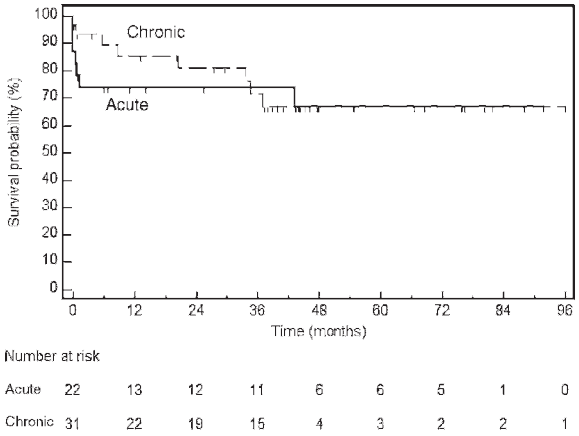

Figure 3 ◆ Kaplan-Meier estimates of actuarial survival in patients treated with TEVAR for acute symptomatic (n=22, 1 lost of follow up); solid line) and chronic aortic type B dissections (n=31, dashed line). P=0.248 by log-rank test.

history of abdominal pain was treated successfully, with immediate re-expansion of the true lumen on angiography after TEVAR; however, he developed colon ischemia and multiorgan failure and died 2 days after subtotal colectomy. A 48-year-old man with a clinically asymptomatic abdomen but occlusion of the celiac trunk and superior mesenteric artery developed severe colon ischemia and died despite early entry closure within 48 hours.

Over a mean follow-up period of 32.1±25 months (range 1–95), 1 patient (acute) was lost to follow-up. With 4 more deaths after the 30-day perioperative period, the overall mortality rate was 18.5% (n=10). The survival estimates by Kaplan-Meier analysis (Fig. 2) at 1, 3, and 5 years after endografting were 80.4%±5.6%, 72.3%±6.7%, and 66.1%±7.5%, respectively. There was no significant difference (p=0.247) in survival rates between acute and chronic type B dissections (Fig. 3).

Among the 4 late deaths, 1 woman died from infrarenal false lumen rupture while she was scheduled for secondary conversion. A type II endoleak and a late proximal type I endoleak had been diagnosed on the postoperative CT, but she refused secondary intervention initially. Although she finally was treated with a proximal extension graft, the proximal endoleak persisted due to a diameter mismatch of the endograft and the aortic arch.

A 78-year-old man with aortobronchial fistula was treated by transfemoral coil embolization of the false lumen to avoid conversion in this high-risk patient; he died 2 weeks later due to another massive hemorrhage. One patient sustained rupture from the false lumen with aortobronchial fistula at 18 months; coil embolization of the false lumen was unsuccessful in controlling the repetitive hemoptysis after endograft placement. Another patient suffered from infrarenal aortic rupture. Freedom from rupture was 97%, 90%, and 80% at 1, 2, and 3 years, respectively.

Thrombosis of the thoracic aortic false lumen was observed in 32 (59.3%) patients, while a completely patent false lumen was seen in 3. Partial false lumen thrombosis was induced in the other 7 (13%), with retrograde perfusion sustained by distal re-entries. The overall expansion rate was 31.5% (17/54). Five patients (3 chronic, 2 acute) developed >50-mm aortic expansion over a 2-year period; 4 underwent open abdominal aortic bifurcated bypass grafting. One woman with Marfan syndrome developed progressive dilatation of the abdominal aorta after valve replacement and TEVAR of the distal arch. In a staged hybrid procedure, she had extra-anatomical mesenteric and renal revascularization and endografting of the thoracoabdominal aorta. Two more patients received aortobiiliac bypass grafting 3 years after TEVAR.

DISCUSSION

Our own results with TEVAR in aortic type B dissections confirm the international experience of many centers. An 11% 30-day mortality rate and 19% morbidity are remarkable for a so-called minimally invasive approach, especially in acute complicated dissections. There was no difference in long-term survival between acute and chronic aortic dissection patients.

For many years, conservative management has been the treatment of choice for acute, uncomplicated Stanford type B dissections,[13,14] achieving good results when combined with strict antihypertensive medication, intensive medical observation, and CTA or MRA surveillance.[15–18] In particular, the effectiveness of β-blockers was proven years ago.[19] Early diagnosis, close observation in the intensive care unit, and aggressive pharmacological therapy helped to decrease mortality significantly, both in the conservative group and in the group undergoing surgical intervention.[19]

Regardless of treatment, complications develop in 30% to 40% of the cases over time. Long-term data suggest that up to 40% of asymptomatic patients will succumb to an aortic-related event or require a direct aortic reintervention over a 7-year period. Several retrospective studies with univariate and multivariate analyses have been performed to define those initial factors of the acute phase that determine the clinical course and long-term prognosis.[20–22] Patient age, persistent pain, and visceral malperfusion were found to be significant independent predictors of early surgical intervention. Paraplegia, lower limb ischemia, pleural effusion, and aortic diameter >45 mm were also unfavorable factors for the prognosis.

Umana et al.[22] treated 189 type B dissections over a period of 36 years and operated conventionally on 67 patients in the acute phase; they found that ''shock'' [hazard ratio (HR) = 14.4] and ''ischemia of the visceral organs'' (HR=10.9) were pivotal factors in the prognosis of their patients. Medical and surgical treatment strategies attained similar results, whereas the postoperative mortality decreased over the years from 57% (1963–1969) to 27% (1990–1999).[22]

In a multicenter study of 465 patients treated with open repair, Hagan et al.[23] reported a 3 times higher mortality rate (31.4%) after open surgery compared to the conservative patient group (10.7%). Lansmann and colleagues,[24] however, had a mortality of 0% with a fairly high associated complication rate of 47% in 34 patients with symptomatic type B dissection who had undergone surgery in the acute phase.

Aortic treatment paradigms have evolved with the development of endovascular stent-graft repair of TAAs, first published by Dake et al.[25] in 1994. The same authors published one of the first studies describing midterm results (4.5 years) of this approach to TAA.[26] Since that time, many authors have published results on TEVAR for the treatment of descending thoracic aortic dissections.[4,5,27–34] Dake et al.[5] initially reported a series of 19 acute symptomatic dissections with 16% early mortality, which is in line with the 11% in our own series. Early mortality in a study from Palma et al.[31] was 6% in 58 patients; of those, 35 were acute but asymptomatic. The excellent results achieved in 82 patients studied by Rehders and Nienaber[35] are, like other series, difficult to interpret due to a lack of differentiation between acute and chronic dissections, ruptures, and mesenteric ischemia.

The potential benefits of endovascular surgery for dissections include low access trauma, minimal loss of blood, no need for aortic cross-clamping, early recovery from perioperative stress, shorter intensive care unit and hospital stays, decreased morbidity and mortality, and (eventually) cost-effectiveness. Endovascular techniques include the occlusion of the entry by an endograft or the percutaneous fenestration of the dissection membrane with stenting of occluded aortic branches in the case of true lumen collapse and organ ischemia.

Although there is no long-term data to support definitive evidence, some authors are now proposing TEVAR as an alternative to surgery in patients with type B dissections. In 1999, Nienaber et al.[4] published a small study that was the first prospective compar-

ison of transluminal and open surgery; they concluded that stent-graft repair is a viable therapeutic option in dissections. Nevertheless, in our opinion, the effectiveness of stent grafting to prevent aortic rupture, chronic aortic expansion, or end-organ ischemia still has to be determined, and many questions remain:

- Is prophylactic scaffolding of a dissected aorta by a stent-graft successful in avoiding future complications, such as false lumen aneurysm, true lumen collapse, and late rupture?
- Do we have sufficient scientific data to support the use of prophylactic endovascular remodeling in patients who are stable and asymptomatic in order to prevent so called late complications, when the incidence of these sequelae is unknown or at least uncertain?
- Do we already have the ideal dissection-specific device and what does it look like (tapered, bare segment, radial force)?
- Are there accepted independent risk factors for adverse outcome to aid in our identification of patients at risk in the acute phase?
- What is the optimal timing for intervention?

Numerous retrospective studies with univariate and multivariate data analysis have been carried out to define initial factors that determine the clinical course and long-term prognosis of acute type B dissection.[17,36] Asymptomatic patients with an open false lumen, an initially large aortic diameter >40 mm, and an ongoing ''open entry'' have an unfavorable prognosis. The 1- and 5-year survival rates of asymptomatic patients under conservative medication amounts to 94% and 86%, respectively. A comparison of these patients, however, to others who have undergone surgical therapy is not possible due to a negative selection bias of operated patients. A recent prospective study of medically treated acute type B dissections confirmed a low incidence of aneurysm formation and rupture during the chronic phase over a mean 6.6-year follow-up.[8] Good quality of life, which differs little from the normal population, supports conservative treatment in asymptomatic patients.[9]

Preliminary work suggests that the timing of treatment plays an important role in the success of endovascular repair.[37] Bortone et al.[38] concluded in a small retrospective study in 21 patients that immediate endovascular repair offers important advantages, such as avoidance of high-risk procedures and postoperative complications with shorter hospital stay. In 8 (61.5%) of 13 patients with delayed treatment, complicated progression of dissection made stent-graft deployment impossible. On the other hand, we know from open surgery the fragility and vulnerability of the acute dissected aortic wall. Most vascular surgeons, therefore, wait to conduct the endovascular repair at some point in the subacute phase (>14 days after onset) if possible.

Morphological Indications and Contraindications

A sufficient proximal fixation site of at least 2 cm is mandatory to prevent type I endoleak with stent-graft implantations in the thoracic aorta.[39] To achieve this, it is often necessary to cover the LSA or perform a hybrid procedure with debranching of the supra-aortic vessels. In our patient cohort, we covered the LSA in 30 out of 54 (56%) patients. One patient developed clinically moderate dizziness and was observed without secondary intervention. Despite extending the landing zone, 2 patients still developed a proximal type I endoleak due to incorrect endograft sizing and inflexible devices that did not properly seal at the proximal anchoring zone. Proximal tight fixation in the distal arch is therefore mandatory. The aortic arch should be free from disease, less than 44 mm in diameter, and without mural thrombus and atherosclerotic plaques to achieve good sealing.

The presence of intramural thrombus in the proximal landing zone and a dissection membrane in the aortic arch segment proximal to the LSA are contraindications to endografting (Fig. 2). In these patients, retrograde dissection is a life-threatening complication that can be caused by the radial forces of the oversized stent-graft in a dissected wall

segment. Tsai et al.[40] demonstrated that aortic arch involvement does not affect mortality at 3 years in 498 patients from the IRAD Registry.

Further Unsolved Problems and Open Questions

Achieving complete false lumen thrombosis and preventing aortic growth and expansion are the main goals in the treatment of dissections. We observed complete or partial false lumen thrombosis in only 60% of patients and severe visceral aortic expansion >50 mm in 5 patients. In Kato's series, only 38% of chronic dissections showed complete obliteration during 6 months of follow-up.[41] False lumen thrombosis, often considered as an endpoint for therapeutic success, is still an ongoing weak point. Short stent-grafts that cover only the main entry, leaving open reentries in the thoracic aorta, are likely contributors to the problem.

A Swedish multicenter study found that only 5% of their 129 patients had enlargement of the covered aortic segment in contrast to 16% who displayed expansion in the uncovered portion of the descending aorta.[42] Reporting on the same phenomenon, Gaxotte et al.[43] noted that complete false lumen thrombosis resulted in decreased aortic diameters, but chronic expansion of the dissected uncovered aorta continued. This correlates with the 16.6% expansion rate of the abdominal aorta in our series.

Another unanswered question concerns the spontaneous course of a mostly asymptomatic ''true lumen collapse.'' The term is difficult to interpret, as dissections are dynamic events with pulsatile movements of the dissecting membrane, which can be observed by cine-MRI. Experimental studies carried out by Chung et al.[44] proved the effectiveness of overstenting the intimal rupture site (''entry'') for treatment of true lumen collapse. However, as long-term results are still lacking, it remains to be seen whether this circumstance can be classified as a criterion of success or aim of therapy. After 2 years, their false lumen thrombosis rate was only 44%.[44] Persisting perfusion of the false channel correlated with late complications, chronic expanding aortic dissection, and death due to rupture.

A consequence of failed depressurization is chronic aortic expansion. The incidence, pathophysiology, and clinical impact of chronic expansion and the factors that influence it have been studied.[36] An initial diameter >4 cm and a persistent entry into the false lumen have been identified as determinants for chronic expanding aortic dissection. In our study, the overall expansion rate of 31.5% (17/54) underlines this significant sequela.

Sueyoshi et al.[45] found an initial aortic diameter >40 mm and an aortic wall thickness >10 mm as risk factors for rupture. Although aneurysms tend to rupture in expanding dissections,[46] even with smaller diameters, there is no level I evidence indicating a diameter that requires intervention. Nevertheless, a diameter >55 mm is a widely accepted indication for treatment of chronic expanding aortic dissection. One patient in our series treated initially at another institution presented with rapidly progressing expansion due to a second entry at the distal end of the stent-graft, causing persisting false channel perfusion (Fig. 4); we implanted a second stent-graft to address the problem.

Mesenteric ischemia has a major impact on outcome. Two of our patients died of multiorgan failure due to persisting visceral ischemia and unresolved malperfusion. If mesenteric ischemia is suspected, an aggressive posture toward laparotomy or laparoscopy is appropriate, even if endovascular revascularization has been achieved. Monitoring laboratory values for potential markers of mesenteric infarction can be crucial. We lost 4 of our patients due to delayed recognition. The surgical treatment of renal and mesenteric ischemia caused by dissection has a mortality of 50% and 80%, respectively.[47]

Spinal cord ischemia after stent-graft repair of dissections varies between 3% and 10% in the literature,[11] but it was 0% in our patients. Potential factors for the lower incidence of spinal cord ischemia during TEVAR are adequate collateral blood supply, no aortic cross clamping, short aortic coverage by the

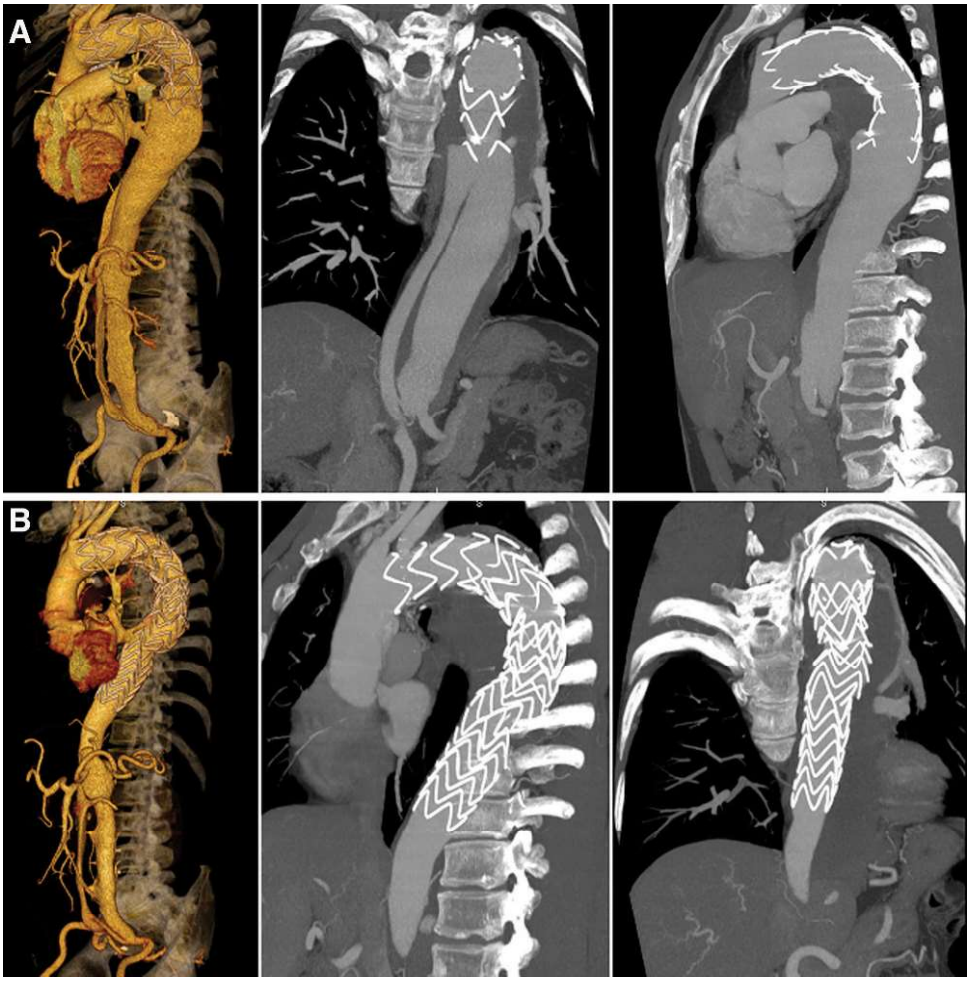

Figure 4 ◆ CTA with 3D volume rendering and multiplanar reconstructions of a man undergoing TEVAR for acute aortic dissection in a referring hospital: (**A**) upper series demonstrates a second entry distal to the stent-graft causing persisting false channel perfusion and rapid expansion. (**B**) Lower images show successful reintervention with a second stent-graft.

stent-graft, and deployment positioning far from the suggested origin of the artery of Adamkiewicz.

The long-term results after endografting of patients with connective tissue disease (Marfan, Ehlers-Danlos) are also unknown. Progressive dilatation, especially in these patients, can cause device migration, endoleak, and repressurization. Thus, in our experience, Marfan patients are questionable candidates for endovascular treatment based on the potential poor midterm outcomes. Indications for TEVAR need a very individual approach in these patients.[48]

Compliance and alignment of stent-grafts in the often tapered and angulated aortic arch are of unknown prognosis. Stiff and calcified membranes in chronic dissections usually cause mismatch and distal stent-graft compression (not collapse).

Current Indications for TEVAR of Type B Dissections

Our own therapy concept calls for endovascular treatment in the symptomatic acute stage or during the chronic course with aneurysmal expansion of more than 55 mm.

Therefore, only 31% of 172 patients presenting with Stanford type B dissection were selected for endovascular surgery in our institution. After type A repair, patients with residual type B dissections are also potential candidates for ''hybrid'' distal stent-grafting. The 57% 30-day mortality with this approach in our series is closely related to complications after type A repair, to a small cohort of 5 sick patients, and thus skeptical judgment. The strategy of immediate hybrid ascending and descending aortic repair, including technical considerations of antegrade versus retrograde stent-graft deployment, needs definitive evaluation.

The so-called ''high-risk patient'' is no longer the leading argument for an endovascular approach. Even in centers that are experienced with open and endovascular techniques, endografting has become the preferred treatment option. Based on our results of conservative treatment and the relatively high complication rate after TEVAR of acute and chronic type B dissections, the currently accepted indications for surgical intervention, depending on clinical pathology and time, are

- Emergency: false lumen rupture and malperfusion (kidney, visceral organs, spinal cord, lower extremities) due to symptomatic true lumen collapse or retrograde dissection. The first choice is TEVAR, but fenestration, open surgery, or hybrid procedures are also appropriate.
- Urgent: persistent pain, refractory hypertension, rapid aortic expansion.
- Elective: late chronic phase with expansion >6 cm (>5 cm for Marfan or Ehlers-Danlos patients) or false aneurysm.

Without a doubt, morphology is one of the most important issues in selecting patients for TEVAR.

CONCLUSION

Despite the benefits of a minimally invasive approach, TEVAR still has a high complication rate (up to 20%) in aortic dissections. Therefore, stent-grafting is limited to acute symptomatic patients and those with chronic expanding dissections over 55 mm in diameter. Hybrid procedures treating patients with chronic expansion of the descending aorta after repair of acute type A dissections need further investigation. For patients with symptomatic or complicated type B dissections, there will continue to be a selection bias in the future because many patients die before reaching the hospital and others who are not surgical candidates will not have access to a facility that specializes in TEVAR.

The only randomized trial so far, the Investigation of Stent-Grafts in Patients with Type B Aortic Dissection (INSTEAD) trial, showed that careful medical management appears to be a viable option and primary strategy for uncomplicated type B dissection with deferred endovascular repair. The role of stent-grafts in the management of uncomplicated acute type B dissections is limited. Asymptomatic patients are referred to conservative treatment until prospective studies can show an advantage for aggressive endovascular treatment in terms of early and long-term survival. The availability and good primary results of stent-graft therapy do not justify an unselected approach.

Interdisciplinary collaboration will allow the incorporation of new technologies and/or biological hypotheses into clinical trials, facilitating their clinical implementation and transforming the dream of individualized treatment into reality. The goal of treatment is always to achieve optimal treatment for those who benefit from intervention.

REFERENCES

1. Meszaros I, Morocz J, Szlavi J, et al. Epidemiology and clinicopathology of aortic dissection. *Chest*. 2000;117:1271–1278.
2. Safi HJ, Miller CC, Subramaniam MH, et al. Thoracic and thoracoabdominal aneurysm repair using cardiopulmonary bypass, profound hypothermia and circulatory arrest via left side of the chest incision. *J Vasc Surg*. 1998;28:591–598.
3. DeBakey M, McCollum C, Crawford E, et al. Dissection and dissecting aneurysms of the aorta: twenty year follow-up of five hundred twenty-seven patients treated surgically. *Surgery*. 1982;92:1118–1134.

4. Nienaber CA, Fattori R, Lund G, et al. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. *N Engl J Med*. 1999;340:1539–1545.
5. Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med*. 1999;340:1546–1552.
6. Eggebrecht H, Herold U, Kuhnt O, et al. Endovascular stent-graft treatment of aortic dissection: determinants of post-interventional outcome. *Eur Heart J*. 2005;26:489–497.
7. Nienaber CA, Skriabina V, Schareck W, et al. To stent or not to stent aortic dissection: good news for a chosen few, but who? [commentary] *Eur Heart J*. 2005;26:431–432.
8. Winnerkvist A, Lockowandt U, Rasmussen E, et al. A prospective study of medically treated acute type B aortic dissection. *Eur J Vasc Endovasc Surg*. 2006;32:349–355.
9. Winnerkvist A, Brorsson B, Rådegran K. Quality of life in patients with chronic type B dissection. *Eur J Vasc Endovasc Surg*. 2006;32:34–37.
10. Böckler D, Schumacher H, Ockert S, et al. Acute and chronic expanding type B dissection: what is the impact of endografts on indications for treatment? *Gefaesschirurgie*. 2003;8:192–199.
11. Böckler D, Schumacher H, Ganten M, et al. Complications after endovascular repair of acute and chronic Stanford type B aortic dissections. *J Thorac Cardiovasc Surg*. 2006;132:361–368.
12. Chaikof EL, Blankensteijn JD, Harris PL, et al. Reporting standards for endovascular aortic aneurysm repair. *J Vasc Surg*. 2002;35: 1048–1060.
13. Marui A, Mochizuki T, Mitsui N. Towards the best treatment for uncomplicated patients with type B acute aortic dissection: a consideration for sound surgical indication. *Circulation*. 1999;100(Suppl II):275–280.
14. Genoni M, Paul M, Jenni R, et al. Chronic beta-blocker therapy improves outcome and reduces treatment costs in chronic type B aortic dissection. *Eur J Cardiothorac Surg*. 2001;19:606–601.
15. Carrel T, Nguyen T, Gysi J. Acute type B aortic dissection: prognosis after initial conservative treatment and predictive factors for a complicated course [in German]. *Schweiz Med Wochenschr*. 1997;127:1467–1473.
16. Iguchi A, Tabayashi K. Outcome of medically treated Stanford B aortic dissection. *Jpn Circ J*. 1998;62:102–105.
17. Kozai Y, Watanabe S, Yonezawa M, et al. Long-term prognosis of acute aortic dissection with medical treatment: a survey of 263 unoperated patients. *Jpn Circ J*. 2001;65:359–363.
18. Wheat MW. Current status of medical therapy of acute dissecting aneurysms of the aorta. *World J Surg*. 1980;4:563–569.
19. Jacobs M, Elenbaas T, Schurink GW, et al. Acute type B aortic dissection: surgical indications and strategy. In: Branchereau A, Jacobs M, eds. *Vascular Emergencies*. Armonk, NY: Futura; 2003:87–97.
20. Genoni M, Paul M, Tavakoli R. Predictors of complications in acute type B dissection. *Eur J Cardiothorac Surg*. 2002;22:59–63.
21. Kato N, Hirano T, Takeda K, et al. Treatment of aortic dissections with a percutaneous intravascular endoprosthesis: comparison of covered and bare stents. *J Vasc Interv Radiol*. 1994;5:805–812.
22. Umana JP, Lai DT, Mitchell RS, et al. Is medical therapy still the optimal treatment strategy for patients with acute type B aortic dissections? *J Thorac Cardiovasc Surg*. 2002;124:897–910.
23. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International registry of acute aortic dissection (IRAD). New insights into an old disease. *JAMA*. 2000;283:897–903.
24. Lansman SL, Hagl C, Fink D, et al. Acute type B dissection: surgical therapy. *Ann Thorac Surg*. 2002;74:1833–1835.
25. Dake MD, Miller DC, Semba CP, et al. Transluminal placement of endovascular stent-grafts for the treatment of descending thoracic aortic aneurysms. *N Engl J Med*. 1994;331:1729–1734.
26. Demers P, Miller DC, Mitchell RS, et al. Midterm results of endovascular repair of descending thoracic aortic aneurysm with first-generation stent grafts. *J Thorac Cardiovasc Surg*. 2004;127:664–673.
27. Schor JS, Yerlioglu ME, Galla JD. Selective management of descending aortic dissection. *Ann Thorac Surg*. 1996;67:2002–2005.
28. Beregi JP, Haulon S, Otal P, et al. Endovascular treatment of acute complications associated with aortic dissection: midterm results from a multicenter study. *J Endovasc Ther*. 2003;10:486–493.
29. Bell RE, Taylor PR, Aukett M, et al. Results of urgent and emergency thoracic procedures treated by endoluminal repair. *Eur J Vasc Endovasc Surg*. 2003;25:527–531.
30. Matravers P, Morgan R, Belli A. The use of stent grafts for the treatment of aneurysms and dissections of the thoracic aorta: a single centre experience. *Eur J Vasc Endovasc Surg*. 2003;26:587–595.
31. Palma JH, de Souza JA, Rodríguez CM, et al. Self-expandable aortic stent-grafts for treatment of descending aortic dissections. *Ann Thorac Surg*. 2002;73:1138–1142.

32. Criado FJ, Clark NS, Barnatan MF. Stent graft repair in the aortic arch and descending thoracic aorta: a 4-year experience. *J Vasc Surg*. 2002;36:1121–1128.
33. Orend KH, Scharrer-Pamler R, Kapfer X, et al. Endovascular treatment in diseases of the descending thoracic aorta: 6-year results of a single center. *J Vasc Surg*. 2003;37:91–99.
34. Xu SD, Huang FJ, Yang JF, et al. Endovascular repair of acute type B aortic dissection: early and mid-term results. *J Vasc Surg*. 2006;43: 1090–1095.
35. Rehders TC, Nienhaber CA. Complications of stent graft placement in the thoracic aorta. In: Branchereau A, Jacobs M, eds. *Complications in Vascular and Endovascular Surgery*. Armonk, NY: Futura Publishing Company, Inc.; 2001:85–192.
36. Kato M, Bai H, Sato K. Determining surgical indications for acute type B dissection based on enlargement of aortic diameter during the chronic phase. *Circulation*. 1995;92:107–112.
37. Kahn SL, Dake MD. Stent graft management of stable uncomplicated type B-dissections. *Perspect Vasc Surg Endovasc Ther*. 2007;19: 162–169.
38. Bortone AS, Schena S, D'Agostino D, et al. Immediate versus delayed endovascular treatment of post-traumatic aortic pseudoaneurysms and type B dissections: retrospective analysis and premises to the upcoming European trial. *Circulation*. 2002;106(12 Suppl 1): I234–I240.
39. von Tengg-Kobligk H, Weber TF, Rengier F, et al. Imaging modalities for the thoracic aorta. *J Cardiovasc Surg (Torino)*. 2008;49:429–447.
40. Tsai TT, Isselbacher EM, Trimarchi S, et al. Acute type B aortic dissection: does aortic arch involvement affect management and outcomes? Insights from the International Registry of Acute Aortic Dissection (IRAD). *Circulation*. 2007;116(Suppl I):I150–I156.
41. Kato M, Matsuda T, Kaneko M, et al. Outcomes of stent-graft treatment of false lumen in aortic dissection. *Circulation*. 1998;98(19 Suppl): II305–312.
42. Resch TA, Delle M, Falkenberg M, et al. Remodeling of the thoracic aorta after stent grafting of type B dissection: a Swedish multicenter study. *J Cardiovasc Surg (Torino)*. 2006;47:503–508.
43. Gaxotte V, Thony F, Rousseau H, et al. Midterm results of aortic diameter outcomes after thoracic stent-graft implantation for aortic dissection: a multicenter study. *J Endovasc Ther*. 2006;13:127–138.
44. Chung JW, Elkins C, Sakai T. True-lumen collapse in aortic dissection: part I, II. Evaluation of causative factors in phantoms with pulsatile flow. *Radiology*. 2000;214:87–94, 99–106.
45. Sueyoshi E, Imada T, Sakamoto I, et al. Analysis of predictive factors for progression of type B aortic intramural hematoma with computed tomography. *J Vasc Surg*. 2002;35: 1179–1185.
46. Juvonen T, Ergin MA, Galla JD. Risk factors for rupture of chronic type B-dissections. *J Thorac Cardiovasc Surg*. 1999;117:776–786.
47. Cambria RP, Brewster DC, Gertler J, et al. Vascular complications associated with spontaneous aortic dissection. *J Vasc Surg*. 1988;7: 199–209.
48. Geisbüsch P, Kotelis D, von Tengg-Kobligk H, et al. Thoracic aortic endografting in patients with connective tissue diseases. *J Endovasc Ther*. 2008;15:144–149.