Aortic dilatation after endovascular repair of blunt traumatic thoracic aortic injuries

Thomas L. Forbes, MD, Jeremy R. Harris, MD, D. Kirk Lawlor, MD, and **Guy DeRose, MD,**
London, Ontario, Canada

Objective: **Endovascular repair of blunt traumatic thoracic aortic injuries (BTAI) has become routine at many trauma centers despite concerns regarding durability and aortic dilatation in these predominantly young patients. These concerns prompted this examination of thoracic aortic expansion after endovascular repair of a BTAI.**
Methods: **The immediate postoperative and most recent computed tomography (CT) scans of patients who had undergone urgent endovascular repair of a BTAI and had at least 1 year of follow-up were reviewed. Diameter measurements were made at four predetermined sites: immediately proximal to the left subclavian artery (D1), immediately distal to the left subclavian artery (D2), distal extent of the endograft (D3), and 15 mm beyond the distal end of the endograft (D4). Split screens permitted direct comparison of measurements between CTs at the corresponding levels.**
Results: **During a 6-year period (2001-2007), 21 patients (mean age, 42.9 years; range, 19-81 years) underwent endovascular repair of a BTAI, 17 with at least 1 year of follow-up (mean, 2.6 years; range, 1-5.5 years). No patients required reintervention during this period. The mean rate of dilatation for each level of the thoracic aorta in mm/year was: D1, 0.74 (95% confidence interval [CI], 0.42-1.06); D2, 0.83 (95% CI, 0.55-1.11); D3, 0.63 (95% CI, 0.37-0.89); D4, 0.47 (95% CI, 0.27-0.67). The rate of expansion of D2 differed significantly vs D4 ($P = .025$).**
Conclusions: **During the first several years of follow-up, the proximal thoracic aorta dilates minimally after endovascular repair of BTAIs, with the segment just distal to the left subclavian artery expanding at a slightly greater rate. Longer-term follow-up is necessary to determine whether this expansion continues and becomes clinically significant. (J Vasc Surg 2010;52:45-8.)**

Injuries to the proximal descending thoracic aorta secondary to blunt trauma (BTAI) remain a commonly lethal injury. In many trauma centers, however, a major paradigm shift has occurred in the treatment of these injuries toward the less invasive and physiologically less stressful endovascular form of repair. Several centers, including ours,[1-5] have reported favorable early results with this mode of repair compared with standard open repair. Recently, several meta-analyses have summarized the world literature and the early advantages of endovascular repair that include lower mortality and stroke rates and minimal risk of the catastrophic complication of paraplegia.[6-9]

Despite these impressive short-term outcomes, questions remain regarding the long-term durability of such endovascular repairs. Somewhat reassuringly, several authors have reported few adverse clinical sequelae requiring reintervention after several years of follow-up.[10,11] However, with the trauma population generally being young, assurances of durability will require decades of clinical and radiologic surveillance. This follow-up is more intense than that previously applied to patients after open repair and provides a unique opportunity to determine the degree of thoracic aortic dilatation when subjected to the radial force of an endograft.

Our center's immediate results after endovascular repair of these injuries has previously been reported.[1,2] The purpose of the present study was to review these patients after a minimum of 1 year of follow-up. Specific attention was paid to clinical outcomes and reinterventions and a review of postoperative imaging, computed tomography (CT), to determine the degree of thoracic aortic dilatation in the vicinity of the injury.

METHODS

This study received approval from the University of Western Ontario's Research Ethics Board for Health Sciences Research Involving Human Subjects.

The vascular surgery database at our university-affiliated medical center was reviewed to identify all patients who had undergone urgent endovascular repair of a blunt thoracic aortic injury during a recent 6-year period (2001-2007). The study was limited to patients who survived the immediate postoperative period and had a minimum of 1 year of postoperative clinical and radiologic follow-up. Patients underwent nongated CT scans postoperatively during the initial hospitalization, after 6 months, 1 year, and then yearly thereafter.

Clinical outcomes, including immediate operative results, delayed complications, and the need for reintervention, were determined through review of the database and patient records.

From the Division of Vascular Surgery, London Health Sciences Centre & The University of Western Ontario.
Competition of interest: none.
Correspondence: Thomas L. Forbes, MD, Division of Vascular Surgery, London HSC & University of Western Ontario, 800 Commissioners Rd E, E2-119, London, ON, N6A5W9 Canada (e-mail: Tom.Forbes@lhsc.on.ca).
The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a competition of interest.
0741-5214/$36.00

doi:10.1016/j.jvs.2010.01.094

Table I. Summary of results of patients treated for a blunt traumatic thoracic aortic injury between 2001 and 2007

Variable	*Outcome*
Patients, No.	21
Age, mean (range), y	42.9 (19-81)
Coverage of left subclavian artery, No. (%)	12 (57)
Survival, %	
30 d	90.5
1, 3, 5 y	85.7
Paraplegia, No.	0
Stroke, No.	0
Reinterventions, No.	0
Follow-up, mean (range) y	2.6 (1-5.5)

Table II. Aortic dilatation results in 17 patients with >1 year of follow-up

Aortic segment	*Growth rate (mm/y)*	*95% CI*	P *value (vs D4)*
D1 (proximal to left subclavian artery)	0.74	0.42-1.06	.07
D2 (distal to left subclavian artery)	0.83	0.55-1.11	.025
D3 (distal end of endograft)	0.63	0.37-0.89	.06
D4 (15-mm distal to end of endograft)	0.47	0.27-0.67	—

CI, Confidence interval; NS, not significant.

In those patients with a minimum of 1-year of follow-up, the immediate postoperative and the most recent CT scans were reviewed on a General Electric Advantage Workstation (General Electric, Waukesha, Wisc). Split screens permitted direct comparison of diameters at the same levels of the thoracic aorta. This method has been described previously.[12]

Thoracic aortic diameters were measured at four predetermined sites: immediately proximal to the left subclavian artery (D1), immediately distal to the left subclavian artery (D2), at the distal end of the thoracic endograft (D3), and 15 mm beyond the distal extent of the endograft (D4). This final site of measurement (D4) is not directly affected by the radial forces of the endograft, and in nonstented aortas would be expected to expand at a similar rate as more proximal segments of the descending thoracic aorta. This level (D4) acts as an internal control.

The largest outer wall to outer wall diameter was recorded at each level. The rate of dilatation was determined at each position of measurement and those subjected to the radial force of the endograft, D1-3, were compared with that beyond the distal extent of the endograft in the descending thoracic aorta (D4).

Summary data are presented as means and their 95% confidence intervals (CI) with a $P < .05$ level of statistical significance. Means were compared using repeated measures analysis of variance with Tukey multiple comparison post test with Instat 3.06 software (GraphPad Software, San Diego, Calif).

RESULTS

Between 2001 and 2007, 21 patients presented to our center with a BTAI and received immediate endovascular repair. The immediate outcome measures of these patients are summarized in Table I. All injuries were successfully treated, with no endoleaks. A more complete description of the patients, as well as the preoperative variables and intraoperative technical details, has previously been reported.[1,2] In general, these patients tended to be young (mean age, 42.9 years), and two patients died in the early postoperative period (9.5% early mortality rate) as a result of multiple injuries. These deaths were not related to the stent graft or the aortic injury. The analysis excluded two other patients because they did not have the requisite 1 year of follow-up.

In 57% of patients, deliberate coverage of the subclavian artery was required due to the anatomic location of the aortic injury. Early in our experience, one patient underwent carotid-subclavian bypass during the same operation as the thoracic endovascular repair.

All injuries were treated with a single thoracic endograft: 11 patients received a Talent/Valiant (Medtronic, Santa Rosa, CA) and 10 received a Zenith TX2 (Cook Medical, Bloomington, Ind). Preoperative aortic diameters were 20 to 30 mm, and endograft diameters were 22 to 34 mm. Selection of the endograft was at the discretion of the treating surgeon and typically included a diameter representing no more than a 10% over-sizing relative to the thoracic aortic diameter on a preoperative CT scan.

All patients were treated in an operating room setting using portable C-arm fluoroscopy and general anesthesia. Early in our experience, adenosine-induced cardiac asystole was used to ensure accurate deployment, but in more recent cases, induction of hypotension has proven sufficient. There were no instances of major procedure-related morbidity, including paraplegia or stroke, and there was no need for reintervention in the early postoperative period.

Diameter measurements were performed on those 17 patients who had been monitored for at least 1 year, both clinically, and radiologically with CT scans. Mean duration of postoperative surveillance of these 17 patients was 2.6 years (range, 1-5.5 years). No reinterventions or procedurally related morbidity occurred during the surveillance period. There were no instances of new-onset hypertension or other possibly device-related medical conditions. The oldest patient in this cohort died 6 months postoperatively from progressive respiratory dysfunction secondary to pneumonia, resulting in a midterm survival rate of 85.7% (Table I).

The rate of dilatation observed at several preselected levels of the proximal descending thoracic aorta is summarized in Table II. The level 15 mm beyond the distal extent of the endograft (D4) expanded at a rate of 0.47 mm/y (range, 0.27-0.67 mm/y). This rate of expansion did not differ with that of the thoracic aorta proximal to the left

subclavian artery (D1) or at the distal extent of the endograft (D3), which dilated at rates of 0.74 mm/y (range, 0.42-1.06 mm/y) and 0.63 mm/y (range, 0.37-0.89 mm/y), respectively. Expansion rates at D1 did not differ between patients with and without subclavian artery coverage. The thoracic aorta immediately distal to the left subclavian artery (D2) expanded at a rate of 0.83 mm/y (range, 0.55-1.11 mm/y), which was significantly greater ($P = .025$) than that of the aorta immediately beyond the distal extent of the thoracic endograft (D4).

When the diameters of all postoperative CTs were reviewed, the D2 expansion rate was linear throughout the follow-up period, although the number of patients is too small to determine statistical significance. There were no instances of endoleak or graft migration during this time period. Given the relatively small number of patients, there was no difference in expansion rates when considering patient age, sex, or type of endograft.

DISCUSSION

Beginning with its initial description,[13] endovascular repair of these traumatic injuries has been regularly documented and described, at least in regards to its immediate outcomes. Although no randomized controlled trials exist, several groups of investigators have compared endovascular repair with the prior gold standard of open repair with an historic or contemporary surgical control group.[2-4] These single-center studies consistently displayed a survival advantage with endovascular repair as well as a reduction in major perioperative morbidity, including paraplegia. Regardless, follow-up information was often limited.

More recently, a prospective nonrandomized multicenter study compared 193 patients who underwent urgent repair of a BTAI (125 with stent grafts and 68 with open repair).[9] Again, endovascular repair resulted in a significant survival advantage and a statistically insignificant trend toward a lower risk of paraplegia (0.8% with endovascular repair and 2.9% after open repair). Of some concern, however, were the 20% graft-related complication and 14.4% endoleak rates in the endovascular group. In addition, six patients with endoleaks eventually required conversion to open repair.

Complications related to endovascular repair have not been limited to specific endograft configuration issues. In a report from Sydney, Australia, 5 of 12 young trauma patients treated with endografts required treatment for new-onset hypertension in the early postoperative period.[14] Anatomic causes, such as an acquired aortic coarctation, were excluded. The authors postulated that the radial force of the endograft interferes with the normal baroreceptor function in the thoracic aorta and results in a less compliant aorta. This response was not seen in the present series, but regardless, surgeons should be aware of its potential in the follow-up of these patients.

Although such complications can occur, the present series illustrates that these life-threatening injuries can be treated with endovascular techniques with minimal morbidity and protection from reinterventions in the first couple of years. Ongoing surveillance is essential, however, because this method of treatment requires placement of an endograft, whose long-term material and stent performance is unknown, into a hostile and dynamic proximal thoracic aortic environment. This necessary surveillance is hindered by a predominantly young mobile trauma population that is often less diligent in attending follow-up appointments than other patient populations.[15]

After a minimum of 1-year follow-up, we observed an increased growth rate in the area of the aortic isthmus (D2) compared with the unstented descending thoracic aorta distal to the endograft (D4). All four areas dilated to some extent, as is the natural history of the thoracic aorta, but the injured segment dilated at a faster rate, at least in the first year after surgery.

One possible shortcoming of this study is that the true preoperative aortic diameters before the injury are not known. The first postoperative CT scan, with endograft in place, was deliberately chosen as the baseline study because preoperative scans also reflect any hemodynamic instability in these multiply injured trauma patients. Differences in aortic diameters of up to 30% have been documented in such hypotensive trauma patients between preoperative CTs and postoperative scans.[16] This becomes problematic because endograft selection based on measurements from a CT scan in a hypovolemic patient could result in stent graft under-sizing and poor apposition to the thoracic aorta. This poor apposition, whether caused by relative over- or under-sizing of the graft, can lead to stent graft collapse. Newer approaches to CT imaging of the thoracic aorta, namely electrocardiography-gated CTs,[17] have shown promising results in more accurate preoperative planning and sizing of thoracic aortic procedures.

The expected durability of any endovascular aortic repair depends on the life expectancy of the patient and the long-term behavior of the aortic segment in question. It has been well documented that infrarenal aortic dilatation occurs after endovascular aneurysm repair and can result in graft migration and type I endoleaks.[18] Until recently, less information has been available on the natural history of the thoracic aorta. In a recent study, 1000 thoracic CTs were reviewed to examine the natural behavior of the thoracic aorta with increasing age.[19] Between teenagers and octogenarians, a 1-cm increase was observed in the mean diameter at the thoracic aortic isthmus. Obviously, if this dilatation, seen in noninjured, nonstented aortas, occurred to this degree after endovascular repair of a BTAI, there would be a risk of type I endoleaks, device migration, and collapse. However, the thoracic aorta subjected to an endograft may behave differently than the steady dilatation seen in nonstented thoracic aortas.

It appears that opposing forces are at play when the thoracic aorta is treated with an endograft. At first glance, the radial force imparted on the aorta by the endograft could be expected to increase the rate of dilatation of the aorta. The degree to which this occurs would be related to the initial aortic diameter, the degree of endograft oversizing, and might be expected to be device specific. The

present study was not able to display such a correlation due to the number of patients.

In opposition to the radial force, the aorta is subjected to an inflammatory fibrous reaction that has been observed after endograft placement. Although our group has had no experience operating on patients with previously placed endografts for BTAI, other surgeons have noted a dense fibrous reaction at the site of injury that is thought to promote healing and endograft adherence.[19] This is thought to, at least partially, mitigate the aortic expansion forces of the endograft itself. Some recent animal research would support this.[20] When endografts, with 10% over-sizing, were placed in the proximal descending thoracic aorta of pigs, intimal thickening and medial fibrosis were observed at explantation after 6 to 15 months. This reaction inhibited the growth of the stented aorta compared to the proximal and distal nonstented segments.

CONCLUSIONS

Endovascular repair of BTAI has resulted in major advances in the treatment of these often-young trauma patients and has become the preferred method of treatment at many trauma centers. The early data are robust enough that attention should be directed to longer-term follow-up. Most studies, including ours, have reported encouragingly low rates of reintervention and longer-term complications. However, in the present study we have observed an increased rate of dilatation of the injured portion of the thoracic aorta compared with the nonstented distal aorta. This is likely an early response to the injury itself, as well as the radial force of the endograft, and will be mitigated by the fibrous, healing reaction others have observed after endograft placement.[19] Of course, longer-term follow-up, both radiographically and clinically, is necessary before confirming such conclusions.

AUTHOR CONTRIBUTIONS

Conception and design: TF
Analysis and interpretation: TF, JH, DL, GD
Data collection: TF
Writing the article: TF
Critical revision of the article: TF, JH, DL, GD
Final approval of the article: TF, JH, DL, GD
Statistical analysis: TF
Obtained funding: Not applicable
Overall responsibility: TF

REFERENCES

1. Lawlor DK, Ott M, Forbes TL, Kribs S, Harris KA, DeRose G. Endovascular management of traumatic thoracic aortic injuries. Can J Surg 2005;48:293-7.
2. Ott MC, Charyk-Stewart T, Lawlor DK, Gray DK, Forbes TL. Management of blunt thoracic aortic injuries: endovascular stents versus open repair. J Trauma 2004;56:565-70.
3. Chung J, Owen R, Turnbull R, Chyczij H, Winkelaar G, Gibney N. Endovascular repair in traumatic thoracic aortic injuries: comparison with open surgical repair. J Vasc Interv Radiol 2008;19:479-86.
4. Riesenman PJ, Farber MA, Rich PB, Sheridan BC, Mendes RR, Marston WA, et al. Outcomes of surgical and endovascular treatment of acute traumatic thoracic aortic injury. J Vasc Surg 2007;46:934-40.
5. Mohan IV, Hitos K, White GH, Harris JP, Stephen MS, May J, et al. Improved outcomes with endovascular stent grafts for thoracic aortic transections. Eur J Vasc Endovasc Surg 2008;36:152-7.
6. Tang GL, Tehrani HY, Usman A, Katariya K, Otero C, Perez E, et al. Reduced mortality, paraplegia, and stroke with stent graft repair of blunt aortic transections: a modern meta analysis. J Vasc Surg 2008:47: 671-5.
7. Xenos Es, Abedi NN, Davenport DL, Minion DJ, Hamdallah O, Sorial EE, et al. Meta analysis of endovascular vs open repair for traumatic descending thoracic aortic rupture. J Vasc Surg 2008;48:1343-51.
8. Doss M, Wood JP, Balzer J, Martens S, Deschka H, Moritz A, et al. Emergency endovascular interventions for acute thoracic aortic rupture: four-year follow-up. J Thorac Cardiovasc Surg 2005;129:645-51.
9. Demetriades D, Velmahos G, Scalea TM, Jurkovich GJ, Kharmy-Jones R, Teixeira PG, et al. Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study. J Trauma 2008;64: 561-71.
10. Canaud L, Alric P, Branchereau P, Marty-Ane C, Berthet JP. Lessons learned from midterm follow-up of endovascular repair for traumatic rupture of the aortic isthmus. J Vasc Surg 2008;47:733-8.
11. Fattori R, Russo V, Lovato L, Di Bartolomeo R. Optimal management of traumatic aortic injury. Eur J Vasc Endovasc Surg 2009:37:8-14.
12. Falkensammer J, Oldenburg WA, Biebi M, Hugl B, Hakaim AG, Crook JE, et al. Abdominal aortic aneurysm neck remodeling after open aneurysm repair. J Vasc Surg 2007;45:900-5.
13. Semba CP, Kato N, Kee ST, Lee GK, Mitchell RS, Miller DC, et al. Acute rupture of the descending thoracic aorta: repair with use of endovascular stent-grafts. JVIR 1997;8:337-42.
14. Tzilalis VD, May J, White GH, Stephen MS, Harris JP. Severe hypertension following implantation of endovascular grafts into the thoracic aorta in young patients. J Endovasc Ther 2005;12:142-3.
15. McCarthy MJ. Is endovascular repair now the first line treatment for traumatic transaction of the thoracic aorta? Eur J Vasc Endovasc Surg 2008;36:158-9.
16. van Prehn J, van Herwaarden JA, Muhs BE, Arnofsky A, Moll FL, Verhagen HJ. Difficulties with endograft sizing in a patient with traumatic rupture of the thoracic aorta: the possible influence of hypovolemic shock. J Vasc Surg 2008;47:1333-6.
17. Van Prehn J, Bartels LW, Mestres G, Vincken KL, Prokop M, Verhagen HJM, et al. Dynamic aortic changes in patients with thoracic aortic aneurysms evaluated with electrocardiography-triggered computed tomographic angiography before and after thoracic endovascular aneurysm repair: preliminary results. Ann Vasc Surg 2009;23:291-7.
18. Litwinski RA, Donayre CE, Chow SL, Song TK, Kopchok G, Walot I, White RA. The role of aortic neck dilatation and elongation in the etiology of stent graft migration after endovascular abdominal aortic aneurysm repair with passive fixation device. J Vasc Surg 2006;44:1176-81.
19. Hartley MC, Langan EM, Cull DL, Taylor SM, Carsten CG, Blackhurst DW. Evaluation of the diameter of the proximal descending thoracic aorta with age: implications for thoracic aortic stent grafting. Ann Vasc Surg 2009;23:639-44.
20. Siegenthaler MP, Celik R, Haberstroh J, Bajona P, Goebel H, Brehm K, et al. Thoracic endovascular stent grafting inhibits aortic growth: an experimental study. Eur J Cardiothorac Surg 2008;34:17-25.

Submitted Dec 3, 2009; accepted Jan 29, 2010.