Eleven-Year Experience Treating Blunt Thoracic Aortic Injury at a Tertiary Referral Center

Running Head: Blunt Thoracic Aortic Injury Outcomes

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ABSTRACT

Background: Blunt thoracic aortic injury treatment has evolved over the past decade particularly with respect to endovascular intervention options. We investigated the trends in blunt thoracic aortic injury management and outcomes over an 11-year span at the sole tertiary referral center in our state.

Methods: We retrospectively reviewed all patients who presented to our institution with blunt traumatic aortic injury between 2007 and 2017. Baseline demographics including aortic injury grade, injury severity score, and abbreviated injury scale were collected. Outcomes were compared by type and timing of treatment, which included either nonoperative management, endovascular repair, or open surgical repair. Bivariate and multivariable analyses were performed to examine treatment group differences and factors associated with 30-day mortality.

Results: In total, 229 patients were reviewed. The distribution of injury severity was: Grade 1 (30%), Grade 2 (8%), Grade 3 (30%), Grade 4 (31%). Overall, 27% underwent endovascular repair, 29% open surgery, and 44% definitive nonoperative management. Over the study period, there was a dramatic decline in open surgery and a corresponding rise in endovascular treatment. 30-day mortality for the entire cohort was 22%. Mortality by treatment subgroup was 30% for nonoperative management, 8.2% for endovascular, and 21% for open surgery. Delaying endovascular or open surgical treatment by at least 24 hours after admission was associated with significantly improved 30-day survival.

Conclusions: Procedural intervention, whether endovascular or surgical, is associated with improved mortality compared to nonoperative treatment. Delayed intervention, particularly in high grade injuries, may allow for initial patient stabilization and improved outcomes.

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Blunt thoracic aortic injury (BTAI) remains a devastating condition often accompanied by multiple traumatic injuries and is associated with high mortality[1]. BTAI is often the result of sudden deceleration in motor vehicle accidents (MVA) and falls, with some estimates indicating that up to 80% of these patients die before reaching the hospital[2-4]. Although BTAI incidence is <1% in trauma patients, it is the second leading cause of death in this population[7-9]. Advances in endovascular therapy have expanded management options for BTAI, and excellent outcomes with thoracic endovascular aortic repair (TEVAR) have led some to question the utility of open aortic repair (OAR)[10,11]. Given the paucity of data in a rapidly evolving field, we sought to analyze how treatment and outcomes of BTAI have changed over the past decade at a large tertiary referral and American College of Surgeons-verified Level 1 trauma center.

PATIENTS AND METHODS

The Institutional Review Board of Indiana University approved this study. All patients with BTAI who presented to our institution between January 2007 and December 2017 were reviewed. Patients were retrospectively identified, and data elements were extracted from prospective institutional trauma and cardiac surgery registries. Patient medical records were individually reviewed. Aortic injury severity was categorized by the grading system introduced by Azizzadeh et al.: Grade 1 (intimal injury), Grade 2 (intramural hematoma), Grade 3 (pseudoaneurysm), and Grade 4 (rupture)[12]. To characterize the overall severity of clinical status at presentation, we utilized intubation status, mechanism of injury, systolic blood pressure (SBP) on admission, injury severity score (ISS), and the abbreviated injury scale (AIS). Treatment strategy was categorized as either nonoperative management (NOM), TEVAR, or OAR. Timing of interventional procedure (TEVAR or OAR) was defined as either Early (<24 hours from time of admission) or Delayed (>24 hours from time of admission). The primary outcome analyzed was 30-day mortality.

Statistical Analysis

We used descriptive statistics such as mean and frequency distribution to describe the study cohort. Bivariate analyses were done utilizing stratified analysis by treatment group. ANOVA and Pearson's chi-square tests were employed to identify differences in characteristics across treatment groups. T-tests for continuous variables and chi-square tests for categorical variables were used to compare between those who were alive or dead at 30-days. In bivariate analyses of categorical variables with expected cell-count less than 5%, we used exact tests to examine any differences. Multivariable analyses using procedure type, age, gender, race, injury mechanism, and AIS-chest were done to examine their association with 30-day mortality. A sub-group analysis among those who underwent TEVAR or OAR was performed using timing of procedure in a multivariable model. Adjusted odds ratio from the multivariable logistic models were reported with 95% confidence intervals. All analyses were performed using Stata/SE 14.2 and hypotheses were tested at 0.05 level of significance.

RESULTS

Patient Characteristics

A total of 229 patients with BTAI were identified in the 11-year study period. General characteristics of the cohort are in Table 1. Mean age was 46 years and the cohort was predominately white and male. The injury mechanism was MVA in nearly 80%. Mean ISS was 37.6±13.8 and mean Chest AIS was 4.5±0.5. At initial presentation, the mean SBP was 122±32 mmHg and the majority of patients were not intubated. Chest computed tomography (CT) or CT angiography (CTA) were the most common imaging modalities used to diagnose BTAI. The overall distribution of injury grade was 30% Grade 1, 8% Grade 2, 30% Grade 3, and 31% Grade 4. Over the study period, there was a slight increase in the number of Grade 1-3 cases, but starting in 2011 there was a dramatic decline in the number of Grade 4 (Figure 1).

Of the entire cohort, 44% received definitive NOM, 27% underwent TEVAR, and 29% underwent OAR. Frequency of treatment type also changed over the study period, with significant decline

in OAR and concomitant increase in TEVAR starting in 2011 (Figure 2). Baseline demographics were similar across the three treatment groups. 30-day mortality for all patients combined was 22%.

Nonoperative Management

Patients who underwent NOM as definitive treatment received anti-hypertensive therapy to maintain strict BP control and blood product transfusion as necessary. 75% of all Grade 1, 68% of all Grade 2, 30% of all Grade 3, and 22% of all Grade 4 injuries were treated with NOM (Table 1). In general, the NOM group was older (mean age 49 years), had a marginally lower Chest AIS (4.4), and were more likely to be involved in single vehicle MVA than those in the TEVAR or OAR groups. Grade 3 and 4 patients treated with definitive NOM were those too severely ill or clinically unstable to withstand an interventional procedure. Overall 30-day mortality for all NOM patients was 30%, the highest of any treatment subgroup. As expected, mortality depended primarily on injury grade, with higher grade associated with increased mortality: 8% for Grade 1, 15% for Grade 2, 48% for Grade 3, and 94% for Grade 4. The mean intensive care unit (ICU) and total hospital length of stay (LOS) for NOM was 7 and 10 days, respectively.

Pulmonary events, which included respiratory failure, reintubation, and acute respiratory distress syndrome (ARDS), were the most common complications and occurred in 21% of NOM patients (Table 2). Infectious complications, including sepsis, intraabdominal abscess, pneumonia, empyema, wound infection, and peritonitis, were the second most common and occurred in 12.7% of NOM patients. These pulmonary and infectious complications affected mostly Mild Injury (Grades 1-2) patients. Cardiac arrest was the third most common complication and occurred in 9.8% of the group, primarily in those with Severe Injury (Grades 3-4). Long-term follow-up was available in a relatively small number of patients (Table 3); this data reflected follow-up with any physician within our health system, not necessarily with our specific group.

Endovascular Surgical Intervention

All patients received medical management for stabilization and BP control prior to undergoing TEVAR. All TEVAR procedures were performed in a hybrid operating room. Mean procedure duration, fluoroscopy time, and radiation dose were 130±80 minutes, 9.9±9.5 minutes, and 619.9±533.0 mGy, respectively. Procedural vascular access was obtained via the transfemoral route in all cases. The left subclavian artery ostium was covered in 36% of cases, and 16% underwent left carotid to subclavian bypass. Our criteria for performing carotid to subclavian bypass were: a) presence of a patent left internal mammary artery to left anterior descending artery bypass graft; b) if the left vertebral artery was dominant or larger than the right vertebral artery on imaging; c) prior thoracic or abdominal aortic procedures with increased risk of spinal cord ischemia.

The types of endografts utilized throughout the study included the Medtronic Talent (Medtronic plc, Dublin, Ireland), Cook Zenith TX2 and Zenith Alpha (Cook Medical LLC, Bloomington, Indiana, USA), Gore TAG and conformable TAG (W.L.Gore & Associates Inc, Flagstaff, Arizona, USA), Cordis PALMAZ XL (Cardinal Health, Santa Clara, California, USA), and Bolton RelayPlus (Bolton Medical Inc, Sunrise, Florida, USA). TEVAR patient outcomes by device manufacturer are displayed in Table 4.

7.2% of all Grade 1, 26% of Grade 2, 42% of Grade 3, and 30% of Grade 4 patients underwent TEVAR. Within this cohort, 51% underwent Early TEVAR and 49% were Delayed. 30-day mortality for the Early and Delayed groups was 16% and 0%, respectively. There was no difference in mortality based upon type of endovascular stent or whether carotid-subclavian bypass was performed. The mean ICU and total hospital LOS for TEVAR patients was 13 and 19 days, respectively (Table 1).

Pulmonary complications were the most common, occurring in 30% of TEVAR patients, followed by major infections (16%), cardiac arrest (6.5%), and deep vein thrombosis (6.5%) (Table 2). These complications occurred virtually exclusively in the Severe Injury (Grades 3-4) groups. There was one early re-intervention required due to proximal collapse of a Gore stent resulting in pseudo-coarctation on postoperative day 10. All patients had postoperative imaging prior to discharge and there were no early endoleaks. Long-term follow-up imaging was available in 36% of TEVAR patients at 1-year, 25% at 2-

years, 20% at 3-years, and 7% at 5-years. There were no late endoleaks or aortic interventions in cases where follow-up data was available.

Open Aortic Repair

OAR was performed in 17% of all Grade 1, 5.2% of Grade 2, 27% of Grade 3, and 46% of Grade 4 injuries (Table 1). All patients received immediate medical management upon presentation before undergoing OAR. Operative details included a left thoracotomy approach with repair performed using an interposition prosthetic graft of appropriate length depending on extent of injury. Pre-operative lumbar spinal drains were not routinely placed. When feasible, intraoperative motor-evoked and somatosensory-evoked potential monitoring were utilized. Full cardiopulmonary bypass support was utilized in 23% of cases, while left heart bypass was utilized in 53%. Mean total procedure, bypass, and cross-clamp (if applicable) times were 222$\pm$98.9 minutes, 98$\pm$79 minutes, and 56$\pm$47 minutes, respectively.

71% of OAR patients underwent Early surgery, usually due to frank rupture necessitating emergency operation. Overall 30-day mortality was 21%, with the Delayed group having lower mortality (5%) than the Early group (28%). There was no difference in mortality based upon bypass strategy, bypass duration, and extent of aortic replacement. Mean ICU and total hospital LOS for OAR patients was 11 and 16 days, respectively.

As with NOM and TEVAR, the most common complications were pulmonary in nature and occurred in 42% of the OAR group (Table 2). Major infections were the second most common (24%), followed by cardiac arrest (9%). All of these affected almost exclusively Grades 3-4. In patients where follow-up data was available (Table 3), there was one mortality after postoperative day 30: death from cardiac arrest on postoperative day 109 after suffering multiple retroperitoneal abscesses and persistent bile leak from a grade 4 liver laceration that had occurred at the time of initial injury.

Patient Characteristics by Thirty-Day Mortality

The entire cohort was evaluated to determine whether there were any variables that might predict 30-day survival. Older age, non-white race, higher ISS and AIS scores, lower admission BP, and intubation status were significantly associated with greater 30-day mortality while gender, injury mechanism, or blood product administration were not. As expected, Grade 4 had much higher mortality than others; Grade 4 patients who received NOM had a 130 times greater 30-day mortality risk than those who underwent TEVAR (p=0.012). Grade 1-3 patients who received NOM had a 7 times higher 30-day mortality risk than those who underwent TEVAR or OAR (Adj OR 6.97, CI 1.18-41.16, p=0.032). Delayed intervention with either TEVAR or OAR was associated with better 30-day survival than Early intervention (Adj OR 0.09, 95% CI 0.00-0.93, p= 0.043).

COMMENT

This study of BTAI represents one of the largest series of this rare but high-acuity diagnosis. Over the study period, our institution remained the primary referral center for major trauma and aortic pathology for the state of Indiana. We identified several notable findings. First, there was a shift over time in utilization of the different treatment options: before 2011, OAR was the most common strategy employed, but after 2011 there was a sharp decline in OAR with concomitant increase in TEVAR and NOM (Figure 1). Over the same period there was a corresponding decrease in Grade 4 incidence. . These changes coincide with the release of the 2011 Society of Vascular Surgery (SVS) clinical practice guidelines recommending TEVAR for Grade 2 to 4[8]. Because there are more institutions in our state offering TEVAR than OAR, and because of the critical nature of Grade 4 injury that makes inter-institutional transfer a high-risk endeavor, it is possible that the Grade 4 patients that would have been transferred to our center prior to 2011 are instead increasingly being treated definitively with TEVAR at other institutions.

Secondly, we find that NOM is a reasonable strategy for Mild Injury (Grades 1-2). The majority (75%) of our Grade 1 were treated with NOM, which mirrors the SVS and Eastern Association for the Surgery of Trauma (EAST) clinical practice guidelines. These guidelines also recommend TEVAR for

Grade 2 to 4, which was based upon then-available data demonstrating NOM mortality of 46% (the least favorable of the three treatment modalities)[8,13]. However, in publishing these guidelines the SVS acknowledged that the quality of evidence for BTAI management was "very low"[8]. In our cohort, 68% of all Grade 2 received NOM with subsequent 30-day mortality of 15%. Despite the SVS and EAST guidelines recommending TEVAR, a number of studies have reported NOM as a viable treatment strategy for Grade 2[9,14,15,16]. Dubose et al. analyzed nine Level 1 trauma centers and found that NOM in Grades 1 and 2 had "very low" rates of aorta-related mortality[15]. Similarly, Sandhu and colleagues conducted a 16-year retrospective analysis and proposed that Grade 2 can be successfully managed with NOM, while Spencer et al. compared NOM and TEVAR in patients with low grade BTAI and concluded that NOM is safe in Grade 2 injury[9,16]. Our practice is similar to these investigators, with the majority of Grades 1 and 2 undergoing NOM with acceptable results. Other institutions may pursue TEVAR for these patients, and we acknowledge this is also a reasonable approach. With conflicting published data on the question of optimal treatment for low grade BTAI, we believe further prospective study on this particular issue of patient and treatment selection will be greatly beneficial.

In our series, Grades 3 and 4 underwent definitive repair by either TEVAR or OAR unless the patient was too critically ill to tolerate procedural intervention or the severity of injuries was such that any intervention would be futile. The most common comorbid injuries that prevented procedural intervention included massive intracranial injury, intraabdominal exsanguination, and severe lung injury. For the TEVAR and OAR groups, our results are similar to those reported by others demonstrating superior outcomes with Delayed intervention. Marcaccio and associates reported a significantly lower mortality with Delayed intervention compared to Early (5.4% vs. 11.9%), and Estrera et al. also supported Delayed repair based on a 12-year analysis in which Delayed intervention had a 2% mortality (versus 27% mortality with Early intervention)[1,6]. An American Association for the Surgery of Trauma study showed that between 1997 and 2007 there were two notable trends: 1) a significant decrease in overall BTAI mortality, and 2) a concomitant increase in the average time interval between injury and treatment from 16 hours in 1997 to 54 hours in 2007, which suggested that the mortality improvements could be partially

attributable to delayed intervention[18]. Based on such results, the 2015 EAST guidelines recommended immediate NOM with a focus on aggressive BP control followed by Delayed repair[3,8,17,19,20].

Our data support this approach of immediate medical correction of major metabolic derangements and unstable physiology prior to TEVAR or OAR. The majority of TEVAR in our study was performed as a Delayed procedure. The majority of OAR were performed prior to 2011 as Early interventions, but the few OAR after 2011 were Delayed interventions and had corresponding improved survival. Currently, we recommend TEVAR instead of OAR unless one of the following is present: a) the aorta caliber is too small to safely accept an endograft without significant oversizing; b) the patient is younger than 20 years old; c) the aortic injury is located in the arch between the left common carotid and subclavian artery. Grade 3 and 4 patients typically have other co-morbid injuries, and heparinization—even if only for a brief duration—during TEVAR certainly poses a risk for exacerbating other hemorrhagic trauma. Our practice is to discuss this risk with the appropriate specialists involved (e.g., neurosurgery), and if a multidisciplinary consensus is to treat the immediate life-threatening BTAI first, then we proceed with TEVAR with as short a duration of anticoagulation as feasible. We prefer to perform TEVAR in a hybrid room if available, but the procedure can certainly be performed in a regular operating room with portable fluoroscopy.

The types of TEVAR endografts utilized varied over the study period. Some of this is attributed to the development of newer generation devices and also to the inventory of devices available at our institution, which is primarily affected by the hospital's purchasing decisions that are beyond surgeons' control. Nonetheless, we found no difference in outcomes by device manufacturer or by specific device. Based on our experience, we believe that no single device or manufacturer is clearly superior to others.

One limitation of our study is inherent to its retrospective nature, which naturally reduces our ability to understand how treatments were selected and the rationale for performing Early versus Delayed intervention. Also, our study reports the experience of a single institution and therefore results may not be generalizable to others. An additional limitation is the low long-term follow-up rate. While we attempt follow-up in all our patients, we have found this to be difficult in the trauma population. Moreover, many

of our patients are transferred from all regions of the state, and this further reduces patients' willingness or ability to follow-up with our center. Lastly, since BTAI patients tend to be relatively young, future research should involve longer-term outcomes of TEVAR patients to better understand the durability of endovascular devices.

Conclusion

Growing experience with endovascular treatment options have significantly altered the management paradigm of BTAI. While TEVAR has become a first-line treatment option for most cases, institutions that treat BTAI patients must be facile in all management modalities including medical and open surgical repair. Future investigative efforts should include multi-institutional efforts at delineating optimal treatment algorithms based on predictive clinical factors as well as long term studies to assess outcomes to beyond the index hospitalization.

REFERENCES

1. Marcaccio CL, Dumas RP, Huang Y, Yang W, Wang GJ, Holena DN. Delayed endovascular aortic repair is associated with reduced in-hospital mortality in patients with blunt thoracic aortic injury. J Vasc Surg 2018;68:64-73.
2. Mousa AY, Dombrovskiy VY, Haser PB, Graham AM, Vogel TR. Thoracic aortic trauma: outcomes and hospital resource utilization after endovascular and open repair. Vascular 2010;18:250-5.
3. Trust MD, Teixeira PGR. Blunt Trauma of the Aorta, Current Guidelines. Cardiol Clin 2017;35:441-451.
4. Teixeira PG, Inaba K, Barmparas G, et al. Blunt thoracic aortic injuries: an autopsy study. *J Trauma* 2001;1:197-202.
5. Shackford SR, Dunne CE, Karmy-Jones R, et al. The evolution of care improves outcome in blunt thoracic aortic injury: A Western Trauma Association multicenter study. J Trauma Acute Care Surg 2017;83:1006-1013.
6. Estrera AL, Gochnour DC, Azizzadeh A, et al. Progress in the treatment of blunt thoracic aortic injury: 12-year single-institution experience. Ann Thorac Surg 2010;90:64-71.
7. Agostinelli A, Carino D, Borrello B, et al. Blunt traumatic injury to the thoracic aorta treated with thoracic endovascular aortic repair: a single-centre 20-year experience. Interact Cardiovasc Thorac Surg 2019;28:17-22.
8. Lee WA, Matsumura JS, Mitchell RS, et al. Endovascular repair of traumatic thoracic aortic injury: clinical practice guidelines of the Society for Vascular Surgery. J Vasc Surg 2011;53:187-92.
9. Sandhu HK, Leonard SD, Perlick A, et al. Determinants and outcomes of nonoperative management for blunt traumatic aortic injuries. J Vasc Surg 2018;67:389-398.
10. Grigorian A, Spencer D, Donayre C, et al. National Trends of Thoracic Endovascular Aortic Repair Versus Open Repair in Blunt Thoracic Aortic Injury. Ann Vasc Surg 2018;52:72-78.

11. Demetriades D, Velmahos GC, Scalea TM, 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.
12. Azizzadeh A, Keyhani K, Miller CC 3rd, Coogan SM, Safi HJ, Estrera AL. Blunt traumatic aortic injury: initial experience with endovascular repair. J Vasc Surg 2009;49:1403-8.
13. Murad MH, Rizvi AZ, Malgor R, et al. Comparative effectiveness of the treatments for thoracic aortic transection. J Vasc Surg 2011;53:193-199.
14. de Mestral C, Dueck A, Sharma SS, et al. Evolution of the incidence, management, and mortality of blunt thoracic aortic injury: a population-based analysis. J Am Coll Surg 2013;216:1110-5.
15. Dubose JJ, Azizzadeh A, Estrera AL, Safi HJ. Contemporary management of blunt aortic trauma. J Cardiovasc Surg 2015;56:751-62.
16. Spencer SM, Safcsak K, Smith CP, Cheatham ML, Bhullar IS. Nonoperative management rather than endovascular repair may be safe for grade II blunt traumatic aortic injuries: An 11-year retrospective analysis. J Trauma Acute Care Surg 2018;84:133-138.
17. Neschis DG, Scalea TM, Flinn WR, Griffith BP. Blunt aortic injury. N Engl J Med 2008;359:1708-16.
18. Demetriades D, Velmahos GC, Scalea TM, et al. Diagnosis and treatment of blunt thoracic aortic injuries: changing perspectives. J Trauma 2008;64:1415-9.
19. Fox N, Schwartz D, Salazar JH, et al. Evaluation and management of blunt traumatic aortic injury: a practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Nurs 2015;22:99-110.
20. Karmy-Jones R, Ferrigno L, Teso D, Long WB 3rd, Shackford S. Endovascular repair compared with operative repair of traumatic rupture of the thoracic aorta: a nonsystematic review and a plea for trauma-specific reporting guidelines. J Trauma 2011;71:1059-72.

Table 1. Patient characteristics overall and by treatment group

Variable	Total (n=229)	TEVAR (n=61)	OAR (n=66)	NOM (n=102)	*p*
Age[a]	45.8±19.7 years	45.4±15.7	40.6±20.0	49.5±19.7	0.0133
Gender					0.525
Male	161(70.3)	42(68.9)	50(75.8)	69(67.7)	
Female	68(29.7)	19(31.2)	16(24.2)	33(32.4)	
Race					0.952
White	182(79.5)	49(80.3)	52(78.8)	81(79.4)	
Non-white	47(20.5)	12(19.7)	14(21.2)	21(20.6)	
SBP[a]	122±32 mmHg	121±26	125±35	121±33	0.6617
Intubation status					0.148
Not intubated	215(93.9)	59(96.7)	63(95.5)	93(91.2)	
Intubated	14(6.1)	2(3.3)	3(4.6)	9(8.8)	
Mechanism of injury					<0.0001
Fall	12(5.2)	4(6.6)	2(3.0)	6(5.9)	
MVA—single	73(31.9)	20(32.8)	14(21.2)	39(38.2)	
MVA—multiple	62(27.1)	20(32.8)	10(15.2)	32(31.4)	
MVA—unspecified	54(23.6)	9(14.8)	33(50.0)	12(11.8)	
Other	28(12.2)	8(13.1)	7(10.6)	13(12.8)	
Timing of procedure					<0.0001
Early	78(61.4)	31(39.7)	47(60.3)	N/A	
Delayed	49(38.6)	30(61.2)	19(38.8)	N/A	
Imaging					0.035
CT	210(91.7)	60(98.4)	61(92.4)	89(87.3)	

Other	19(8.3)	1(1.6)	5(7.6)	13(12.8)	
Injury grade					<0.0001
I	69(30.1)	5(8.2)	12(18.2)	52(51.0)	
II	19(8.3)	5(8.2)	1(1.5)	13(12.8)	
III	69(30.1)	29(47.5)	19(28.8)	21(20.6)	
IV	72(31.4)	22(36.1)	34(51.5)	16(15.7)	
Blood products given					<0.0001
Yes	78(34.1)	32(52.5)	7(10.6)	39(38.2)	
No	151(65.9)	29(47.5)	59(89.4)	63(61.8)	
ISS[a]	37.6±13.6	37.9±11.9	37.5±12.7	37.6±13.6	0.9778
AIS[a]					
Chest	4.5±0.6	4.7±0.5	4.6±0.5	4.4±0.6	0.0026
Head, Neck	3.0±1.2	3.0±1.0	2.9±1.1	3.1±1.2	0.8543
Face	1.8±0.5	1.7±0.6	2.0±0.5	1.7±0.5	0.1966
Abdomen	2.9±1.0	2.8±0.8	3.2±1.1	2.9±1.1	0.1913
External	1.1±0.4	1.1±0.4	1.1±0.2	1.2 ±0.5	0.4075
Extremity	2.4±0.7	2.5±0.7	2.3±0.7	2.5±0.7	0.3527
Hospital days[a]	14.2±13.4	19.2±15.3	15.7±14.7	10.8±10.7	0.0003
ICU days[a]	10.2±0.4	13.1±10.2	11.3±12.1	7.0±8.4	0.0003

Values are expressed as number(%) unless otherwise indicated. a, mean±standard deviation; AIS, abbreviated injury scale; CT, computed tomography; ICU, intensive care unit; ISS, injury severity score; MVA, motor vehicle accident; NOM, nonoperative management; OAR, open aortic repair; SBP, systolic blood pressure; TEVAR, thoracic endovascular aortic repair

Table 2. Complications by treatment type and injury severity

	All	Mild Injury (Grade 1 +Grade 2)	Severe Injury (Grade 3 + Grade 4)
NOM			
Pulmonary	21(20.6)	15(71.4)	6(28.6)
Major infections	13(12.7)	10(76.9)	3(23.1)
Cardiac arrest	10(9.8)	1(10)	9(90)
Urinary tract infection	6(5.9)	5(83.3)	1(16.7)
Deep vein thrombosis	6(5.9)	4(66.7)	2(33.3)
TEVAR			
Pulmonary	18(29.5)	5(27.8)	13(72.2)
Major infections	10(16.4)		10(100)
Cardiac arrest	4(6.6)		4(100)
Deep vein thrombosis	4(6.6)		4(100)
Urinary tract infection	3(4.9)		3(100)
OAR			
Pulmonary	28(42.4)	7(25)	21(75)
Major infections	16(24.2)		16(100)
Cardiac arrest	6(9.1)	1(16.7)	5(83.3)
Deep vein thrombosis	5(7.6)		5(100)
Urinary tract infection	2(3.0)	1(50)	1(50)

Values are expressed as number(%).

Table 3. Post-discharge follow-up availability

Follow-up (years)	TEVAR (n=61)	OAR (n=66)	NOM (n=102)
1	40(65.6)	36(54.5)	41(40.1)
2	31(50.8)	30(45.5)	37(36.3)
5	13(21.3)	23(34.8)	19(18.6)

Values are expressed as number(%) unless otherwise indicated.

Table 4. TEVAR patient volume and outcomes by year and by device manufacturer

Year	**TEVAR Device Manufacturer**					
	Gore	Cook	Medtronic	Cordis	Bolton	**Total**
2007	-	-	-	1	-	**1**
2008	7	-	-	-	-	**7**
2009	-	-	6	-	-	**6**
2010	1	1	2	-	-	**4**
2011	1	1	-	-	-	**2**
2012	4	1	-	-	-	**5**
2013	8	-	-	-	-	**8**
2014	2	1	-	-	1	**4**
2015	8	2	2	-	-	**12**
2016	3	3	-	-	-	**6**
2017	2	3	-	-	1	**6**
Total	**36**	**12**	**10**	**1**	**1**	**61**
Mortality						
30-day	2 (5.6)[a]	1 (8.3)[b]	1 (10.0)[c]	-	-	**4**
> 30-day	-	1 (8.3)[d]	-	-	-	**1**
Reintervention						
30-day	1 (2.8)[e]	-	-	-	-	**1**
> 30-day	-	-	-	-	-	**0**
Endoleak						
30-day	-	-	-	-	-	**0**
> 30-day	-	-	-	-	-	**0**

Values are expressed as number(%) unless otherwise indicated. a, one mortality due to cardiac arrest, one due to brain death; b, mortality due to cardiac arrest; c, mortality due to brain death; d, mortality

due to respiratory failure on postoperative day 54; e, reintervention on postoperative day 10 due to collapse of proximal stent resulting in pseudocoarctation; >30-day results based only on patients with follow-up data available

FIGURE LEGENDS

Figure 1. Year to year incidence of patients by injury severity.

Figure 2. Blunt thoracic aortic injury treatment selection over time. NOM, nonoperative management; OAR, open aortic repair; TEVAR, thoracic endovascular aortic repair.