

Determinants and outcomes of nonoperative management for blunt traumatic aortic injuries



Harleen K. Sandhu, MD, MPH,^a Samuel D. Leonard, MS,^a Alexa Perlick, BS,^a Naveed U. Saqib, MD, FACS,^{a,b} Charles C. Miller III, PhD,^a Kristofer M. Charlton-Ouw, MD, FACS,^{a,b} Hazim J. Safi, MD, FACS,^{a,b} and Ali Azizzadeh, MD, FACS,^{a,b} *Houston, Tex*

ABSTRACT

Objective: The natural history and parameters for successful nonoperative management of blunt traumatic aortic injuries (BTAIs) involving the descending aorta are poorly understood. We examined our experience with nonoperative BTAI treatment (anti-impulse, blood pressure) and evaluated for determinants of successful outcomes.

Methods: We performed a review of our institutional prospective trauma registry database for all BTAI patients from 1999 to 2015. Computed tomography angiography was used to classify aortic injuries on the basis of severity: grade I, intimal tear; grade II, intramural hematoma; grade III, aortic pseudoaneurysm; and grade IV, free rupture. Grade IV injuries were excluded from nonoperative management. Baseline characteristics, clinical outcomes, and follow-up lesion resolution were compared within the medically managed cohort and between surgical and nonoperative groups using univariate and multivariable analysis.

Results: Among 338 BTAI patients admitted between 1999 and 2015, 67 BTAI patients were managed nonoperatively; 26 (54%) had grade I BTAI, 22 (46%) had grade II, and 2 (4%) had grade III. Both grade III injuries required a late thoracic endovascular aortic repair after initial medical management and were excluded from analysis. In all, 48 were managed with initial medical therapy, and the remaining 19 died on admission or before definitive treatment. Among the 48 medically managed, the median age was 34 years, and 14 (29%) were female. Six of the 48 (12%) were transferred from other facilities. There was no significant difference in baseline characteristics or early outcomes between BTAI grades. Median injury resolution time was 39 days for grade I and 62 days for grade II ($P = .03$). Compared with a surgical cohort, BTAI grade and Abbreviated Injury Scale score for the chest were the only significant determinants of propensity to operate.

Conclusions: Based on these limited data, it appears that patients with minimal aortic injuries (grades I and II) may be managed medically, with the majority resolving within 8 weeks. Minimal aortic injury is associated with low mortality and excellent intermediate-term outcomes. Further prospective studies are required to validate these findings. (*J Vasc Surg* 2018;67:389-98.)

Thoracic blunt traumatic aortic injury (BTAI) is associated with significant mortality and is the second most common cause of death, following trauma.¹ Historical estimates have reported 80% to 85% prehospital mortality attributable to BTAI patients, and 15% to 23% of those

who survived to reach the hospital would eventually succumb to associated injuries during the initial 24 hours.²⁻⁴ Contemporary analysis of the National Trauma Data Bank suggests a significant contribution of major concomitant injuries (head, 31%; abdominal, 29%) to the overall morbidity in these patients, as indicated by their high Injury Severity Score (ISS).⁴ Given the multi-organ nature of the injuries in most patients with BTAI, thoracic endovascular aortic repair (TEVAR) has quickly replaced open repair as the treatment of choice for most anatomically suitable patients with BTAI.⁵⁻⁸

Based on severity, BTAI is classified into four grades: grade I, intimal tear; grade II, intramural hematoma; grade III, pseudoaneurysm; and grade IV, rupture (Fig 1).⁷ Current Society for Vascular Surgery clinical practice guidelines recommend nonoperative management for grade I BTAIs and an urgent (<24 hours) TEVAR (or open repair if anatomically unsuitable) for grade II to grade IV BTAIs. Despite the societal recommendations and practice guidelines, the optimal management approach for grade II BTAI (intramural hematoma) remains controversial. Recent data from multicenter

From the Department of Cardiothoracic and Vascular Surgery, McGovern Medical School at the University of Texas Health Science Center at Houston (UTHHealth)^a; and Memorial Hermann Heart & Vascular Institute-Texas Medical Center.^b

Author conflict of interest: A.A. and K.C.O. are consultants to W. L. Gore & Associates and Medtronic.

Presented at the Forty-first Annual Meeting of the Southern Association for Vascular Surgery, Naples, Fla, January 18-21, 2017.

Correspondence: Ali Azizzadeh, MD, FACS, Professor and Chief, Division of Vascular Surgery, Department of Cardiothoracic and Vascular Surgery, McGovern Medical School at UTHHealth, 6400 Fannin St, Ste 2850, Houston, TX 77030 (e-mail: ali.azizzadeh@uth.tmc.edu).

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 conflict of interest.

0741-5214

Copyright © 2017 by the Society for Vascular Surgery. Published by Elsevier Inc. <http://dx.doi.org/10.1016/j.jvs.2017.07.111>

studies report favorable outcomes after nonoperative management among patients who presented with minimal aortic injury (grades I and II BTAI).^{7,9-12} However, natural history and parameters for successful nonoperative management of these minimal BTAIs remain elusive.

In view of the paucity of evidence on the role of nonoperative management in minimal aortic injuries (grades I and II), we examined our experience with medical (anti-impulse, blood pressure) management of BTAI and evaluated for determinants of successful clinical outcomes and resolution of injury.

METHODS

This retrospective study was approved by McGovern Medical School at UTHealth's Committee for the Protection of Human Subjects, which serves as our local Institutional Review Board, and was conducted in compliance with Health Insurance Portability and Accountability Act regulations. Given the retrospective nature of the study, informed consent was waived for analysis. We queried our prospective institutional trauma registry from September 1999 to December 2015 and identified all patients with the diagnosis of BTAI. Because TEVAR was introduced in our practice in 2005, we included all patients from 2004 to 2015 in the analysis to remove any bias introduced by the open era (before introduction of TEVAR) and kept all analysis limited to the endovascular era. We identified 273 patients from 2004 to 2015 who suffered BTAI. Fig 2 represents a schematic for selection of patients.

Data on patient demographics, physiologic status, and admission injury severity (ISS and Abbreviated Injury Scale [AIS]) were collected. A trained radiologist and vascular surgeon reviewed all available computed tomography (CT) angiography imaging to ensure agreement on BTAI grading. Once characterized, specific measurements of individual injuries were made, in addition to examination of associated variables, including presence of associated mediastinal hematoma, diameter of lesion (for pseudoaneurysms), and depth of the mediastinal hematoma at the level of the aortic arch and the descending thoracic aorta.

We have previously described our treatment algorithm, including indications, as well as our techniques for open repair and TEVAR (Fig 3).^{7,13,14} The early procedures were conducted with off-label use of the TAG device (W. L. Gore & Associates, Flagstaff, Ariz), the first thoracic aortic device approved by the U.S. Food and Drug Administration, which was available in diameters of 26 to 40 mm, intended for patients with aortic diameters of 23 to 37 mm. During this phase, young trauma patients with aortic diameters <23 mm were treated with open repair. The approval of additional smaller diameter devices in 2008 enabled us to apply TEVAR to a wider range of patients. As a result, TEVAR gradually replaced open repair as the treatment of choice for all anatomically

ARTICLE HIGHLIGHTS

- **Type of Research:** Single-center retrospective analysis of prospectively collected data
- **Take Home Message:** There were 48 patients with blunt traumatic descending thoracic aortic injuries managed nonoperatively because of grade I (intimal tear) or grade II (intramural hematoma) aortic injuries. The majority of injuries resolved within 8 weeks. There was no aorta-related mortality.
- **Recommendation:** These data suggest that most minimal descending thoracic aortic injuries (grades I and II) resolve within 8 weeks with medical therapy alone.

suitable patients with thoracic aortic injuries at our center. The progressive shift from open repair to TEVAR for repair of BTAI at our institution is demonstrated in Fig 4.

Most of the data on presentation, admission management, and outcomes were abstracted from the trauma registry and follow-up. In-hospital variables were collected through review of individual patients' records. When available, causes of death were ascertained and categorized as stroke, refractory hemorrhage with hemodynamic instability, traumatic brain injury, cardiac arrest not related to ongoing hemorrhage, sepsis secondary to contamination or infection, respiratory failure, multiorgan failure, and aorta-related mortality (ARM). ARM was defined as death directly attributed to BTAI or complications of BTAI treatment. Autopsy results were not available in all patients; therefore, a detailed review of patients' hospital, operative, and radiologic records was performed to make an ARM determination.

Retrospective review of medical records included available outpatient follow-up clinical and radiologic data.

Statistical analysis. Analysis was performed in two separate stages. The first stage involved assessment of the clinical correlates and outcomes of nonoperative management as well as determination of the factors associated with injury resolution on follow-up. For this analysis, 150 surgically managed patients were excluded (Fig 2). Of these 150 patients, 108 were managed with TEVAR and 42 had open repair. There were a total of 28 minimal aortic injuries in this surgical group of 150 patients, and 23 of them received TEVAR. In addition, 59 patients died before a confirmatory CT scan could be performed. Of the remaining 64 patients, two patients with grade III injuries were initially stabilized using our nonoperative management protocol for an intentionally delayed TEVAR, and 14 patients died soon after transfer or admission secondary to nonsurvivable injuries (mean ISS of 40, severe aortic injury and head trauma or cardiac arrest). Therefore, these cases were excluded from the study cohort because the conservative management was

CLASSIFICATION OF TRAUMATIC AORTIC INJURY

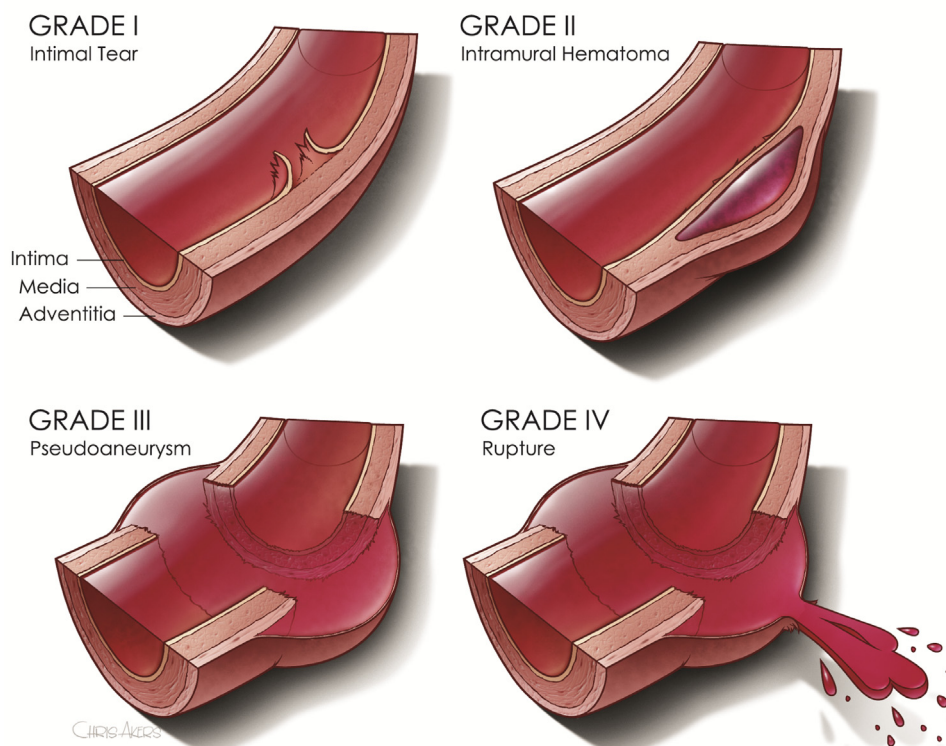


Fig 1. Illustration of the classifications of traumatic aortic injury.

not definitive (delayed TEVAR or palliative intent). The remaining 48 patients with 26 grade I and 22 grade II BTAs constituted the study cohort for the principal (or first-stage) analysis. Adjusted analyses were conducted by multivariable statistics using multiple logistic regressions. Healing rates were estimated by plotting cumulative frequencies of healing, stratified by injury grade, with actuarialized denominators.

To better define the group of patients with grade II BTAs who might be candidates for TEVAR, we performed a secondary (or second-stage) analysis involving all BTAI patients who were diagnosed with grade II BTAI. This presents the most significant area of treatment uncertainty and was assessed as a subgroup. For this level of analysis, we compared the 22 nonoperatively managed BTAs with 23 TEVARs performed for grade II BTAs during the same time period. Because the total number of adverse events was small, propensity scores were computed for medical vs surgical treatment in the grade II injury subcohort, and propensity-adjusted analysis of treatment effect was conducted.

Traditional risk factor-adjusted treatment effect analyses were also conducted by appropriate regression methods. We then examined the independent and joint effects of treatment allocation (nonoperative vs TEVAR) and other risk factors on short-term outcomes.

Computations were performed using SAS software version 9.4 (SAS Institute, Cary, NC). Continuous variables with a normal distribution are reported using means \pm standard deviation; those that were not normally distributed are reported using median and interquartile range. Descriptive statistics were computed by contingency table methods for discrete variables and unpaired *t*-test or Wilcoxon rank sum, depending on data distribution. Multivariable analyses for predictors of short-term outcomes were conducted using multiple logistic regression, and additional attributable-risk estimates were computed using generalized linear models. These models present adjusted percentage risk for calculated outcome that can be attributable to the risk factor. Resolution rate and survival were assessed using Kaplan-Meier analysis and Cox proportional hazards regression.

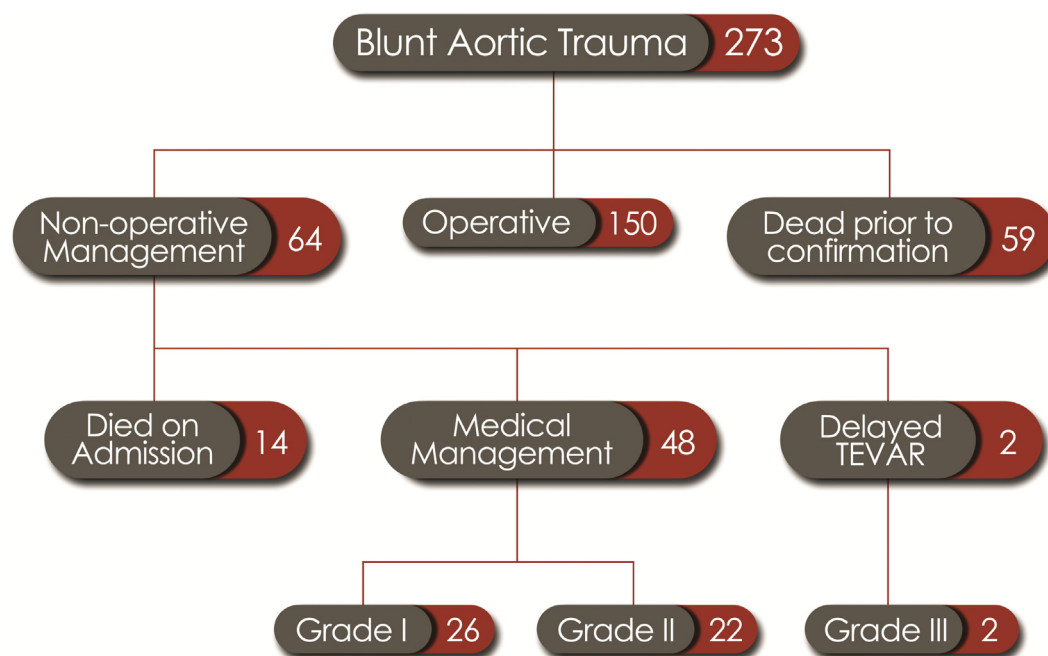


Fig 2. Flow diagram depicting the process of the study population selection from the overall cohort of patients with blunt traumatic aortic injury (BTAI). TEVAR, Thoracic endovascular aortic repair.

RESULTS

Stage 1 analysis: Nonoperative management of grade I vs grade II BTAs

Characteristics of the patients. Demographics and presenting features of the patients are described in Table I. Although not statistically different given the small numbers, overall, grade II patients were slightly older and were frequently male and illicit drug users, with severe abdominal AIS (AIS code of 4 and above) and altered mental status, and had higher admission heart rate and blood pressure. Grade I patients commonly reported symptoms such as chest pain, shortness of breath, leg pain, and back pain. However, this did not reach statistical significance. Mean ISS was 34.7 ± 9.9 and was higher among grade II BTAs. All patients received radiologic confirmation using CT scan, angiography, or intravascular ultrasound. Incidence of mediastinal hematoma was significantly higher among grade II BTAs (46% vs 15%; $P = .02$).

Clinical outcomes. Table II depicts the clinical and follow-up outcomes comparing grade I BTAs with grade II. There were no aorta-related deaths in this group. All-cause mortality was 4.2%. Both of these deaths occurred in patients who suffered a concomitant traumatic brain injury and succumbed to it. Overall, in the medically managed population (grades I and II), age >35 years (odds ratio [OR, 13.8; $P < .025$) and increasing ISS (OR, 1.10/ISS; $P = .05$) were independently associated with increased adverse outcome (a composite variable including in-hospital acute renal failure, stroke, paraplegia, or death within 30 days; Table III).

Injury resolution. Follow-up CT scans were available for 70.8% patients. In all, 27 of 32 (84%) resolved (56% of study cohort) and 5 of 32 (16%) had persistent injury (10% of study cohort). Median resolution time was 6 (interquartile range, 3-8) weeks. On univariate analysis, patients who presented with mediastinal hematoma on imaging had lower resolution (4/14 [28.6%]) on follow-up imaging compared with those who had no mediastinal hematoma (23/34 [67.7%]; OR for failure to heal, 5.23; $P < .013$). Except for higher Glasgow Coma Scale score at admission (13.3 ± 3.6 vs 10.1 ± 5.6 ; $P = .027$) and revised trauma scores (7.3 ± 1.1 vs 6.1 ± 2.1 ; $P = .038$) among those who resolved, no other presenting or baseline characteristics were significantly different among those who resolved as opposed to those who did not. Resolution of BTAI tended to occur more quickly among grade I injuries compared with grade II (Fig 5; $P = .03$).

Stage 2 analysis: Nonoperative management vs TEVAR in grade II BTAs

Characteristics of the patients and unadjusted outcomes. Clinical demographics and outcomes comparing medically managed grade II BTAs with TEVAR are detailed in Table IV. Patients with grade II BTAs had a mean age of 39.4 ± 14.9 years, and they were frequently male.

Patients who underwent TEVAR were more likely to be transferred from other institutions for further management and were slightly older with lower admission blood pressure and heart rate compared with the nonoperative group. Nonoperatively managed patients tended to have more severe abdominal AIS ($P = .039$) and ISS ($P = .058$).

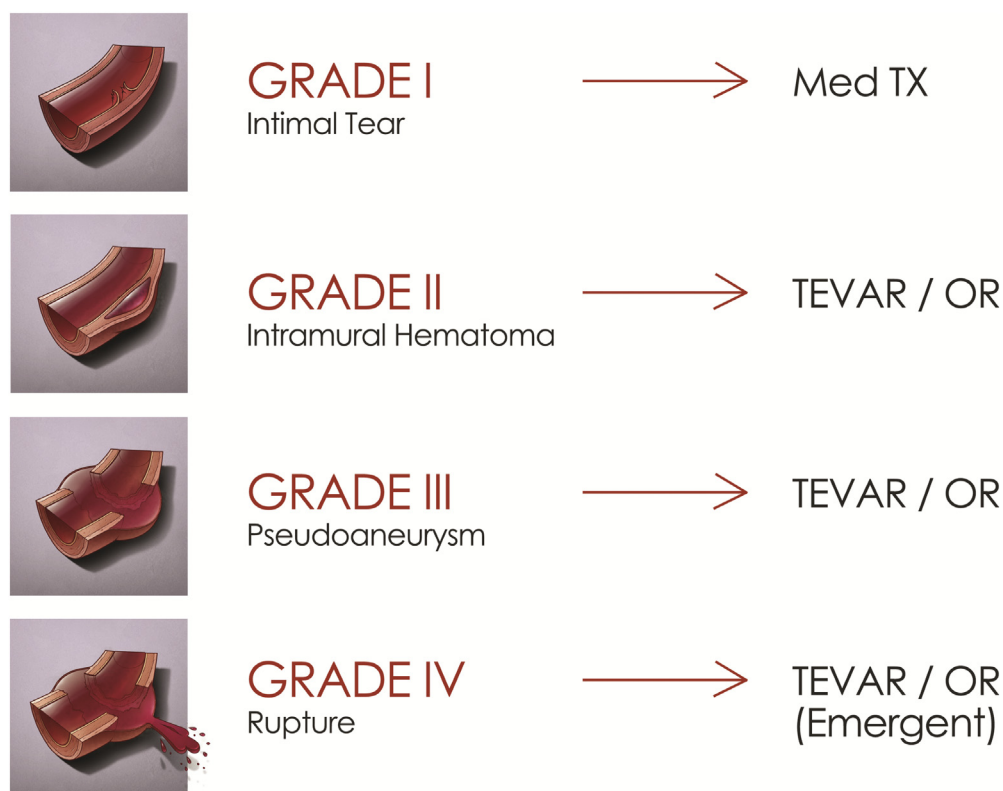


Fig 3. Treatment algorithm for management of blunt traumatic aortic injury (BTAI) by grade of injury. *Med Tx*, Medical treatment; *OR*, open repair; *TEVAR*, thoracic endovascular aortic repair.

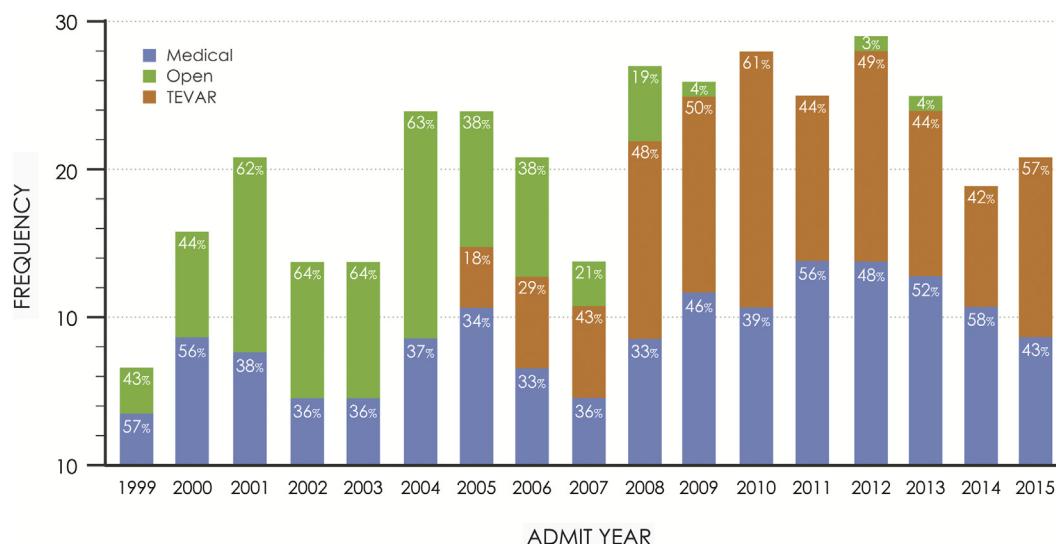


Fig 4. Longitudinal trends in blunt traumatic aortic injury (BTAI) management during the 17-year span: year-to-year variability in the distribution of BTAI patients by various management approaches. *TEVAR*, Thoracic endovascular aortic repair.

There were more cases of acute renal failure among non-operatively managed BTAs, although stroke and other clinical outcomes were similar to those of TEVAR patients. There was no ARM in either group. No deaths were reported in the TEVAR group, whereas there was one death (cause of death was attributed to traumatic brain injury) in the nonoperative BTAI group.

Propensity for TEVAR and propensity-adjusted outcomes. Within the grade II injury subcohort, propensity for medical vs surgical treatment was determined by age <35 years, the total ISS, and presence of arch hematoma. The final model for propensity for TEVAR was constructed [$\exp(-2.7258 + (\text{Age} > 35 * -1.6053) + (\text{ISS} * 0.1046) + (\text{arch hematoma} * 1.7979))$] and indicated

Table I. Characteristics of patients of nonoperative management cohort

Variable	Total (N = 48; 100%)	Grade I (n = 26; 54.2%)	Grade II (n = 22; 45.8%)	P value
Male	34 (70.8)	18 (69.2)	16 (72.7)	.791
Female	14 (29.2)	8 (30.8)	6 (27.3)	.791
Age, years	37.5 ±15.1	36.7 ±14.6	38.5 ±15.8	.678
Accident to admission, hours	1.0 (0.8-1.1)	1.1 (0.9-1.3)	0.9 (0.7-1.1)	.267
CAD	2 (4.2)	0 (0.0)	2 (9.1)	.116
COPD	1 (2.1)	0 (0.0)	1 (4.6)	.458
Diabetes	4 (8.3)	1 (3.9)	3 (13.6)	.320
Hyperlipidemia	2 (4.2)	1 (3.9)	1 (4.6)	1.000
Hypertension	12 (25.0)	6 (23.1)	6 (27.3)	.738
Smoking	24 (50.0)	13 (50.0)	11 (50.0)	1.000
Alcohol	12 (25.0)	5 (19.2)	7 (31.8)	.316
Illicit drug use	8 (16.7)	3 (11.5)	5 (22.7)	.442
Cocaine	3 (6.3)	0 (0.0)	3 (6.3)	.052
Marijuana	5 (10.4)	2 (7.7)	3 (6.3)	.649
Methamphetamine	1 (2.1)	0 (0.0)	1 (4.6)	.458
Opiates	3 (6.3)	1 (3.9)	2 (9.1)	.587
Hypotension	14 (29.2)	9 (34.6)	5 (22.7)	.367
Diaphoresis	1 (2.1)	0 (0.0)	1 (4.6)	.458
Chest pain	16 (33.3)	12 (46.2)	4 (18.2)	.041
Shortness of breath	10 (20.8)	7 (26.9)	3 (13.6)	.307
Abdominal pain	9 (18.8)	6 (23.1)	3 (13.6)	.478
Back pain	7 (14.6)	4 (15.4)	3 (13.6)	.864
Altered mental status	21 (43.8)	9 (34.6)	12 (54.6)	.166
Paraparesis	2 (4.2)	1 (3.9)	1 (4.6)	1.000
Leg pain	10 (20.8)	6 (23.1)	4 (18.2)	.735
Severe AIS: abdomen	12 (25.0)	4 (15.4)	8 (36.4)	.094
AIS: abdomen	2 (1-3.5)	2 (0-3)	3 (2-4)	.086
Severe AIS: chest	47 (97.9)	25 (96.2)	22 (100)	1.000
AIS: chest	4.1 ± 0.2	4.0 ± 0.2	4.1 ± 0.3	.487
AIS: extremity	2.6 ± 0.7	2.6 ± 0.7	2.5 ± 0.8	.527
AIS: head	3 (2-4)	3 (2-4)	3 (2.5-4)	1.000
Admission SBP, mm Hg	116 (96-136)	105 (92-138)	126 (101-132)	.392
Admission heart rate, beats/min	100 (90-110)	99 (90-104)	102.5 (93-116)	.069
Admission hemoglobin level, mg/dL	14 (12.3-14.8)	14.1 (12.3-15.0)	13.9 (11.9-14.6)	.551
Revised trauma score	6.8 ± 1.7	6.9 ± 1.6	6.7 ± 1.8	.583
Admission GCS score	15 (10-15)	15 (13.5-15)	14 (9-15)	.425
ISS	34.7 ± 9.9	33.5 ± 10.2	36.1 ± 9.7	.367
CT for confirmation	47 (97.9)	25 (96.2)	22 (100)	1.000
Angiography or IVUS	10 (20.8)	5 (19.2)	5 (22.7)	1.000
Mediastinal hematoma	14 (29.2)	4 (15.4)	10 (45.8)	.022
Arch hematoma	5 (10.4)	1 (3.9)	4 (18.2)	.165
DTA hematoma	11 (22.9)	4 (15.4)	7 (31.8)	.302
30-Day death ^a	2 (4.2)	1 (3.9)	1 (3.9)	1.000

AIS, Abbreviated Injury Scale; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; CT, computed tomography; DTA, descending thoracic aorta; GCS, Glasgow Coma Scale; ISS, Injury Severity Score; IVUS, intravascular ultrasound; SBP, systolic blood pressure; Severe AIS, scores ≥4.

Categorical variables are presented as number (%). Continuous variables are presented as mean ± standard deviation or median (interquartile range).

^aBoth deaths occurred secondary to traumatic brain injury.

Table II. Outcomes among patients managed nonoperatively for blunt traumatic aortic injury (BTAI)

Variable	Total (N = 48; 100%)	Grade I (n = 26; 54.2%)	Grade II (n = 22; 45.8%)	P value
Stroke	2 (4.2)	1 (3.9)	1 (4.6)	1.000
Paraplegia	1 (2.1)	1 (3.9)	0 (0.0)	1.000
Acute renal failure	6 (12.5)	2 (7.7)	4 (18.2)	.392
ICU LOS, days	4.5 (2-14)	4 (2-14)	5.5 (3-15)	.416
Hospital LOS, days	17 (8-24.5)	17 (8-24)	17 (8-46)	.242
Resolution	27 (56.3)	17 (65.4)	10 (45.5)	.166
Persistent	5 (10.4)	2 (7.7)	3 (13.6)	.649
Resolution time, days	39 (22-62)	35 (21-55)	61 (32-75)	.079
Aortic mortality	0 (0.0)	0 (0)	0 (0)	1.000
All-cause death	2 (4.2)	1 (4)	1 (5)	1.000

ICU, Intensive care unit; LOS, length of stay.
Categorical variables are presented as number (%). Continuous variables are presented as median (interquartile range).

Table III. Independent predictors of poor short-term outcomes of nonoperatively managed blunt traumatic aortic injuries (BTAs)

Variable	Odds ratio ^a	95% Confidence interval ^a	Attributable risk ^b	P value ^b
Age >30 years	13.8	1.4-136.3	27.3% ± 10.4%	.012
ISS	1.1	1.0-1.2	1.0% ± 0.5%	.053

ISS, Injury Severity Score.
Poor outcomes composite includes in-hospital acute renal failure, stroke, paraplegia, or death within 30 days.
^aDerived from multiple logistic regression analysis.
^bDerived from generalized linear model analysis.

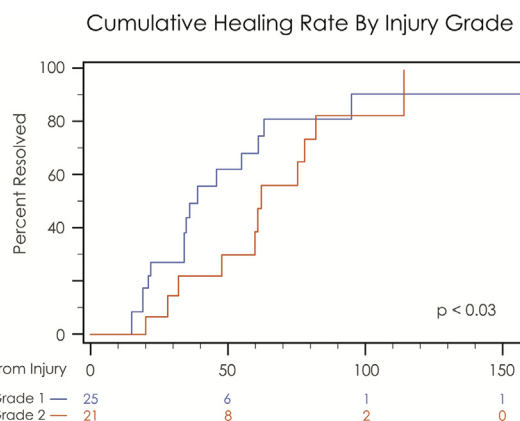


Fig 5. Cumulative blunt traumatic aortic injury (BTAI) healing curves by grade of injury. This figure shows a comparison of cumulative probability of injury resolution (healing rate) among grade I vs grade II BTAs after nonoperative management.

that medical therapy is more common for greater severity of injury (arch hematoma and increasing ISS) and for younger patients. After adjustment for propensity to receive medical therapy, use of medical therapy did not significantly affect adverse outcome (stroke, paraplegia, renal failure, or death) in grade II injuries (OR, 1.54; $P < .0676$). After propensity-adjusted multivariable analysis for poor outcomes, only ISS appeared to be a

significant risk factor (OR, 1.2; 95% confidence interval, 1.00-1.51; $P = .04$).

DISCUSSION

This study reports data from a group of patients who presented to one of the largest trauma centers in the United States during an 11-year period. The retrospective review is based on patient information collected by the Memorial Hermann Trauma Registry with supplemental information from our departmental database. The analysis represents one of the largest retrospective reviews investigating nonoperative management of grades I and II BTAI. Current Society for Vascular Surgery guidelines recommend TEVAR for aortic injury grades II to IV, although recent retrospective reviews have shown that observation may be safe in selected cases of BTAI involving intramural hematomas.^{7,9-12} In a recent study by Rabin et al,¹² the authors reported significant success in treatment of grade I and grade II aortic injuries through nonoperative management, with 31% death attributed to secondary injury including pseudocoarctation, extensive mediastinal hematoma, and large left hemothorax.

Traditionally, nonoperative management consists of antihypertensive therapy with impulse control (negative inotropic therapy) and, when needed, supplementation with a vasodilator, all aimed at minimizing shear stress on the vessel through reduction in blood pressure and

Table IV. Outcomes among patients with grade II blunt traumatic aortic injury (BTAI) comparing nonoperative management with thoracic endovascular aortic repair (TEVAR)

Variable	Total (N = 45; 100%)	Nonoperative management (n = 22; 48.9%)	TEVAR (n = 23; 51.1%)	P value
Male	35 (77.8)	16 (72.7)	19 (82.6)	.491
Female	10 (22.2)	6 (27.3)	4 (17.4)	.491
Transfer	5 (11.1)	0 (0.0)	5 (21.7)	.049
Age, years	39.4 ± 14.9	38.5 ± 15.8	40.2 ± 14.3	.636
Severe AIS: abdomen	11 (24.4)	8 (36.4)	3 (13.0)	.069
AIS: abdomen	2 (0-3)	3 (2-4)	2 (0-3)	.039
Severe AIS: chest	44 (97.8)	22 (100)	22 (95.7)	1.000
AIS: chest	4.2 ± 0.4	4.0 ± 0.3	4.2 ± 0.4	.228
AIS: extremity	2.5 ± 0.7	2.5 ± 0.8	2.4 ± 0.6	.796
Admission SBP, mm Hg	116 (99-134)	126 (101-132)	112 (94-136)	.503
Admission heart rate, beats/min	98 (86-114)	102 (93-116)	93 (74-107)	.059
Admission GCS score	14 (10-15)	14 (9-15)	14 (11-15)	.550
ISS	30 (26-41)	39 (29-42)	29 (24-38)	.058
Mediastinal hematoma	17 (39.5)	10 (45.5)	7 (33.3)	.416
Arch hematoma	6 (13.9)	4 (18.2)	2 (9.5)	.664
DTA hematoma	14 (32.6)	7 (31.8)	7 (33.3)	.916
Stroke	2 (4.6)	1 (4.6)	1 (4.6)	1.000
Acute renal failure	5 (11.4)	4 (18.2)	1 (4.6)	.345
ICU stay, days	4.5 (3-14)	5.5 (3-15)	4 (3-12)	.486
LOS, days	17 (8.5-31.5)	17 (8-46)	16.5 (9-31)	.981
All-cause mortality	1 (2.2)	1 (4.6)	0 (0)	1.000

AIS, Abbreviated Injury Scale; DTA, descending thoracic aorta; GCS, Glasgow Coma Scale; ICU, intensive care unit; ISS, Injury Severity Score; LOS, length of stay; SBP, systolic blood pressure; Severe AIS, scores ≥4.
Categorical variables are presented as number (%). Continuous variables are presented as mean ± standard deviation or median (interquartile range).

cardiac contractility.¹⁵ Although there is considerable variation, standard nonoperative management therapy targets a systolic blood pressure between 100 and 120 mm Hg accompanied by a heart rate between 60 and 90 beats/min.^{12,15,16} Starnes et al reported guidelines that included antiplatelet therapy (aspirin 81 mg) if the patient could tolerate it.¹⁷ Fabian et al¹⁸ reported no aortic rupture in patients who were managed nonoperatively.

Therapy is aimed at preventing lesion progression or rupture.

We identified younger age as being related to medical management. We further assessed this effect during the study period and found that date of admission is a stronger predictor of TEVAR than age, which suggests a gradual adoption of TEVAR in younger patients as smaller diameter devices became available after 2005. We also found several measures of injury severity to be higher in medically managed patients, indicating that more severely injured patients were managed expectantly rather than with immediate TEVAR (Tables III and IV).

Standard protocol for duration of nonoperative antihypertensive therapy in grade I BTAI patients has not

been determined or extensively studied. We realize that standard nonoperative management of blood pressure and heart rate may be too aggressive to be maintained out of the hospital after discharge and may be difficult to study without formal ambulatory blood pressure monitoring. We recommend further investigation of post-hospital discharge variables influencing injury progression and resolution. Our data and current protocol suggest that discharge after CT imaging demonstrating stable injury is safe and is associated with a low probability of injury progression.

However, some institutions have reported that unrepaired injuries can progress to chronic pseudoaneurysms, which require repair or rupture within 1 year of hospital discharge.^{9,12,16} Our data demonstrated that persistent injuries can be present in up to 16% of patients, so the potential for elective outpatient repair of minor aortic injuries is present. Our results suggest that minimal aortic injury is associated with low mortality and excellent intermediate-term outcomes. In addition, follow-up imaging is a critical aspect of nonoperative management of BTAI that currently has no established protocols. Surveillance may not be necessary after discharge if the aortic lesion is radiographically resolved

before discharge. For lesions that persist, routine follow-up imaging before hospital discharge and at 1 month, 6 months, 12 months, and then annually appears reasonable.^{9,12,19}

Limitations. Our single-center study has important limitations that must be acknowledged. Treatment allocation is nonrandomized, and propensity-based methods have limited statistical power in a small sample. Selection of a cohort subjected to retrospective data analysis can be difficult to interpret, although use of a radiologist blinded to treatment group should help mitigate bias in this respect. In our study, the radiologists reviewed CT images but were not involved in direct clinical care of these patients. Most of the minimal injuries were managed medically, so although no formal protocol for blinding of the radiologists was in place, treatment did not vary in these patients and would not have influenced the readings with any foreseeable directional bias. The trauma registry from which the study population arises is comprehensive, and all trauma patients are entered into it. This may help reduce selection bias but also reduces the amount of detailed patient information that would be available to further stratify risk. Moreover, detailed patient information on time from injury to treatment initiation is difficult to obtain from such a registry in a retrospective manner.

Loss to follow-up is a common limitation in retrospective cohort studies involving the trauma population, specifically nonoperative medical management of aortic injuries. Follow-up imaging is required to determine resolution or progression, and in our population, we were able to obtain this in 71% at an average time of 40 days. Kidane et al²⁰ reported that half of the 36 retrospective studies included in their meta-analysis looked at only in-hospital outcomes, and as a result, most pointed to a null effect. Although we achieved excellent intermediate-term outcome, an additional limitation involves limited long-term outcome review of BTAI. As a result, we recommend further long-term prospective studies to validate these findings.

An ongoing initiative to address this objective is the international multicenter prospective registry supported by the nonprofit Aortic Trauma Foundation (www.aortictrauma.org), which aims to investigate the optimal treatment of patients with BTAI and to develop evidence-based, multidisciplinary practice guidelines.

CONCLUSIONS

Based on these limited data, it appears that minimal aortic injuries (grades I and II) may be managed medically, with the majority resolving within 8 weeks. Minimal aortic injury is associated with low mortality and excellent intermediate-term outcomes. Further prospective studies are required to validate these findings.

AUTHOR CONTRIBUTIONS

Conception and design: HKS, SL, AP, NS, CM, KCO, HJS, AA

Analysis and interpretation: HKS, CM, KCO, AA

Data collection: SL

Writing the article: HKS, SL, NS, CM, KCO, AA

Critical revision of the article: HKS, SL, AP, NS, CM, KCO, HJS, AA

Final approval of the article: HKS, SL, AP, NS, CM, KCO, HJS, AA

Statistical analysis: HKS, CM

Obtained funding: Not applicable

Overall responsibility: HKS

HKS and SL contributed equally to this article and share first authorship.

REFERENCES

1. Fox N, Schwartz D, Salazar JH, Haut ER, Dahm P, Black JH, et al. Evaluation and management of blunt traumatic aortic injury. *J Trauma Nurs* 2015;22:99-110.
2. Parmley LF, Mattingly TW, Manion WC, Jahnke EJ. Non-penetrating traumatic injury of the aorta. *Circulation* 1958;17:1086-101.
3. Fabian TC, Richardson JD, Croce MA, Smith JS, Rodman G, Kearney PA, et al. Prospective study of blunt aortic injury: multicenter trial of the American Association for the Surgery of Trauma. *J Trauma* 1997;42:374-80; discussion: 380-3.
4. Arthurs ZM, Starnes BW, Sohn VY, Singh N, Martin MJ, Andersen CA. Functional and survival outcomes in traumatic blunt thoracic aortic injuries: an analysis of the National Trauma Databank. *J Vasc Surg* 2009;49:988-94.
5. Lebl DR, Dicker RA, Spain DA, Brundage SI. Dramatic shift in the primary management of traumatic thoracic aortic rupture. *Arch Surg* 2006;141:177.
6. Lee WA, Matsumura JS, Mitchell RS, Farber MA, Greenberg RK, Azizzadeh A, 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.
7. Azizzadeh A, Keyhani K, Miller CC, Coogan SM, Safi HJ, Estrera AL. Blunt traumatic aortic injury: initial experience with endovascular repair. *J Vasc Surg* 2009;49:1403-8.
8. Kwolek CJ, Blazick E. Current management of traumatic thoracic aortic injury. *Semin Vasc Surg* 2010;23:215-20.
9. DuBose JJ, Leake SS, Brenner M, Pasley J, O'Callaghan T, Luo-Owen X, et al. Contemporary management and outcomes of blunt thoracic aortic injury: a multicenter retrospective study. *J Trauma Acute Care Surg* 2015;78:360-9.
10. Osgood MJ, Heck JM, Rellinger EJ, Doran SL, Garrard CL, Guzman RJ, et al. Natural history of grade I-II blunt traumatic aortic injury. *J Vasc Surg* 2014;59:334-41.
11. Paul JS, Neideen T, Tutton S, Milia D, Tolat P, Foley D, et al. Minimal aortic injury after blunt trauma: selective nonoperative management is safe. *J Trauma* 2011;71:1519-23.
12. Rabin J, DuBose J, Sliker CW, O'Connor JV, Scalea TM, Griffith BP. Parameters for successful nonoperative management of traumatic aortic injury. *J Thorac Cardiovasc Surg* 2014;147:143-9.
13. Azizzadeh A, Ray HM, Dubose JJ, Charlton-Ouw KM, Miller CC, Coogan SM, et al. Outcomes of endovascular repair for patients with blunt traumatic aortic injury. *J Trauma Acute Care Surg* 2014;76:510-6.
14. Estrera AL, Gochmour DC, Azizzadeh A, Miller CC, Coogan S, Charlton-Ouw K, et al. Progress in the treatment of blunt

- thoracic aortic injury: 12-year single-institution experience. *Ann Thorac Surg* 2010;90:64-71.
15. Caffarelli AD, Mallidi HR, Maggio PM, Spain DA, Miller DC, Mitchell RS. Early outcomes of deliberate nonoperative management for blunt thoracic aortic injury in trauma. *J Thorac Cardiovasc Surg* 2010;140:598-605.
 16. Mosquera VX, Marini M, Guliás D, Cao I, Muñiz J, Herrera-Noreña JM, et al. Minimal traumatic aortic injuries: meaning and natural history. *Interact Cardiovasc Thorac Surg* 2012;14:773-8.
 17. Starnes BW, Lundgren RS, Gunn M, Quade S, Hatsukami TS, Tran NT, et al. A new classification scheme for treating blunt aortic injury. *J Vasc Surg* 2012;55:47-54.
 18. Fabian TC, Davis KA, Gavant ML, Croce MA, Melton SM, Patton JH, et al. Prospective study of blunt aortic injury: helical CT is diagnostic and antihypertensive therapy reduces rupture. *Ann Surg* 1998;227:666-76; discussion: 676-7.
 19. Harris DG, Rabin J, Starnes BW, Khoynzhad A, Conway RG, Taylor BS, et al. Evolution of lesion-specific management of blunt thoracic aortic injury. *J Vasc Surg* 2016;64:500-5.
 20. Kidane B, Plourde M, Chadi SA, Iansavitchene A, Meade MO, Parry NG, et al. The effect of loss to follow-up on treatment of blunt traumatic thoracic aortic injury. *J Vasc Surg* 2015;61:1624-34.

Submitted Mar 23, 2017; accepted Jul 12, 2017.

DISCUSSION

Dr David L. Cull (Greenville, SC). Our understanding of the natural history and optimal management of blunt traumatic aortic injury has changed radically in the past decade. Treatment is now delayed until the patient's medical condition is stabilized, and open surgery to repair the aorta has been replaced by thoracic endovascular aortic repair (TEVAR). Several studies have recently reported favorable outcomes with medical management alone for grade I and II injuries. In Greenville, we have selectively treated grade III injuries with medical management. My new partner, Sagar Gandhi, just reported our experience in the December issue of the *Journal of Vascular Surgery*. Dr Sandhu and his colleagues report a series of 26 patients with grade I aortic injuries and 22 patients with grade II aortic injuries who were treated with medical therapy consisting of negative inotropic medications and vasodilators to control blood pressure. Their results provide additional evidence that the medical management of grade I and II blunt aortic injuries, in the intermediate term, is associated with low risk of aorta-related mortality. Of the 71% of patients who had a follow-up computed tomography (CT) scan, 84% of the radiographic injuries resolved, most within 8 weeks. Because the current literature and the Society for Vascular Surgery guidelines already support medical management of grade I injuries and this study does not follow these patients long enough to add substantially to what we already know, I would like to focus the discussion on the 22 patients with grade II injury because that seems to be the next horizon in the management of these injuries.

I have three questions for the authors.

Your manuscript reports that the management algorithm for blunt traumatic aortic injury by your group has traditionally been open surgery or TEVAR for grade II injuries. Given the favorable results of this study, have you altered your management algorithm? Are you now advocating medical therapy for all

patients with grade II injuries or only in selective cases? Specifically, are there radiographic findings, such as extent of intramural or mediastinal hematoma, or clinical factors beyond injury grade that favor medical therapy or TEVAR?

A follow-up CT scan was obtained in only 71% of patients in this study. The average time to follow-up CT was 40 days. Given the limited, short follow-up of this and previous retrospective studies that have examined this issue, do we really know the outcome of medical management for grade II injury? Until we have those data, should operative intervention still be considered the treatment of choice for these patients?

During the 11 years of this study, there has been a significant shift from open surgery to TEVAR and nonoperative medical management of blunt traumatic aortic injury at your institution. With this shift, have you been able to detect any change in the outcome of these patients?

I would like to thank the Society for allowing me to discuss this paper.

Dr Harleen K. Sandhu. Currently, we have not specifically altered our treatment algorithm. This is our first study to evaluate the role of medical therapy in managing patients with grade II aortic injury. Further evaluation and long-term follow-up are needed before an alteration in treatment algorithm can be recommended. To address this issue, we are leading an international multicenter prospective registry with the Aortic Trauma Foundation to further investigate the optimal management of aortic injuries.

Operative intervention should remain the treatment of choice for patients with grade II aortic injury until further investigation with the multicenter registry provides long-term follow-up.

As you are aware, our group has previously reported the outcomes of TEVAR compared with open repair. Our results demonstrated decreased adverse outcomes with TEVAR compared with open repair (odds ratio, 0.33).



CrossMark