Blunt Thoracic Aortic Injuries: Crossing the Rubicon

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I want to thank the leadership of the American College of Surgeons for the great honor and privilege of delivering the 2011 Scudder Oration. I feel both honored and humbled, having in mind the stature and major contributions of all the previous Scudder Orators.

I should like to dedicate this lecture to a lot of people, because I owe a lot to many! First, to my parents, who are now in their late 80s, still in great health, on the island of Cyprus. To my wife Elizabeth, and our children, Alexis, Stefanie, and Nicholas. To my past faculty for their major contributions—most of them currently hold leadership positions in prestigious centers across the country, and I am proud of them. To my current faculty—they are my second family. We dream, plan, and build together, and I am very proud of them. I want to single out Dr Tom DeMeester, who recruited me from Johannesburg, South Africa, in 1992, and trusted me with the leadership of the Los Angeles County and University of Southern California trauma program. For the next 17 years, he gave me his strong and unconditional support. I also want to thank the current Chairman of Surgery, Dr Vaughn Starnes, for his strong support of the trauma and Acute Care Surgery program during the last 3 years.

The title of my lecture is "Blunt Thoracic Aortic Injuries: Crossing the Rubicon." The Rubicon is a small river in Northern Italy. In 49 BC, Julius Caesar led his army across the Rubicon in violation of the law, making war inevitable. Today, crossing the Rubicon means passing the point of no return beyond which someone must continue on the current course of action because turning back is physically impossible or dangerous or prohibitively expensive. I selected the topic of blunt thoracic aortic injuries (BTAI) for my oration because, in my opinion, during the last decade no other organ injury has seen more changes in diagnosis, treatment, and outcomes than thoracic aortic trauma.

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History

The first case of BTAI was reported in 1557 by the anatomist Andreas Vessalius, in a victim who fell from a horse. Four hundred years later, in the 1950s, there was the first acute repair of an aortic rupture by De Bakey's group. In the 1970s came development of functional shunting techniques and graft materials.[1] In the 1990s, we witnessed the first reports supporting routine use of CT as a screening method in patients with a suspicious mechanism of injury,[2] and soon afterward CT angiography was advocated as a definitive diagnostic tool instead of angiography. In 1997, the first endovascular repair of a patient with BTAI was reported,[3] and in the 2000s, endovascular aortic repair became the new preferred therapeutic approach in most trauma centers.

Epidemiology

The majority of BTAI result from motor vehicle crashes (70%), followed by motorcycle crashes (13%), falls from heights (7%), auto vs pedestrian (7%), and other mechanisms.[4] Overall incidence of thoracic aortic injuries in patients reaching hospital care is <0.5%. In a series of 5,838 pedestrian injuries, the incidence of BTAI was 0.3%.[5] In another study of 1,163 admissions after high-level falls, the incidence of BTAI was 0.1.[6] The presence of a pelvic fracture is a marker of increased risk for thoracic aortic injury. In an analysis of 1,450 pelvic fractures, the incidence of BTAI was 1.4%.[7] However, it seems that these patients who reach hospital care are only the tip of the iceberg, and the real incidence of BTAI is much higher. The vast majority of patients die at the scene and never reach hospital care. The incidence of aortic injuries in fatal traffic injuries is very high. In a recent analysis of 304 deaths due to traffic injuries or high-level falls in the county of Los Angeles, 102 patients (33%) had a rupture of the thoracic aorta. About 80% of deaths occurred at the scene and only 20% in the hospital.[8] In another autopsy analysis of 25 fatalities in a recent train crash in Los Angeles, thoracic aortic rupture was found in 8 patients (33%). All mortalities occurred at the scene.[9] It is obvious that the only way to prevent these deaths is better automobile design, and automobile manufacturers need to address this major issue. The CDC reported 41,911 traffic-related deaths in 2008.[10] If we extrapolate these autopsy findings, it is estimated that every year, about 14,000 traffic fatalities have BTAI.

The incidence of BTAI increases with age and is rare in the pediatric population. In a National Trauma Databank

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Abbreviations and Acronyms

AAST = American Association for the Surgery of Trauma
BTAI = blunt thoracic aortic injury
OR = odds ratio
TEE = transesophageal echocardiography

analysis, the incidence of thoracic aortic injury in children younger than 16 years old was 7 times lower than in adults (0.03% vs 0.21%).[11] In an analysis of 5,838 automobile vs pedestrian injuries, there were no aortic injuries in the age group 14 years and younger. The incidence increased to 0.2% in the group 15 to 65 years, 0.5% in the group 56 to 65 years, and 1.5% in the group older than 65 years.[5]

About 40% of patients with BTAI who reach the hospital alive have a severe associated injury (body area Abbreviated Injury Scale score >3), and mean Injury Severity Score is 40. The most common severe associated trauma involves the head.[4]

Site and type of aortic injury

The most common anatomical site of aortic injury is the medial aspect of the lumen, distal to the left subclavian artery (Fig. 1). Injury at this site is found in about 93% of hospital admissions and in about 80% of autopsy studies. Siegel and colleagues, in computer simulation and cadaver studies, showed that at the time of the crash the intra-aortic pressure increases to a mean of 1,449 mmHg. This high pressure combined with rotational forces, exerts a highly focused stress at the isthmus (Fig. 2). In addition, the tensile strength at the isthmus was found to be only 63% of that of the proximal aorta.[12,13] The most common type of injury is a false aneurysm (58%), followed by dissection (25%) and intimal tear (20%).[4]

Natural history of BTAI

The majority of patients with BTAI die at the scene, before reaching hospital care. In an analysis of 242 fatal BTAI, Burkhart and colleagues reported that 57% of the deaths occurred at the scene or on arrival to the hospital, 37% died within the first 4 hours of admission, and 6% died >4 hours after admission.[14] In another autopsy study of 102 victims with BTAI, about 80% of the deaths occurred at the scene and only 20% in hospital.[8] For those patients reaching hospital care, timely diagnosis and prompt aggres-

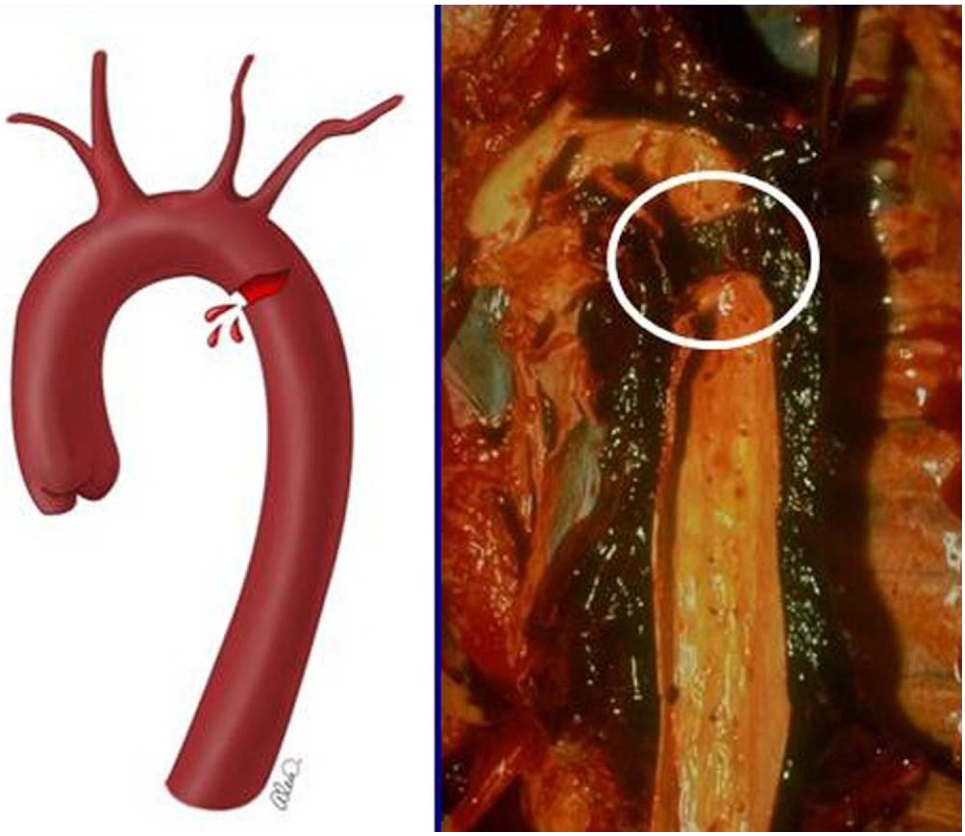

Figure 1. The aortic isthmus is involved in >90% of blunt thoracic aortic injuries reaching hospital care and in about 80% of fatal aortic injuries. (Illustration reprinted with permission of Alexis Demetriades.)

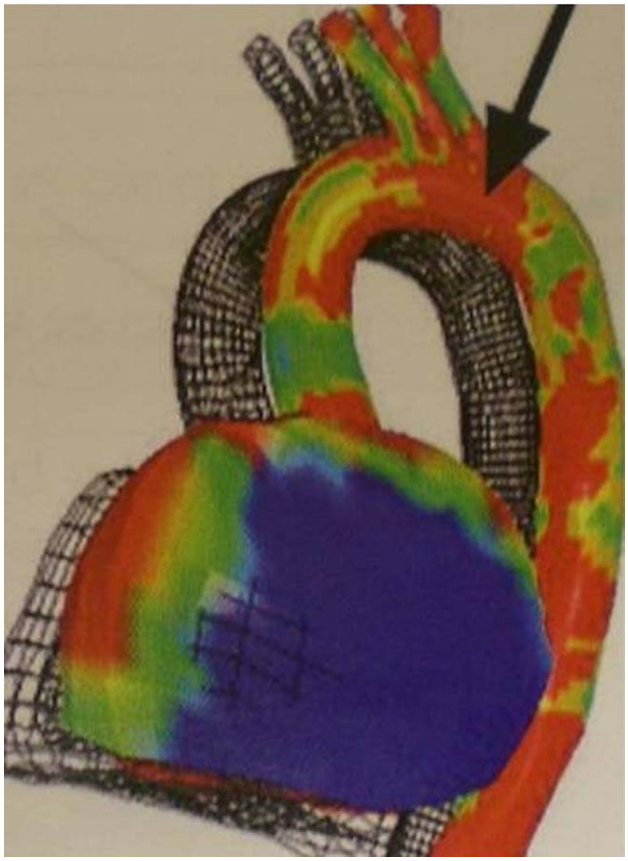

Figure 2. Computer simulation and cadaver studies showed that at the time of the crash, intra-aortic pressure increases to a mean of 1,449 mmHg. This pressure combined with rotational forces exerts the biggest stress at the isthmus. Adapted from: Siegel JH, Smith JA, Siddiqi SQ. Change in velocity and energy dissipation on impact in motor vehicle crashes as a function of the direction of crash: key factors in the production of thoracic aortic injuries, their pattern of associated injuries and patient survival. A crash injury research engineering network (CIREN) study. J Trauma 2004;57:760–777; discussion 777–778, with permission; and Siegel JH, Belwadi A, Smith JA, et al. Analysis of the mechanism of lateral impact aortic isthmus disruption in real-life motor vehicle crashes using a computer-based finite element numeric model: with simulation of prevention strategies. J Trauma 2010;68:1375–1395, with permission.

sive blood pressure control are essential in preventing free rupture of the contained aortic injury.

Screening and diagnosis

The supine chest x-ray has been the standard screening tool for BTAI for many decades. Radiologic findings suspicious for aortic trauma include widened upper mediastinum (>8 cm on a anterior-posterior supine chest film at the level of the aortic knob), obliteration of the aortic contour, loss of the perivertebral pleural stripe, depression of the left mainstem bronchus, deviation of a nasogastric tube to the right,

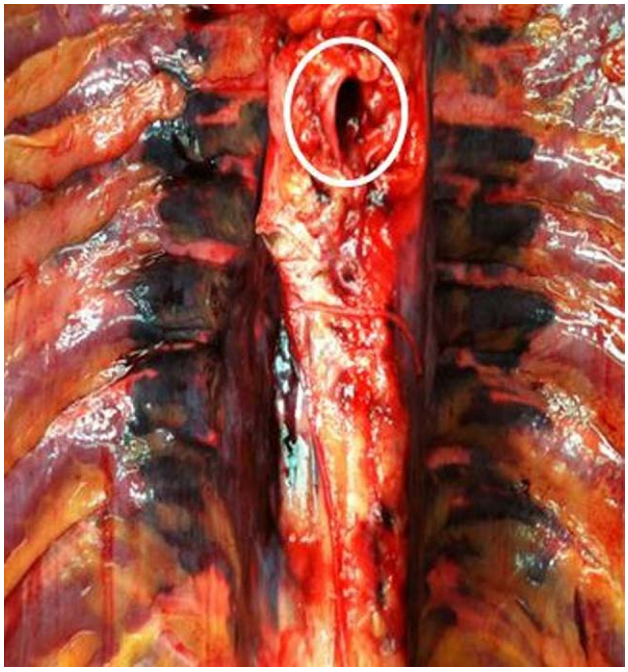

Figure 3. Autopsy specimen shows an aortic transection (circle) and surrounding mediastinal hematoma. This hematoma will show on the chest x-ray as a widened mediastinum.

a left apical pleural hematoma (apical cap), and a massive left hemothorax. The presence of fractures of the sternum, scapula, upper ribs, or clavicle in a multi-trauma patient is a marker of increased risk for BTAI. The widened mediastinum is the most common finding, but it has low sensitivity and specificity. Other traumatic conditions, such as a fracture of the sternum or the thoracic spine can cause a widened mediastinum. The abnormal radiological findings on chest x-ray are secondary to bleeding and hematoma formation around the aortic tear (Fig. 3). However, in many cases, there is no substantial periaortic hematoma and the mediastinum appears normal on chest x-ray (Fig. 4). In a study by Plurad and colleagues,[15] 14% of 43 patients with BTAI had a normal mediastinum on chest x-rays. Other studies have also shown that chest radiography is a poor screening tool for BTAI.[2,16-18] In a prospective study in 1998, we concluded that high-risk deceleration injuries should undergo routine CT scan evaluation of the mediastinum irrespective of chest radiographic findings.[2] Subsequent studies confirmed our findings and reached the same conclusion.[17,18] The sensitivity and negative predictive value of the CT scan in the diagnosis of BTAI approaches 100%.[19]

Aortography, which had been the gold standard for the definitive diagnosis of BTAI until the late 1990s, has now been replaced by CT angiography. The new-generation multi-slice CT scanners with 3-dimensional reformation

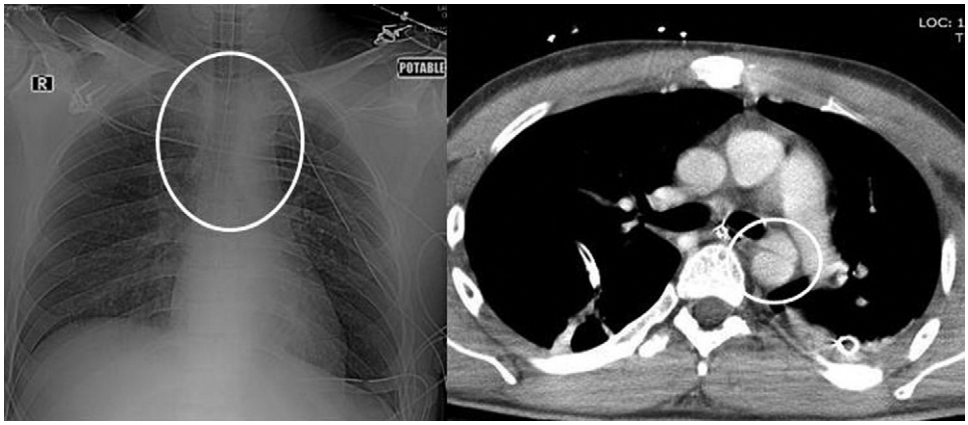

Figure 4. High-speed traffic injury. The mediastinum on the chest x-ray seems unremarkable. Routine CT scan shows an aortic injury (circle). Note the absence of any major periatortic hematoma, which explains the normal mediastinum on plain radiography.

have almost 100% sensitivity and specificity, a 90% positive and 100% negative predictive value, an overall diagnostic accuracy of 99.7%,[18] and provide impressive anatomical details of the aortic arch and the injury site (Fig. 5).

The initial enthusiasm for transesophageal echocardiography (TEE) as a diagnostic tool has been replaced by skepticism and failed to gain popularity because of conflicting reports about its accuracy and concerns about its availability 24 hours a day.[20-22] The dramatic shift from angiography and TEE to CT scanning in the diagnosis of BTAI is demonstrated by 2 studies sponsored by the American Association for the Surgery of Trauma in 1997 and 2007.[4,23,24] Use of angiography and TEE for the diagnosis of thoracic aortic injuries decreased from 87% and 12%, respectively, in 1997, to only 8% and 1% in 2007[6] (Table 1).

In summary, CT angiography is now the new standard modality for screening and definitive diagnosis of BTAI. Formal aortography might have a diagnostic role in patients undergoing angiography for other injuries, such as

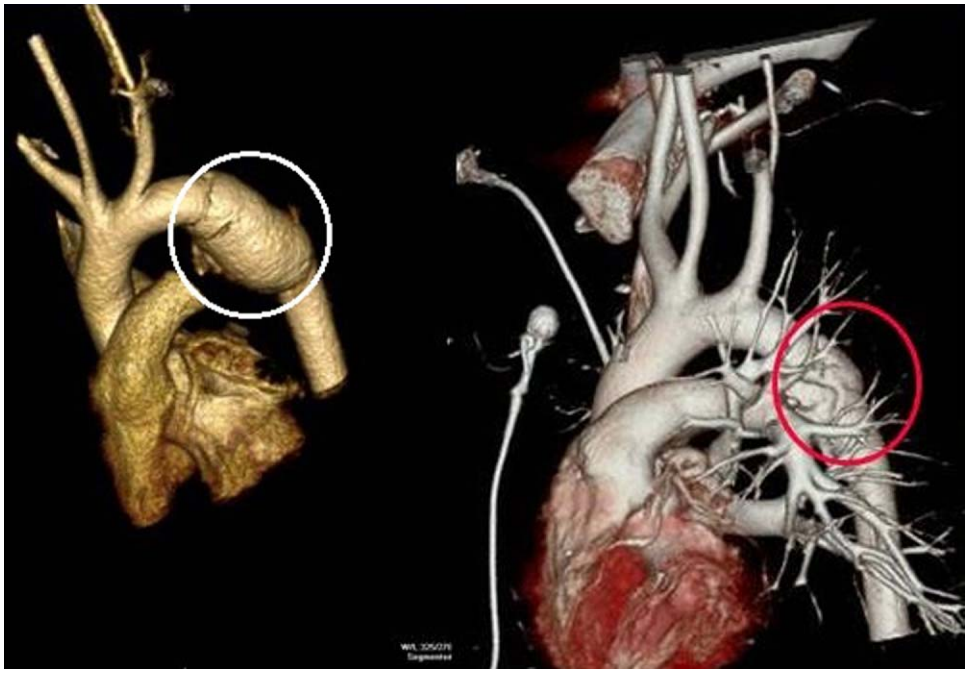

Figure 5. New CT scan technology provides excellent details of the anatomy of the aortic arch and type and site of the aortic injury (circles).

Table 1. Changing Perspectives: Diagnostic Modalities for Blunt Thoracic Aortic Injury: American Association for the Surgery of Trauma 1 (1997) vs American Association for the Surgery of Trauma 2 (2007)

Diagnostic modality	AAST 1 (n = 253)		AAST 2 (n = 193)		p Value
	n	%	n	%	
Aortogram	207	87	16	8.3	<0.001
CT scan	88	34.8	180	93.3	<0.001
TEE	30	11.9	2	1.0	<0.001

AAST, American Association for the Surgery of Trauma; TEE, transesophageal echocardiogram.

pelvic fractures, complex liver injuries, etc. TEE might be useful in critically ill patients in the intensive care unit who cannot be transferred safely to the radiology suite for CT scan.

Initial management of thoracic aortic injuries

Patients with active extravasation (Fig. 6) from the aortic tear need an immediate life-saving operation without delay. However, the vast majority of patients reaching hospital alive have contained aortic ruptures. Prevention of free rupture in this group of patients by means of rigorous blood pressure control is the most urgent priority. The risk of free rupture is highest in the first few hours after the injury, with >90% of ruptures occurring within the first 24 hours. Without rigorous blood pressure control, risk of rupture is about 12%, and rigorous blood pressure control reduces the risk to about 1.5%.[24,25] Systolic blood pressure should be kept as low as tolerated, in most patients at about 90 to 110 mmHg. In elderly or head-injury patients, optimal systolic pressure might be slightly higher. Cautious restriction of intravenous fluids and administration of β-blockers are the most commonly used modalities for blood pressure control.

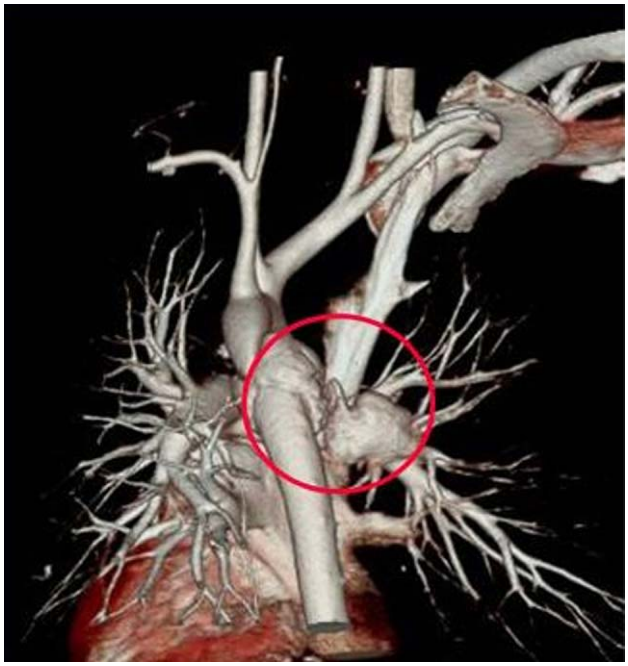

Figure 6. CT scan with 3-dimensional reformation showing a major aortic tear with considerable active extravasation (circle). This type of injury requires immediate operation.

Timing of definitive management

Early studies showed that, if untreated, risk for rupture of a

Table 2. Epidemiological and Clinical Characteristics in Early and Delayed Aortic Repair Groups

Characteristic	All patients (n = 178)	Early repair (n = 109)	Delayed repair (n = 69)	p Value
Male, n (%)	137/178 (77.0)	81/109 (74.3)	56/69 (81.2)	0.290
Age, y, mean ± SD	39.4 ± 18.2	39.1 ± 17.7	39.9 ± 19.1	0.776
Age older than 55 y, n (%)	32/177 (18.1)	18/109 (16.5)	14/68 (20.6)	0.493
Age older than 70 y, n (%)	15/177 (8.5)	7/109 (6.4)	8/68 (11.8)	0.214
Hypotension on admission, n (%)	29/175 (16.6)	19/109 (17.4)	10/66 (15.2)	0.694
GCS ≤8, n (%)	43/175 (24.6)	25/108 (23.1)	18/67 (26.9)	0.579
ISS, mean ± SD	39.3 ± 11.4	38.2 ± 10.6	40.9 ± 12.6	0.123
Head AIS >3, n (%)	31/176 (17.6)	15/107 (14.0)	23.2 (16/69)	0.119
Abdomen AIS >3, n (%)	35/177 (19.8)	19/108 (17.6)	16/69 (23.2)	0.362
Extremity AIS >3, n (%)	17/176 (9.7)	11/107 (10.3)	6/69 (8.7)	0.728
Any severe associated injury, n (%)	68/176 (38.6)	37/107 (34.6)	31/69 (44.9)	0.169
Open repair, n (%)	63/178 (35.4)	38/109 (34.9)	25/69 (36.2)	0.852
Endovascular repair, n (%)	115/178 (64.6)	71/109 (65.1)	44/69 (63.8)	0.852
Hours from injury to procedure, mean ± SD	54.6 ± 101.6	10.2 ± 5.9	126.2 ± 137.0	<0.001

The p values for categorical variables were derived from 2-tailed chi-square test; p values for continuous variables were derived from Student's *t*-test or Mann Whitney test.
AIS, Abbreviated Injury Scale; GCS, Glasgow Coma Scale; ISS, Injury Severity Scale.

Table 3. Type of Aortic Injuries in Early and Delayed Repair Groups

	All patients (n = 178)		Early repair (n = 109)		Delayed repair (n = 69)		
Injury	n	%	n	%	n	%	p Value
Intimal tear	36/170	21.2	26/104	25.0	10/66	15.2	0.126
Pseudoaneurysm	100/170	58.8	55/104	52.9	45/66	68.2	0.048
Dissection	42/170	24.7	27/104	26.0	15/66	22.7	0.634

contained aortic injury is highest in the first 24 hours after injury.[24,26] For this reason, definitive repair of the aortic injury has been considered as an emergency, and this practice remained the standard of care for many decades. However, subsequent studies showed that early and rigorous blood pressure control decreases wall stress in the region of the injury and reduces the risk of free rupture.[25,27] It is highly unlikely that a patient with contained BTAI undergoing rigorous blood pressure control and surviving the first 4 hours will progress to free rupture.[25] On the basis of these reports and experience gained from management of nontraumatic thoracic and abdominal aortic dissections, the concept of delayed repair of contained BTAI was first entertained in the late 1990s and early 2000s.[25,27-29] This approach was initially used in selected patients with major associated injuries, in an effort to stabilize these injuries before aortic repair. The concept of delayed repair was subsequently expanded to low-risk patients with no severe associated injuries or major comorbidities.

Early reports with delayed repair of BTAI were confusing and contradictory because of their retrospective nature, small patient populations and selection of predominately high-risk patients for delayed repair. Some studies showed improved outcomes with delayed repair, and others failed to show any benefits. Maggisano and colleagues[30] in 1995 and Pate and colleagues[28] in 1999, reported improved outcomes with delayed repair. However, Wahl and colleagues[31] in 1999 and Hemmila and colleagues[25] in 2004, reported higher complication rates, longer hospital stay, and higher direct costs with delayed repair. The Eastern Association for the Surgery of Trauma Practice Management Guidelines in 2000 recommended prompt repair of BTAI, with delayed repair reserved for patients with major associated injuries or high-risk patients.[32]

Table 4. Adjusted Odds Ratio for Mortality: Early vs Delayed Repair[33]

Patients	Adjusted OR (95% CI)	Adjusted p Value
All patients	7.78 (1.69−35.70)	0.008
Patients without major associated injuries	9.08 (0.88−93.78)	0.064
Patients with major associated injuries	9.39 (0.93−95.18)	0.058

OR, odds ratio.

To address this unresolved issue, we performed a large, prospective study under the auspices of the American Association for the Surgery of Trauma (AAST). This study analyzed outcomes in a series of 178 patients with BTAI, according to timing of definitive repair (early, <24 hours; delayed, >24 hours).[33] The 2 groups were similar with regard to injury severity, major associated injuries, type of aortic injury, and type of aortic repair (operative vs endovascular) (Tables 2 and 3). Mean time from injury to repair was 10.2 hours in the early group and 126.2 hours in the delayed group. The overall crude mortality in the delayed repair group was significantly lower than the early repair group (5.8% vs 16.5%; p = 0.034). Multivariate analysis adjusting for injury severity; severe extrathoracic injuries; Glasgow Coma Score and hypotension on admission; age; and method of aortic injury repair, showed a significantly increased risk of death in the early repair group (adjusted odds ratio [OR] = 7.78; 95% CI, 1.69−35.70; adjusted p value = 0.008). Subanalysis of the group of patients with major associated injuries confirmed the survival benefit with delayed repair. Crude mortality was 21.6% in early repair vs 3.2% in delayed repair (OR = 8.28; 95% CI, 0.97−70.38; p = 0.033). In a multivariate analysis adjusting for Glasgow Coma Score ≤8, blood pressure <90 mmHg, age (55 years or younger vs older than 55 years), and open vs endovascular repair, the adjusted risk of death in the early repair group was 9.39 (95% CI, 0.93−95.18; p = 0.58). Adjusted risk of death with early repair in the subgroup of patients without major associated injuries was 9.08 (95% CI, 0.88−93.78; p = 0.064) (Table 4). Incidence of paraplegia was similar in the 2 groups (early repair 1.8%, delayed repair 1.4%).[4]

Table 5. Time from Admission to Definitive Repair: American Association for the Surgery of Trauma 1 Study (1997) vs American Association for the Surgery of Trauma 2 Study (2007)[23]

	Time to operation, h
AAST 1 (1997)	
Open repair (n = 207)	16.5
AAST 2 (2007)	
Open repair (n = 68)	67.6
Endovascular repair (n = 125)	48.1

AAST, American Association for the Surgery of Trauma.

Survival benefits of delayed repair have been confirmed in a subsequent large retrospective study by Estrera and colleagues.[34] In an analysis of 145 repairs of BTAI from the Utah Trauma Registry, the authors identified delayed repair as the only independent factor protective against mortality. The reasons for better survival with delayed repair are not clear. It is possible that delayed repair allows better resuscitation, management of other serious life-threatening injuries, and definitive repair of the aortic injury in an optimal environment. Optimal time from injury or admission to repair is unknown and should be individualized, taking into account many factors, such as the presence of other severe injuries or comorbid conditions, the physiological status of the patient, and the type and severity of the aortic injury. Delayed repair should not be attempted in cases with active leaking from the aortic injury (Fig. 6). Also, it might be advisable that, in patients with large contained injuries, the repair should be done urgently, ie, within a few hours of diagnosis.

Delayed repair has become the new standard of care. This major change is obvious when comparing the time from injury to repair between the 2 AAST studies in 1997 and 2007. In the 1997 AAST 1 study, mean time from injury to repair of the aorta was 16.5 hours, and in the 2007 AAST 2 study, this time period increased to 54.6 hours[23] (Table 5).

Endovascular repair

Operative repair of all BTAI remained the only acceptable standard of care for many decades. However, in the last decade, there have been 2 major changes in the definitive care of these injuries, ie, endovascular repair and conservative management with observation.

Endovascular repair has numerous theoretical advantages. It can be performed under sedation and local anesthesia, it minimizes blood loss, is tolerated well by high-risk patients, minimizes risk of paraplegia, and reduces mortality.

The first case report of endovascular stent-graft treatment of a BTAI was published in 1997.[3] In the next decade, the design of these devices improved considerably (Figs. 7 and 8), but the experience remained limited, with sporadic case reports and small case series. Collective review of the literature in 2007 showed only 284 cases of BTAI from 62 centers treated with endovascular techniques. Only 15 of these cases were analyzed prospectively.[35] In addition, the results were viewed with a degree of skepticism because of the risk of publication bias; centers with good outcomes are more likely to publish their results than centers with poor outcomes. In addition, early publications supported the use of endovascular techniques in high-risk patients, with open repair remaining as the procedure of choice in low-risk patients.[36] As experience with endovascular procedures increased, the trauma and vascular surgeons and interventional radiologists became more liberal with the indications of this technique.

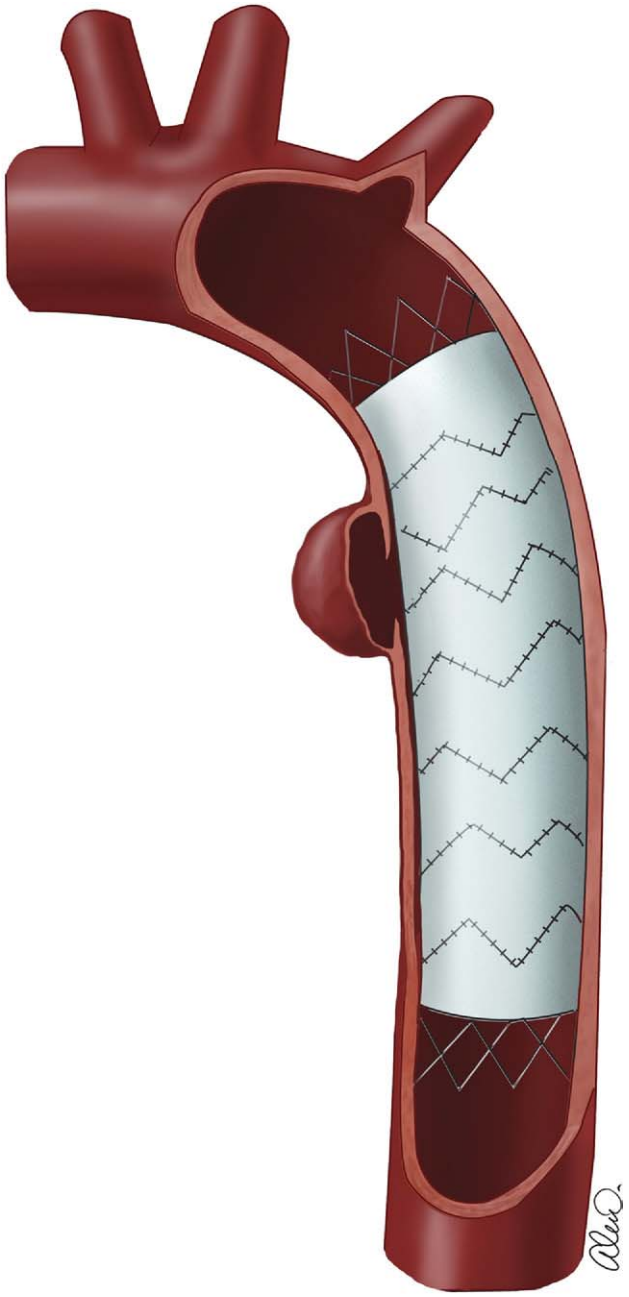

Figure 7. Schematic modern stent-graft deployed and covering the aortic injury. (Reprinted with permission of Alexis Demetriades.)

To address some of these concerns, we performed a large, multicenter prospective AAST study.[4] The study included 193 patients with BTAI from 18 participating trauma centers. The first surprise was the extensive use of endovascular treatment, even in low-risk patients. Overall, 65% of

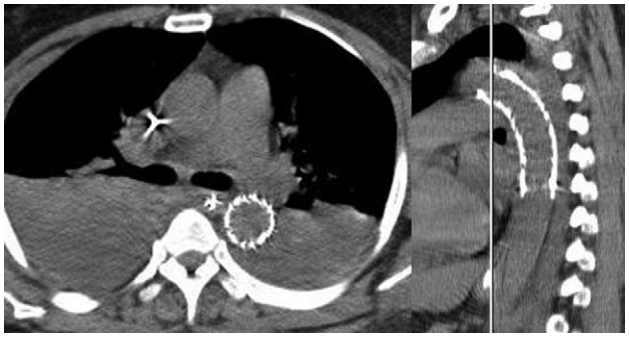

Figure 8. CT scan appearance of a successfully deployed thoracic aortic stent.

BTAI, 60% of patients with no major associated injuries, and 57% of patients younger than 55 years old who had no major associated injuries were treated with endovascular stent-grafts.

In comparing the epidemiological and injury severity characteristics of the 2 treatment groups, the endovascular repair group was significantly older than the open repair group (age older than 70 years, 13.7% vs 4.4%; p = 0.04). Types of aortic injury were similar in the 2 groups (Table 6). Overall crude mortality was 13.0%, 23.5% in the open repair, and 7.2% in the endovascular repair group (p = 0.001). Multivariate analysis adjusting for age, hypotension on admission, Glasgow Coma Score ≤8, and severe extrathoracic trauma, showed a significantly higher mortality in the open repair group (adjusted OR = 8.42; 95% CI, 2.76−25.69; p < 0.001). A second multivariate analysis in the subgroup of 115 patients with no major associated injuries again showed a significantly higher mortality in the open repair group (adjusted OR = 13.08; 95% CI, 2.53−67.53; p = 0.002). A third multivariate analysis in the subgroup of 74 cases with severe associated injuries again showed a higher mortality in the open repair group (adjusted OR = 5.68; 95% CI, 1.09−29.45; p = 0.04) (Table 7).

Table 6. Open vs Endovascular Repair: Epidemiological and Injury Severity Characteristics (n = 193)

Characteristic	Open repair (n = 68), %	Endovascular repair (n = 125), %	p Value
Male	80.9	72.8	0.211
Age older than 70 years	4.4	13.7	0.044
GCS ≤8	25.4	26.0	0.923
Hypotension on admission	20.9	13.8	0.207
Mean ISS	38.9	39.4	0.826
Extrathoracic injury with AIS >3	31.3	43.4	0.103
Type of aortic injury			
Intimal tear	19.7	21.0	0.832
Aneurysm	57.6	58.8	0.869
Dissection	28.8	23.5	0.431

AIS, Abbreviated Injury Scale; GCS, Glasgow Coma Scale.

Table 7. Open vs Endovascular Repair: Mortality

Patients	Adjusted OR (95% CI) for death	Adjusted p Value
All patients (n = 193)	8.42 (2.76−25.69)	<0.001
Patients with no major associated injuries (n = 115)	13.08 (2.53−67.53)	0.002
Patients with no major associated injuries (n = 74)	5.68 (1.09−29.45)	0.04

OR, odds ratio.

Although early survival results with the endovascular repair were impressive, the incidence of device-related complications was alarmingly high. Overall, 32 device-related complications developed in 25 (20%) of the 125 endovascularly treated patients. The most common complication was an endovascular leak, which occurred in 18 (14.4%) patients (Fig. 9). Nine of these patients required a second or third device, 6 required open repair, and 3 were observed. Other serious device-related complications included 4 access-vessel injuries requiring emergency operation, 4 left subclavian artery occlusions, 1 left common

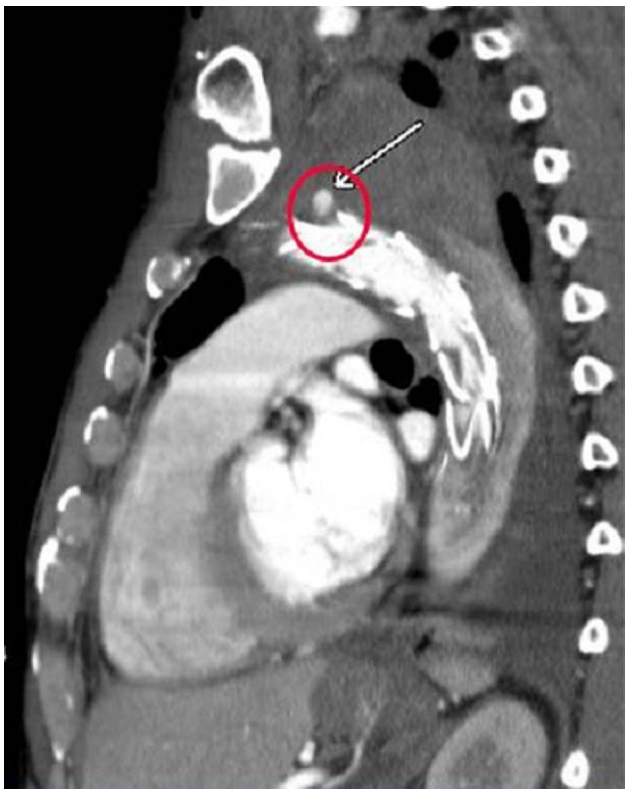

Figure 9. CT scan shows an endoleak at the top of the stent.

Table 8. Complications after Endovascular Repair of Blunt Thoracic Aortic Injuries: Low-Volume (<15 Cases) vs High-Volume (≥15 Cases)

Complications	Adjusted OR (95% CI)	Adjusted p Value*
Systemic complications	3.88 (1.69–8.91)	0.001
Device-related complications	2.70 (1.08–6.71)	0.003

*Multivariate analysis adjusting for major extrathoracic injuries, Glasgow Coma Scale on admission, hypotension on admission, and age.
OR, odds ratio.

carotid artery occlusion, 2 strokes, 1 paraplegia, and 1 stent-graft collapse.[4] There was some strong evidence that high-volume centers (≥15 endovascular procedures) had considerably lower systemic and device-related complications than low-volume centers (Table 8). This finding suggests that these procedures should be performed in selected centers of excellence.

When comparing practices and outcomes between the 2 AAST studies on BTAI, which were performed 10 years apart (1997 and 2007),[4,24] we see some dramatic changes. The use of endovascular stent-grafts saw a meteoric rise from 0% in 1997 to 65% in 2007. In the open repair group, use of distal aortic perfusion increased from 65% in 1997 to 84% in 2007. Overall mortality decreased from 22.0% to 13.0%, and procedure-related paraplegia from 8.7% to 1.6%[23] (Table 9).

In summary, the AAST 2 study[4] demonstrated impressive early improved survival and reduced paraplegia results with endovascular repair. However, the high incidence of device-related serious complications is a cause of major concern, and there is an urgent need for device improvement. The most common complication was endoleak, which occurred in 14.4% of cases. There are 2 significant contributing factors for the development of this complication: the first is poor sizing of the stent; optimal deployment requires oversizing the device by 10% to 20%.[36,37] Currently, this is not always possible, especially in young patients who have a small aorta, because commercially available devices come in a limited range of fairly large sizes. Deployment of an excessively oversized stent-graft increases the risk of endoleak, infolding, and even collapse (Fig. 10). Technological advances have now allowed the design of small-diameter stents (diameter 16 to 18 mm), which are currently being tested in clinical trials. A second factor that can increase risk of endoleak is the shape of the stent-graft. The currently available straight stent-grafts do not always allow close opposition of the device with the aortic wall, especially in the inner corner. New designs and testing of curved prostheses are currently in progress and most probably will decrease the incidence of endoleaks.

Table 9. Therapeutic Modalities and Outcomes in Blunt Thoracic Aortic Injuries: American Association for the Surgery of Trauma 1 Study (1997) vs American Association for the Surgery of Trauma 2 Study (2007)

Therapy and outcomes	AAST 1 (1997) (n = 207)*		AAST 2 (2007) (n = 193)		
	n	%	n	%	p Value
Open repair	207	100	68	35.2	<0.001
Clamp/sew	73/207	35.3	11/68	16.2	0.003
Bypass	134/207	64.7	57/68	83.8	0.003
Endovascular repair	0/207		125/193	64.8	<0.001
Overall mortality	53/241†	22.0	25/193	13.0	0.02
Paraplegia	18/207	8.7	2/193	1.6	0.001

*Excluding patients in extremis or those managed nonoperatively.
†Excluding patients in extremis or free rupture.
AAST, American Association for the Surgery of Trauma.

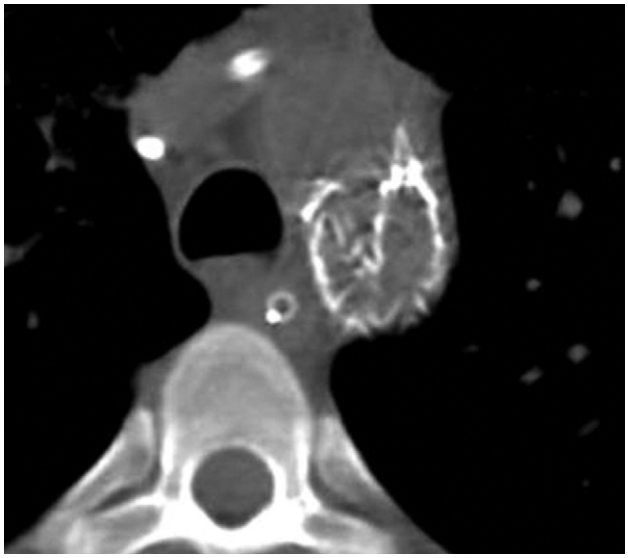

Figure 10. Partial collapse of the aortic stent-graft is usually the result of excessive oversizing (Courtesy of Riyad Karmy-Jones, MD).

Access-vessel injuries to the femoral or external iliac artery requiring emergency operation occur in a considerable number of patients, 3% in the AAST 2 study,[4] and in some series up to 16%.[38] This complication is the result of the large diameter of the currently available deployment sheaths. A new generation of introducer sheaths are currently being tested and, if successful, most likely will reduce the risk of this complication.

Occlusion of the left subclavian artery and sometimes the left common carotid artery is another serious device-related complication (Fig. 10). Although some subclavian artery occlusions are tolerated well, many others need delayed revascularizations with carotid–subclavian bypass because of steal syndrome[38] (Fig. 11). Other observed complications include late thrombosis or migration of the stent-graft[12] (Fig. 12).

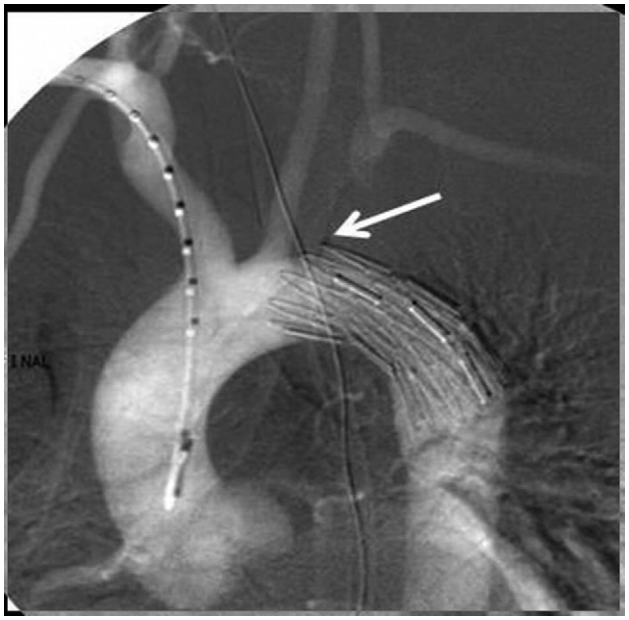

Figure 11. Occlusion of the left subclavian artery (arrow) by deployed stent-graft, resulting in vertebral artery steal syndrome. This patient might need carotid-subclavian artery bypass.

Development of curved fenestrated/branched grafts is promising and might make possible the endovascular management of injuries of the aortic arch and preservation of flow in the major arch branches (Fig. 13).

Finally, it is possible that engineering advances will allow the construction of custom-made stent-grafts for each patient, taking into account the anatomy and shape of the aortic arch and the size and site of the aortic injury.

Although many of the early complications can be addressed with device improvements, long-term results with endovascular treatment remain a cause for concern. The long-term mechanical properties of stent-grafts are unknown. There are case reports of fracture or collapse of the stent many months or years after insertion.[38] Another major concern is the behavior of the stent with the changing anatomy of the thoracic aorta that occurs with age. This is particularly important in young patients with long life expectancy. With advancing age, the aorta becomes tortuous and dilated and the behavior of the device with these changes is unknown.

The available medium and long-term results after endovascular treatment are limited and results are conflicting. In a recent study, Patel and colleagues,[39] followed up 19 patients with BTAI treated with endovascular stent-graft for a mean of 36 months. One patient (5.3%) required reintervention for a collapsed stent. However, another study by Fernandez and colleagues[38] reported more concerning short and medium-term results. In a retrospective series of 20 patients with BTAI treated with endovascular stents and a follow-up range between 5.5 and 108 months, the authors reported major problems; 4 insertion-site injuries requiring operation, 2 subclavian artery occlusions requiring revascularization 35 and 37 days after the procedure because of steal syndrome; 1 collapsed stent 6 months after the procedure requiring reintervention; 1 thrombosis of the stent requiring intervention at 1 year; 1 fractured stent at 4 years; and 7 intragraft mural thrombi were observed.

Another unanswered question is the frequency and duration of follow-up after endovascular treatment. Currently, and without any scientific evidence, it is recommended that radiologic follow-up be performed every 6 months initially and later on every 12 months. The method of follow-up, ie, plain chest radiography or CT with intravenous contrast, is still unresolved, although most surgeons

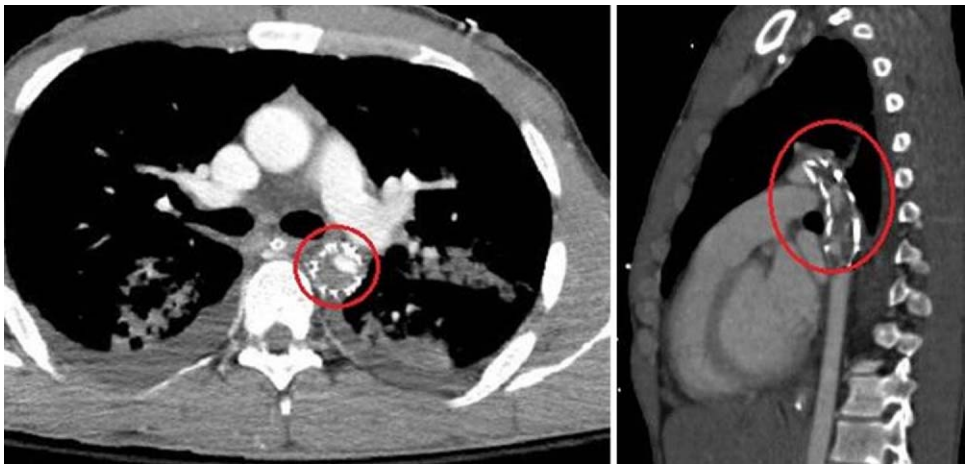

Figure 12. CT angiogram shows thrombosis of the aortic stent-graft 1 year after deployment of the device for BTAI. Paraplegia developed in the patient as a result of the thrombosis.

Figure 13. Development of curved fenestrated/branched stent grafts can allow endovascular management of injuries to the aortic arch (courtesy of Cherrie Abraham, MD).

recommend CT because thrombosis of the stent might not show in plain films.

Forbes and colleagues,[40] in a study of 17 patients with BTAI treated with endovascular repair and with a minimum of 1 year follow up, reported an increased rate of dilation of the proximal thoracic aorta, just distal to the subclavian artery. The clinical significance of this observation is unknown.

It is obvious that because of the fast pace of adoption of endovascular repair, there is a major and potentially dangerous deficiency of knowledge about medium and long-term results. I believe that there is an absolute and urgent need for a national registry monitoring the natural history and safety of these devices. Such a registry could be led by one of our national organizations, such as the American College of Surgeons, AAST, or the Society of Vascular Surgeons. If I had to give only one message in this oration, this is the one!

Role of the surgeon in endovascular repair in BTAI

The meteoric shift from open repair to endovascular repair of BTAI raises some important questions about the role and training of the vascular surgeon in the care of these patients. In selecting vascular surgery as a specialty, residents consider training in endovascular capabilities as an important factor and express concerns about the potential loss of patients to other specialties.[41] However, training in the endovascular management of BTAI poses considerable practical challenges because of the small patient population. The average trauma center admits about 4 cases of BTAI per year. Effective use of endovascular simulators can be a critical component of the training. It might be advisable that these cases be managed in selected high-volume centers of excellence, as demonstrated by the superior results shown in the AAST 2 study.[4] Most patients with BTAI have multiple injuries, and a multidisciplinary team that includes trauma surgeons, surgical intensivists, vascular and cardiac surgeons, neurosurgeons, orthopaedic surgeons, and interventional radiologists is essential for optimal outcomes.

Nonoperative management

A third therapeutic approach to BTAI, which evolved in the last decade, is nonoperative management. This approach has been used in selected patients with minor grade I or II aortic injuries (eg, intimal tears or intramural hematomas). Although the published experience is still limited, preliminary results are encouraging. It seems that it is safe to manage most of these cases with blood pressure control and observation, without any other intervention. Serial

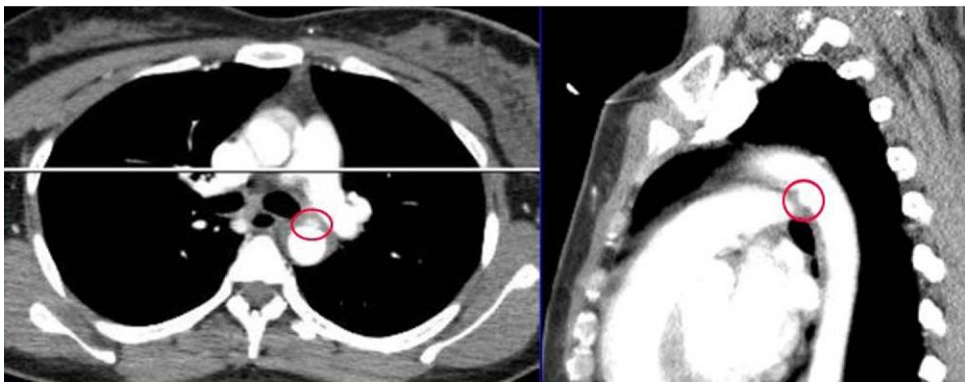

Figure 14. Minimal aortic injuries (circle) can be managed nonoperatively with CT-scan monitoring until healing.

CT-scan monitoring should be performed until complete resolution. Malhotra and colleagues,[42] in a small series of 6 patients with minimal aortic injuries who were observed, reported complete resolution in 2, no change in 1, and progression to small aneurysms in 3. Kepros and colleagues,[43] in another small series of 5 patients, reported complete resolution in all of them, within 3 to 19 days. Caffarelli and colleagues,[44] in a larger series of 27 patients treated nonoperatively and with a median follow-up of 107 days, reported no change of the lesion in 19, complete resolution in 5, progression requiring open repair in 1, and endovascular stenting in 2. In view of these findings, it is suggested that this option be considered in very selected patients and with close CT-scan monitoring of the progression of the lesion (Fig. 14).

CONCLUSIONS

During the last decade, we have experienced major changes in diagnosis, therapeutic modalities, and outcomes in BTAI. Routine CT-scan screening for aortic injuries in suspicious mechanisms of injury has now become a standard of care in most modern trauma centers; diagnostic CT angiography has replaced diagnostic angiography and is the new standard; delayed definitive repair of the aortic injury is now the preferred approach in most cases; endovascular repair has largely replaced open repair; and nonoperative observational management is emerging as an acceptable alternative therapeutic modality in selected cases. All of these changes have resulted in major improvements in survival and reduction of procedure-related paraplegia. There are still some unresolved issues and areas of concern. We still need improvement of the endovascular devices to reduce the high incidence of device-related complications. We still need medium and long-term results after endovascular repair. We still need a national registry to monitor the mechanical properties and safety of endovascular devices. We still need larger studies to evaluate the natural history and safety of nonoperative observational management. Despite these concerns, we have reached a point of no return and must continue on the current course. We have crossed the Rubicon!

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