

Aorta, July 2013, Volume 1, Issue 2: 117–122 DOI: http://dx.doi.org/10.12945/j.aorta.2013.12-009 Received: November 20, 2012 Accepted: May 13, 2013 Published online: July 2013

Acute Traumatic Thoracic Aortic Injury

Considerations and Reflections on the Endovascular Aneurysm Repair

Luca Di Marco, MD, PhD*, Davide Pacini, MD, Roberto Di Bartolomeo, MD

Cardiac Surgery Department, S. Orsola-Malpighi Hospital, University of Bologna, Bologna, Italy

Abstract

Traumatic rupture of the thoracic aorta is a lifethreatening lesion and it occurs in 10 to 30% of fatalities from blunt thoracic trauma and is the second most common cause of death after head injury. Immediate surgery is often characterized by a high mortality and morbidity rate. Delayed repair of traumatic aortic injuries has significant survival benefits and a much lower mortality rate compared with early open repair. Despite developments in operative techniques, there still remains considerable operative mortality and morbidity associated with a surgical approach even if delayed. Endovascular stent grafts for the thoracic aorta represents an alternative to the conventional approach for traumatic aortic rupture. Because of the lower invasivity avoiding thoracotomy and use of heparin, endovascular repair can be applied in acute patients without the risk of destabilizing pulmonary, head or abdominal traumatic lesions. However, despite the good deal of convincing evidence for endovascular treatment for thoracic aortic diseases and for traumatic aortic injuries as a valid and efficacious alternative to surgery, several reports show a variety of late complications of thoracic endografts especially for first-generation stent-grafts. In light of this, is the endovascular treatment really safe, efficacious and free from complications in the long term? This manuscript aims to offer a moment of reflection on this important chapter of aortic pathology. Copyright © 2013 Science International Corp.

Key Words

Aortic · Traumatic · Endovascular · Acute injury

Introduction

Traumatic rupture of the thoracic aorta is a lifethreatening lesion and it occurs in 10 to 30% of deaths due to blunt thoracic trauma and it is the second most common cause of death after head injuries [1,2]. The highest mortality usually occurs within the first few hours after injury; almost 90% of patients die at the scene of the accident and approximately one third of patients who arrive at the hospital die before surgical treatment [3,4].

The first references about aortic rupture date back to Vesalius in 1557, after falling from a horse. Nowadays traumatic aortic rupture is the second most common cause of trauma-related deaths, leading to 8000 deaths per year in USA [5].

The majority of tears or ruptures occur at the aortic isthmus; at this site, the relatively mobile thoracic aorta joins the fixed arch and the insertion of the ligamentum arteriosus. In 1947, Strassman reported in a cohort of 7000 autopsies only 0.7% of patients with traumatic aortic rupture. But several recent investigations have shown that traumatic aortic rupture occurs in 22% of fatal blunt trauma [1,6]. In the era of high-speed motor vehicles, there has been an increased

© 2013 Aorta. Published by Science International Corp. ISSN 2325-4637

Fax +1 203 785 3346 E-Mail: aorta@scienceinternational.org http://aorta.scienceinternational.org

Accessible online at: http://aorta.scienceinternational.org *Corresponding Author: Luca Di Marco, MD, PhD Cardiac Surgery Department S. Orsola-Malpighi Hospital University of Bologna Bologna, Italy Tel: +390516363361, Fax: +39051345990, E-Mail: ludima08@libero.it incidence of traumatic aortic injuries. In fact, injury is often associated with rapid deceleration in road traffic accidents or falls. The use of seat belts has partially modified the characteristics of the trauma impact that leads to aortic injury. However, air bags and seatbelts do not protect against this type of impact. On the other hand, the frequency of lethal injuries in head-on collisions is lowered by the mandatory use of restraints, which protect the victim from thoracic and head lesions, but not from the mechanism producing aortic injury.

The diagnosis and management of traumatic thoracic aortic injuries have undergone some major changes in the last few years. In fact, the replacement of chest X-rays by routine Computed Tomography (CT) scan for screening purposes in high-speed deceleration injuries has resulted in an earlier and more frequent diagnosis of traumatic aortic injuries. Angiography has largely been replaced by CT scan for the definitive diagnosis of traumatic aortic ruptures.

Nowadays, angio CT-scan represents the gold standard for the diagnosis of traumatic aortic ruptures. It is widely available in emergency departments and it allows us to study the total body in a few seconds, also highlighting minimal aortic lesion.

The best time to intervene in the aortic lesion and whether surgery should be preceded or followed by the treatment of associated traumatic lesions have long been a matter of debate. Immediate surgery has been characterized by a high mortality and morbidity rate, ranging between 20 to 40%. In a retrospective report of 144 patients undergoing surgery within an average of six hours after arrival in hospital, there was an intraoperative mortality of 10.2% and postoperative mortality of 18.4% with major postoperative morbidity such as paraplegia reaching 10.5% [7]. In a recent multicenter trial involving 274 patients collected over 2.5 years, the overall mortality was 31%, with 14% of operative mortality in stable patients undergoing planned thoracotomy [8]. In light of this very high risk for immediate surgical intervention, in the past the surgical repair of an aortic rupture had been delayed because of coexisting injuries such as central nervous system trauma, severe respiratory insufficiency, extended body-burns sepsis, and contaminated open wounds, rendering the surgical risk too high as reported by Akins in 1981 [9].

Because of the high morbidity and mortality, since 1992 we have delayed aortic repair in all patients who have arrived alive at the hospital unless signs of impending aortic rupture such as hemodynamic instability, massive hemothorax, and/or contrast media extravasation on CT-scan were present [10]. Several studies have shown that delayed repair of traumatic aortic injuries has significant survival benefits and a much lower mortality rate compared with early open repair [11]. In 2005 we reported an improvement of patient outcome with traumatic rupture of the thoracic aorta by delaying surgical repair until after management of major associated injuries and in the absence of signs of impending rupture [12].

In light of this, in our institutions, for the management of traumatic aortic lesions, we routinely adopt the algorithm reported in Figure 1. Surgery or endovascular treatment is delayed in the case of stable patients, while immediate surgical or endovascular repair is reserved for unstable patients with signs of impending rupture.

It is clear that despite developments in operative techniques, considerable operative mortality and morbidity associated with a surgical approach still remain, even if delayed. Endovascular stent grafting of the thoracic aorta provides an exciting new alternative to the conventional approach for treating traumatic aortic rupture and it is emerging as the preferred technique for elective or emergent treatment of descending thoracic aortic lesions. It is a less invasive approach compared with open surgery and it is preferable for stabilization of the aortic lesion in patients with multiple traumatic injuries. Because of the lower invasiveness, avoiding thoracotomy and the use of heparin, endovascular repair can be applied in the acute patients without the risk of destabilizing pulmonary, head, or abdominal traumatic lesions. In early clinical series, endovascular treatment demonstrated lower morbidity and mortality in comparison with open surgical repair even in high-risk patients [13]. At present, several reports in the literature have provided data on comparative results of endovascular therapy with respect to open surgery, supporting the use of stent graft in traumatic aortic injuries, both in acute and chronic cases. In a 2008 meta-analysis, Xenos showed that endovascular treatment of descending thoracic aortic trauma is a valid alternative to open surgical repair. It is associated with lower postoperative mortality and ischemic spinal cord complication rates [14].

In 2008 Demetriades, in a multicenter study of the American Association for the surgery of Trauma re-

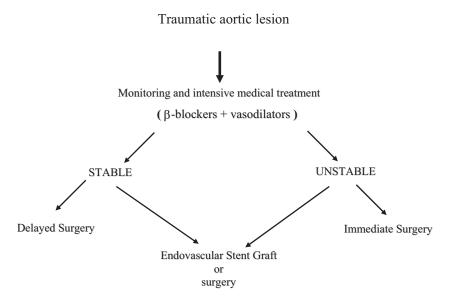


Figure 1. Bologna's strategy in case of traumatic aortic rupture.

ported a considerable reduced mortality rate in the endovascular repair group as compared with open surgical repair [15].

Even if the majority of traumatic injuries are stable lesions, in approximately 5% of them, the risk of rupture may be high in the acute phase. Signs of impending rupture such as uncontrolled blood pressure, contrast medium extravasation on CT-scan, repeated hemothorax, periaortic hematoma, or irregular lesions are considered signs of instability [16].

Even for endovascular treatment in the acute phase we deal with the same question: emergency or delayed treatment? Sometimes the aortic tear, acting with a valve mechanism, may cause a pseudocoarctation syndrome producing a reduction of flow in the descending aorta with lower extremity ischemia. This complication represents an emergency, accounting for 10% of victims. In these unstable patients, endovascular techniques offer a suitable alternative to emergency open repair, even if some limitations of the endovascular procedures exist such as possible facial bone trauma which contraindicates transesophageal echo or frequent aortic wall intramural hematoma with consequent high risk of stent-graft migration.

Because endovascular treatment requires some peculiar anatomic conditions, not all the patients can be treated. Proper peripheral vascular access (at least 7–8 mm of diameter of femoral or iliac artery) is necessary, but this condition is not always available, especially in young patients. One of the most important anatomic characteristics of any lesion allowing endovascular treatment is the presence of an adequate proximal neck (at least 5 mm of non-diseased aortic wall from the origin of the subclavian artery). The aortic isthmus is usually very close to the left subclavian artery and sometimes the lesion is in contiguity with, or at a limited distance from, the origin of the vessel. Stent grafting of the descending thoracic aorta ideally requires a proximal landing zone distal to the origin of the left subclavian artery and proximal to the visceral arteries.

Several studies have reported the artificial creation of a landing zone by covering the left subclavian artery with the stent graft, with or without previous subclavian to carotid transposition or bypass grafting, increasing the risk of ischemic complications such as stroke, left arm and spinal cord ischemia, or cerebellar infarction [17–19]. Revascularization of the left subclavian artery with transposition or bypass from the carotid artery has been shown to prevent these complications.

Sepehripour in a meta-analysis of 2011 showed that when coverage of the left subclavian artery is anatomically necessary, partial coverage is better than complete coverage in order to avoid these complications. Revascularization may be considered, but these decisions should consider the individual patient scenario [20].

But is the endovascular treatment really safe, efficacious, and free from complications in the long term? In general, despite the fact that there is a good deal of convincing evidence for endovascular treatment for thoracic aorta diseases and for traumatic aortic injuries as a valid and effective alternative to surgery, several reports show a variety of late complications, especially for first-generation thoracic endografts [21].

Both short- and midterm outcomes after endografting thoracic aortic lesions are encouraging, with significantly lower morbidity and early mortality compared with open surgery. However, despite emerging popularity and growing interest as an alternative to surgery, endograft design and manufacturing have not kept pace with growing clinical ambition. Major challenges associated with endovascular procedures using the current generation of endografts range from the relative rigidity and size of the delivery system to the failure of thoracic endografts to conform snugly with the anatomy of the aortic arch [22].

Various structural and positional changes in older first-generation endografts have been reported. The time-related changes in shape, physical structure and position of the stent-grafts have, as a consequence, secondary endoleaks, graft thrombosis, aneurysm rupture, and reperfusion from collaterals [23]. The aorta of patients with traumatic injuries differs from atherosclerotic diseased vessels for which these grafts were designed. Usually these patients are younger and have normal, smaller proximal aortic diameters, such that grafts are oversized by 10% to 20%. Since smaller endografts are not available, large mismatches between the diameter of the aorta and the endograft may occur, thus increasing the risk for endograft collapse. In case of bigger oversizing, the endograft undergoes significant compression forces and torque at the proximal descending aortic angle. Excessive oversizing, especially if by greater than 25%, may cause wrinkling of the stent graft and make it subject to collapse [24]. The collapse may occur within 24 hours after stent implantation or after some months. Graft collapse may cause total aortic closure and distal malperfusion [25–27].

Usually, it is common to use more than one component to treat a thoracic aortic aneurysm, which causes some risk of device separation as the aortic wall remodels, especially if there is insufficient component overlap or coupling [28]. As a consequence, type I and type III endoleaks are more likely to occur due to challenging seal zones and device migration increasing the likelihood of developing systemic pressures within the aneurysm sac.

Physiological coarctation of the aorta from protrusion of a thoracic stent graft into the arch is a complication of thoracic stent grafting distinct from the more commonly described graft collapse. The protrusion of the stent graft into the arch causes the obstruction of the aorta. Patients may present with symptoms of aortic arch coarctation such as proximal hypertension, left upper extremity ischemia, or left carotid or vertebral insufficiency despite having a patent endograft [29]. Perforation of the aortic wall by a stent graft is an infrequent complication of thoracic stent-graft implantation. This complication is usually due to the metallic component of the stent causing friction against the aortic wall because of continuous pulsatility of the aorta. This complication underlines the importance of completely examining the longterm durability and compatibility of prosthetic materials [30].

Aortoesophageal fistula secondary to thoracic aortic stent-graft placement is an unusual but catastrophic complication of endovascular repair of the thoracic aorta with very limited therapeutic options. The fistulae may arise secondary to the development of pseudoaneurysm, endoleak into the residual aneurysm sac, or erosion of the stent graft through the aorta from graft infection [31–34].

In light of the above and in consideration of the complications which endoprostheses may undergo, continuous and meticulous follow-up of these devices with regular imaging is advised [35].

It is clear that frequent and sustained surveillance is essential for safe management of patients. The primary motivation for close surveillance includes the evaluation of residual aneurysm sac size, presence of endoleak, and device migration allowing an early identification of potential adverse events. In fact a reintervention rate of 10% per year has been reported for treatment of problems identified on follow-up surveillance [36].

The CT-scan is usually the method of choice for periodic assessments during follow-up protocols after endovascular aneurysm repair (EVAR). The routine use of contrast-enhanced CT-scans has become more controversial since repeated scans with their inherent ionizing radiation have been suggested to have carcinogenic potential. This evidence suggests that lessfrequent CT scans may simplify the follow up protocol, reduce radiation exposure and the total cost of EVAR [37,38].

In spite of the fact that endovascular techniques can now be considered an effective alternative to open surgery in the treatment of traumatic thoracic aortic injuries, the long-term durability of a stent graft for traumatic aortic lesions is still unknown. Techniques and technologies continue to improve and the results obtained should be viewed as work in progress.

Early outcomes appear successful, but these results may be deceiving especially for those patients with compromised anatomy and the risk of late stent-graft migration, loss of device integrity and local erosion or rupture of the aortic wall. At present, the lack of long-term data and the evolving technology of stentgraft design should be an incentive for exercising great care in patient selection [39,40].

Comment on this Article or Ask a Question

References

- 1. Richens D, Kotidis K, Neale M, Oakley C, Fails A. Rupture of the aorta following road traffic accidents in the United Kingdom 1992-1999. The results of the co-operative crash injury study. Eur J Cardio-Thoracic Surg. 2003;23: 143-148. 10.1016/S1010-7940(02)00720-0
- 2. Bertrand S, Cuny S, Petit P, Trosseille X, Page Y, Guillemot H, et al. Traumatic rupture of thoracic aorta in real-world motor vehicle crashes. Traffic Inj Prev. 2008;9:153-161. 10. 1080/15389580701775777
- Attar S, Cardarelli MG, Downing SW, Rodriguez 3. A, Wallace DC, West RS, et al. Traumatic aortic rupture: recent outcome with regard to neurologic deficit. Ann Thorac Surg. 1999;67:959-964. 10.1016/S0003-4975(99)00174-5
- P. Traumatic aortic rupture: twenty-year metaanalysis of mortality and risk of paraplegia. Ann Thorac Surg. 1994;58:585-593. 10.1016/0003-4975(94)92270-5
- 5. Fattori R, Russo V, Lovato L, Di Bartolomeo R. Optimal management of traumatic aortic injury. Eur J Vasc Endovasc Surg. 2009;37:8-14. 10.1016/j.ejvs.2008.09.024
- Strassman G. Traumatic rupture of the aorta. Am Heart J. 1947;33:508-515. 10.1016/0002-8703(47)90098-7
- Oller DW, Flowe KM, et al. Thoracic aorta injuries: management and outcome of 144 patients. J Trauma. 1996;40:547-555; discussion 555-556.10.1097/00005373-199604000-00005
- 8. Fabian TC, Richardson JD, Croce MA, Smith JS Jr, Rodman G Jr, 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: 374-380; discussion 380-383.
- Akins CW, Buckely MJ, Daggett W, McIlduff 17. Feezor RJ, Lee WA. Management of the left JB, Austen WG. Acute traumatic disruption of the thoracic aorta: a ten-year experience. Ann Thorac Surg. 1981;31:305-309. 10.1016/ S0003-4975(10)60955-1
- 10. Galli R, Pacini D, Di Bartolomeo R, Fattori R, Turinetto B, Grillone G, et al. Surgical indi-

cations and timing of repair of traumatic ruptures of the thoracic aorta. Ann Thorac Surg. 1998;65:461-464. 10.1016/S0003-4975(97)01302-7

- 11 . Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Blunt traumatic thoracic aortic injuries: early or delayed repair-Results of an American Association for the Surgery of Trauma prospective study. J Trauma. 2009;66:967-973. 10.1097/TA.0b013e31817dc483
- 12. Pacini D, Angeli E, Fattori R, Lovato L, Rocchi G, Di Marco L, et al. Traumatic rupture of the thoracic aorta: ten years of delayed management. J Thorac Cardiovasc Surg. 2005;129: 880-884. 10.1016/j.jtcvs.2004.10.012
- 4. Von Oppell UO, Dunne TT, De Groot MK, Zilla 13. Appoo JJ, Moser WG, Fairman RM, Cornelius KF, Pochettino A, Woo EY, et al. Thoracic aortic stent-grafting: improving results with newer generation investigational devices. J Thorac Cardiovasc Surg. 2006;131:1087-1094. 10.1016/j.jtcvs.2005.12.058
 - 14. Xenos ES, Abedi NN, Davenport DL, Minion DJ, Hamdallah O, Sorial EE, et al. Metaanalysis of endovascular versus open repair for traumatic descending thoracic aortic rupture. J Vasc Surg. 2008;48:1343–1351. 10. 1016/j.jvs.2008.04.060
- 7. Hunt JP, Baker CC, Lentz CW, Rutledge RR, 15. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study. J Trauma. 2008;64:561-571. 10.1097/TA. 0b013e3181641bb3
 - 16. Pate JW, Fabian TC, Walker W. Traumatic rupture of the aortic isthmus: an emergency? World J Surg. 1995;19:119-125; discussion 125-126.
 - subclavian artery during TEVAR. Semin Vasc Surg. 2009;22:159-164. 10.1053/j. semvascsurg.2009.07.007
 - 18. Cooper DG, Walsh SR, Sadat U, Noorani A, Hayes PD, Boyle JR. Neurological complications after left subclavian artery coverage

during thoracic endovascular aortic repair: a systematic review and meta-analysis. J Vasc Surg. 2009;49:1594-1601. 10.1016/j.jvs.2008. 12.075

- 19. Steinberg GK, Drake CG, Peerless SJ. Deliberate basilar or vertebral artery occlusion in the treatment of intracranial aneurysms. Immediate results and long-term outcome in 201 patients. J Neurosurg. 1993;79:161-173. 10.3171/jns.1993.79.2.0161
- 20. Sepehripour AH, Ahmed K, Vect JA, Anagnostakou V, Suliman A, Ashrafian H, et al. Management of the left subclavian artery during endovascular stent grafting for traumatic aortic injury-A systematic review. Eur J Vasc Endovasc Surg. 2011;41:758-769. 10. 1016/j.ejvs.2011.01.007
- 21. Kasirajan K, Milner R, Chaikof EL. Late complications of thoracic endografts. J Vasc Surg. 2006;43:94A-99A 10.1016/j.jvs.2005.10. 064
- 22. Nienaber CA, Kische S, Ince H. Thoracic aortic stent-graft devices: problems, failure modes, and applicability. Semin Vasc Surg. 2007;20:81-89. 10.1053/j.semvascsurg.2007. 04.005
- 23. Umscheid T, Stelter WJ. Time-related alterations in shape, position, and structure of self-expanding, modular aortic stent-grafts: a 4-year single-center follow-up. J Endovasc Surg. 1999;6:17-32.
- 24. Annamalai G, Cook R, Martin M. Endograft collapse following endovascular repair of traumatic aortic injury. Diagn Interv Radiol. 2011; 17:84-87. 10.4261/1305-3825.DIR.2184-09.0
- 25. Lazar HL, Varma PK, Shapira OM, Soto J, Shaw P. Endograft collapse after thoracic stent-graft repair for traumatic rupture. Ann Thorac Surg. 2009;87:1582-1583. 10.1016/j. athoracsur.2008.09.012
- 26. Idu MM, Reekers JA, Balm R, Ponsen K-J, de Mol BAJM, Legemate DA. Collapse of a stentgraft following treatment of a traumatic thoracic aortic rupture. J Endovasc Ther. 2005; 12:503-507. 10.1583/04-1515R.1
- 27. Jonker FHW, Schlosser FJV, Geirsson A, Sumpio BE, Moll FL, Muhs BE. Endograft collapse

after thoracic endovascular aortic repair. J Endovasc Ther. 2010;17:725-734. 10.1583/10-3130.1

- 28. Makaroun MS, Dillavou ED, Kee ST, Sicard G. Chaikof E, Bavaria J, et al. Endovascular treat- 33. Chiba D, Hanabata N, Araki Y, Sawaya M, ment for thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. J Vasc Surg. 2005;41:1-9. 10.1016/j.jvs.2004.10.046
- 29. Go MR, Siegenthaler MP, Rhee RY, Gupta N, Makaroun MS, Cho JS. Physiologic coarctation of the aorta resulting from proximal protrusion of thoracic aortic stent grafts into the arch. J Vasc Surg. 20;48:1007-1011. 10. 1016/j.jvs.2008.05.027
- 30. Cosin O, Rousseau H, Otal P, Cron C, Chabbert V, Joffre F. Late perforation of a thoracic aortic Dacron graft by a metallic stent-graft component. J Endovasc Ther. 2006;13:676-680. 10.1583/06-1881.1
- 31. Eggebrecht H, Baumgart D, Radecke K, von Birgelen C, Treichel U, Herold U. Aortoesophageal fistula secondary to stentgraft repair of the thoracic aorta. J Endovasc 37. Brenner DJ, Hall EJ. Computed tomogra-Ther. 2004;11:161-167. 10.1583/03-1114.1
- 32. Yavuz S, Kanko M, Ciftci E, Parlar H, Agirbas H, Berki T. Aortoesophageal fistula secondary to

thoracic endovascular aortic repair of a descending aortic aneurysm rupture. Heart Surg Forum. 2011;14:E249-251. 10.1532/HSF98. 20101179

- Yoshimura T, Aoki M, Shimoyama T, et al. Aortoesophageal fistula after thoracic endovascular aortic repair diagnosed and followed with endoscopy. Intern Med. 2013;52: 451-455. 10.2169/internalmedicine.52.9139
- 34. Muradi A, Yamaguchi M, Kitagawa A, Nomura Y, Okada T, Okita Y, et al. Secondary aortoesophafeal fistula after thoracic endovascular aortic repair for a huge aneurysm. Diagn Interv Radiol. 2013;19:81-84. 10.4261/ 1305-3825.DIR.5912-12.1
- 35. Hopkinson BR. Late failure of early-model endografts: a complication whose time has come? J Endovasc Surg. 1998 Aug;5:273.
- 36. Milner R, Kasirajan K, Chaikof EL. Future of endograft surveillance. Semin Vasc Surg. 2006:19:75-82.
- phy-an increasing source of radiation exposure. N Engl J Med. 2007;357:2277-2284. 10.1056/NEJMra072149

- 38. Dias NV, Riva L, Ivancev K, Resch T, Sonesson B, Malina M. Is there a benefit of frequent CT follow-up after EVAR? Eur J Vasc Endovasc Surg. 2009;37:425-430. 10.1016/j.ejvs.2008. 12.019
- 39. Jones WB, Taylor SM, Kalbaugh CA, Joels CS, Blackhurst DW, Langan EM 3rd, et al. Lost to follow-up: a potential under-appreciated limitation of endovascular aneurysm repair. J Vasc Surg. 2007;46:434-440; discussion 440-441. 10.1016/j.jvs.2007.05.002
- 40. Sarac TP, Gibbons C, Vargas L, Liu J, Srivastava S, Bena J, et al. Long term follow-up of type II endoleak embolization reveals the need for close surveillance. J Vasc Surg. 2012;55:33-40. 10.1016/j.jvs.2011.07.092

Cite this article as: Di Marco L, Pacini D, Di Bartolomeo R. Acute Traumatic Thoracic Aortic Injury: Considerations and Reflections on the Endovascular Aneurysm Repair. Aorta 2013;1(2):117-122. DOI: http://dx.doi.org/ 10.12945/j.aorta.2013.12-009