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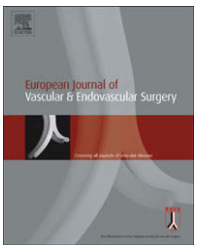

THORACIC AORTIC DISEASE

Optimal Management of Traumatic Aortic Injury☆

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Abstract *Background:* The best time to intervene in traumatic aortic injuries has long been a matter of debate. While emergency surgery is characterized by high morbidity and mortality, initial medical management of uncomplicated aortic injury and subsequent delayed surgery resulted in better outcome.
Methods and results: From analysis of medical literature of the last 10 years, major paradigm shift in management of traumatic injuries includes the use of different imaging methods for diagnosis , with a almost complete elimination of aortography and transesophageal echocardiography in favour of CT scan, and a significant change in method of definitive repair, shifting from exclusively open techniques in 1997 to predominantly endovascular repairs in 2007. At present several reports in literature provide data on comparative results of endovascular therapy with respect open surgery, supporting the use of stent-graft in traumatic injuries, both in acute and chronic cases. The authors' personal experience comprises 58 patients treated with endovascular stent-graft repair, with no mortality or treatment failure even during 11 years follow-up.
Conclusions: For many years traumatic aortic injury has been considered a highly lethal lesion and a potential cause of death in blunt chest trauma. Because of the lower invasivity endovascular repair can be applied in traumatic aortic injury with very low risk and limited impact on trauma destabilization. Long term follow-up seems indicate a substantial durability of the procedure.

Introduction

Traumatic aortic injury (TAI) is a lesion extending from the intima to the adventitia, occurring as a result of trauma. The era of high-speed motor vehicles has brought with it an increased incidence of TAI. Between 1936 and 1942, in a cohort of 7000 autopsies, Strassman[1] found only 51

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patients with traumatic aortic rupture secondary to vehicular collision, whereas several recent investigations have shown that TAI occurs in 10—30% of adults sustaining fatal blunt trauma. Recent investigations have shown that TAI occurs in 10—30% of adults sustaining fatal blunt trauma[2,3]; it therefore represents one of the most common causes of death at the scene of vehicular accidents, accounting for 8000 victims/year in USA. The more sophisticated prehospital care and the proliferation of rapid transport for patients have resulted in an average increase in the number of patients treated. The lesion may be generated by many different types of sudden-deceleration injury, including car and motorcycle collisions, falls from a height or blast injuries, airplane and train crashes, skiing and equestrian accidents. The region subjected to the greatest strain is put upon the isthmus, where the relatively mobile thoracic aorta joins the fixed arch and the insertion of the ligamentum arteriosus. Aortic ruptures occur at this site in 80% of the pathological series and in 90—95% of the clinical series.[2—9] Another hypothesis considers a major role in bending stress by chest compression: a direct impact produces flexion of the aortic arch upon hilar structures acting as fulcrum.[10] An additional force such as a sudden increase in hydrostatic pressure, acting with ''water hammer'' effect, has also been postulated. Considering the different causes and types of impact which produce the aortic lesion, it is reasonable to propose that not only one mechanism but a combination of many is also involved in its determination.

The mandatory use of a seat belt, first introduced in the 1970s in Finland, and progressively throughout the world, 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. Such injuries can be expected to gain prominence in road traffic injury statistics,[2] since 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.

Pathology

In the 1950s Loren Parmley, a pathologist in the US Armed Forces performed an historic analysis on 296 cases of TAI among victims of Korea's war.[9] This study, which is up to now the widest pathological series, clearly defined the characteristics of the aortic lesion and emphasized the time relationships between the trauma and subsequent death, underlying the high lethality of the untreated lesions. According to the Parmley study (9 out of 296 aortic injuries) the lesion may be classified as follows: (1) intimal hemorrhage; (2) intimal hemorrhage with laceration; (3) medial laceration; (4) complete laceration of the aorta; (5) false aneurysm formation; (6) periaortic hemorrhage. Areas of intimal hemorrhage were noted at autopsy in patients who died of other fatal lesions. The endothelial layer may be intact or the hemorrhage may be associated with circumscribed laceration of the endothelial and internal elastic lamina of the intima. It is probable that lesions of this type occurred frequently but were not recognized. When blood lifts up the upper edge of the intimo-medial lesion, separates the media and expands in the sub-adventitial space. At histology, the internal elastic lamina may be interrupted, thereby exposing the media. The tear is transverse in 80—90% of patients and involves all or part of the aortic circumference. When the lesion involves intimal and medial layers, a false aneurysm formation occurs. The aneurysm is fusiform in the case of a circumferential lesion involving the entire wall on the transversal plane, while in a partial lesion in which only a portion of the wall is lacerated, it appears as localized diverticulum. Periaortic hemorrhage is frequently associated with the aortic injury independently of the type of lesion. Complete rupture of the aorta including the adventitia and the periadventitial connective tissue leads to immediate death. However, mediastinal hematoma may permit temporary survival. In the Parmley report, 9 of 38 patients who survived temporarily had complete transection, their survival being dependent on the hematoma contained in periaortic and mediastinal tissues. Soon after the injury, the pouch of the aneurysm contains a thrombus consisting of fibrin and enmeshed red blood cells. It follows fibroblastic proliferation and neo-vascularization in the aortic wall. After 2—3 weeks the thrombus becomes organized and the wall of the pouch lined with endothelial cells initiates the healing process.

Trend in management of aortic injuries

For many years it has been conventional to consider traumatic aortic injury to be a highly lethal injury. This concept was primarily based on the historical study by Parmley.[9] Remarkably, Parmley's analysis estimated that 85% of the victims died on the scene from free aortic rupture; of those who survived at least for 1 h, 30% died within 6 h, 49% within 24 h and 90% within four months. This article has been referred to every subsequent report on this topic and has influenced the general opinion over the next 50 years, leading to the concept that immediate surgery should be advocated in traumatic aortic injury in any case. However, as reported in several clinical series,[10—17] the risk of a complete rupture of the aorta is not very high after 4—6 h, especially if patients, once admitted to hospital, are immediately submitted to pharmacological treatment with controlled hypotension.

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.[10—18] Immediate surgery has been characterized by a high mortality and morbidity rate (20—40%): in a report of 144 patients undergoing surgery within an average of 6 h 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%.[4] In a recent prospective multicenter trial[11] among 274 patients collected over 2.5 years, the overall mortality was 31%, with 14% of operative mortality in stable patients undergoing planned thoracotomy.

This first phase after the trauma is life threatening and accident victims should be taken to hospital as quickly as possible. A prompt diagnosis of aortic wall injury is mandatory and an aggressive intravenous

therapy with vasodilators and beta-blocking drugs must be started to reduce the aortic wall stress and the risk of lethal aortic rupture. According to Pate[10] the risk of rupture of a periaortic hematoma contained in the mediastinum can be avoided if the systolic blood pressure is constantly maintained below 120 mmHg. On the subsequent days a process of organization of the hematoma usually develops and with time it will turn into a strong fibrous tissue, with the formation of a pseudoaneurysm[19] that has the same risk of rupture as a true aneurysm of similar size. Patients must be admitted to an intensive care unit with continuous monitoring of ECG, arterial and central venous pressure, renal function and peripheral metabolism. An arterial systolic pressure exceeding 90 mmHg should be an indication to limit fluid replacement and any hemodynamic support in hypotensive patients. Monitoring of respiratory function and eventual intubation and mechanical ventilation is fundamental in polytraumatized patients with respiratory insufficiency due to central nervous system injury, pulmonary contusion and pleural effusion with measurement of chest tube outputs.

The diagnosis and treatment of blunt TAI have undergone many significant changes over the last decade. Changes in management paradigms have been reported by the American Association for the Surgery of Trauma (AAST). Comparing two observational multicentric studies, the AAST 1[11] and AAST 2,[20] covering 10 years of time interval, there was an almost complete elimination of aortography and transesophageal echocardiography (TEE) with increase of CT scan as the methods of definitive diagnosis of TAI: the percentage of patients who had CT scan diagnosis of the aortic injury increased from 34.8% in AAST 1 to 93.3% in AAST 2. Moreover the time from admission to definitive repair increased significantly from AAST 1 to AAST 2 (16.5 ± 70.8 h vs. 54.6 ± 106.6 h, $p < 0.001$), as well as the method of definitive repair, shifting from exclusively open techniques in 1997 to predominantly endovascular repairs in 2007. In the AAST 2 study, the vast majority of repairs (125 of 193 or 64.8%) were performed with endovascular stent grafts. Most importantly, there were major outcome differences between the two studies: a significant reduction in the overall mortality during the second study period (from 31% in AAST 1 to 13.0% in the AAST 2), along with a significant reduction of procedure-related paraplegia (from 8.7% to 1.6%).

The overall mortality and the incidence of major complications are lower when it is possible to delay surgery. All the necessary procedures of distal aortic perfusion can be safely performed with heparin without the risk of aggravation of other traumatic lesions. It is important to remember that 90% of patients with aortic injury have associated with other open and closed traumatic lesions of different areas (orthopedic, abdominal, closed-head injury) which may cause a rapid evolution into shock and coma, thus influencing the patient's outcome. Therefore, the treatment of associated lesions is fundamental in these patients and it is another incentive to delay surgical intervention in the aorta. CT scan and MRI offer non-invasive assessment of the anatomical characteristics of the aortic lesions and can be used to monitor their evolution.[18,21–23] Signs indicative of imminent free rupture, such as an increase in size of the periaortic hematoma or pseudo-aneurysm, recurrent hemothorax, associated with a poorly controlled arterial pressure, can be promptly identified and therefore provide an indication for emergency surgery.

Endovascular treatment

Early results and comparison with open surgery

From 1996 the introduction of endovascular techniques for the thoracic aorta in the clinical practice opened lesser invasive option especially for patients in which emergency treatment is necessary. After initial limited series and case reports, endovascular treatment has become the method of choice in management of TAI.[24–27,28–31] At present several reports in literature provided data on comparative results of endovascular therapy with respect to open surgery, supporting the use of stent graft in traumatic injuries, both in acute and chronic cases. The authors' personal experience is summarized in Table 1. Technical success was obtained in all patients submitted to stent graft repair. No mortality or failure of the endovascular procedure or persistent endoleak was observed during follow-up.

Because of the lower invasivity, 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. The Talent Thoracic Retrospective Registry,[31] which collected data on 457 patients treated for thoracic aortic lesions in 7 major European centers, reported excellent results of endovascular repair in traumatic lesions: among 85 acute and chronic aortic injuries, this type of aortic lesions resulted to be associated with the lowest morbidity and mortality and presented the best long term outcome. Significantly a very low rate of primary and secondary endoleaks is reported in case series, probably due to the good wall conditions (no atherosclerotic alteration of the aortic wall, no mural thrombus) at the neck sites.

Table 1

Acute aortic injury	**27/58**
Mean age (± SD)	36,1 (10,2)
ASA Class (± SD)	3 (1,7)
Time from accident to treatment (median, range)	24 hours (5-288)
Mortality	0
Complications	CVA (1 pt)
Chronic pseudoaneurysm	**31/58**
Mean age (± SD)	44,3 (15,4)
Time from accident to treatment (median, range)	3 months (0,5-216)
Mortality	0
Complications	0

Endovascular Repair TAI (University of Bologna, 1997–2008).

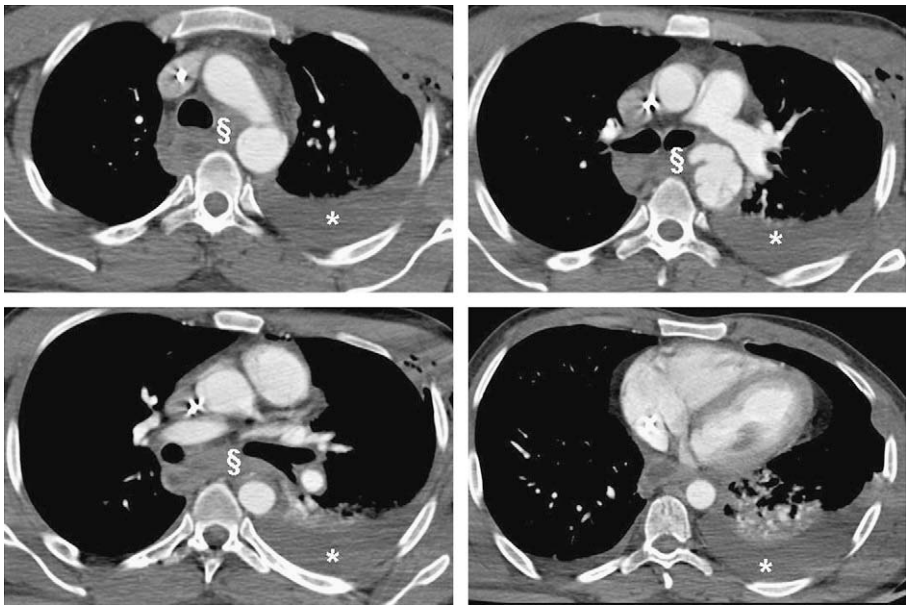

Figure 1 Axial CT images of an aortic isthmic rupture with aortic wall discontinuity, periaortic mediastinal hematoma (§) and pleural effusion (*) with associated lung atelectasia.

Endovascular management of acute and chronic cases

At present several standard measurements of thoracic stent grafts are promptly available allowing its use in emergency. In the past few years, initial medical management and delayed surgery of the aortic traumatic injury represent an important advance in the difficult management of polytrauma, substantially reducing operative and overall mortality. However, there are some patients in whom

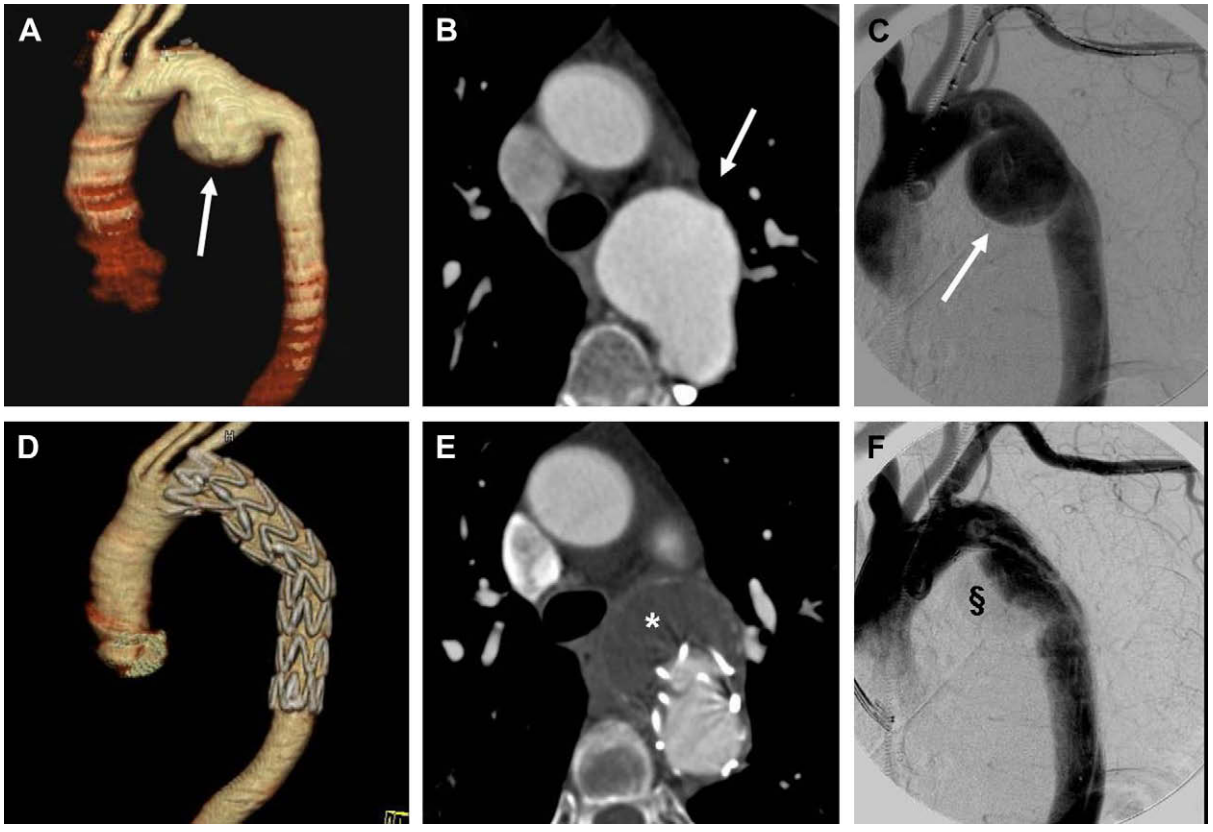

Figure 2 Multidetector CT images (A, B, D, E) and angiographic images (C, F) of a large chronic post-traumatic pseudo-aneurysm before (A—C) and after (D—F) endovascular treatment: the aneurysm (arrows) is partially thrombosed immediately after procedure (§) and fully sealed on CT scan at patient discharge (*).

delayed surgery cannot be applied. Even if the majority of traumatic aortic 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 aortic profile discontinuity at CT scan (Fig. 1), periaortic hematoma, repeated hemothorax, and uncontrolled blood pressure are considered signs of instability. 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, which represents a surgical emergency, is accounted for 10% of victims.[32] In these unstable patients endovascular techniques offer a suitable alternative to emergency open repair. There is no requirement for full heparinization and the blood loss is minimal. The risk of paraplegia seems to be very low even in extensive atherosclerotic aneurysms in which the aortic coverage with the stent graft extends from the left subclavian artery to the celiac axis. Therefore, due to the limited longitudinal extension of a traumatic lesion, only one segment of stent graft is used with very low risk of intercostal arteries' occlusion.

In the chronic post-traumatic aneurysm endovascular treatment represents a favorable alternative treatment of an asymptomatic disease that is frequently recognized several years after the trauma. Chronic post-traumatic aneurysms are potential evolving lesions. Death from rupture may occur many years after injury sometimes without any signs and symptoms onset.[19] Because it is impossible to predict which aneurysm still remains quiescent, elective repair is always recommended for both symptomatic and asymptomatic lesions. Advances in surgical techniques and spinal cord protection over the years significantly reduced operative mortality and paraplegia in elective surgical repair of the thoracic aorta. In the largest surgical series, operative mortality for chronic

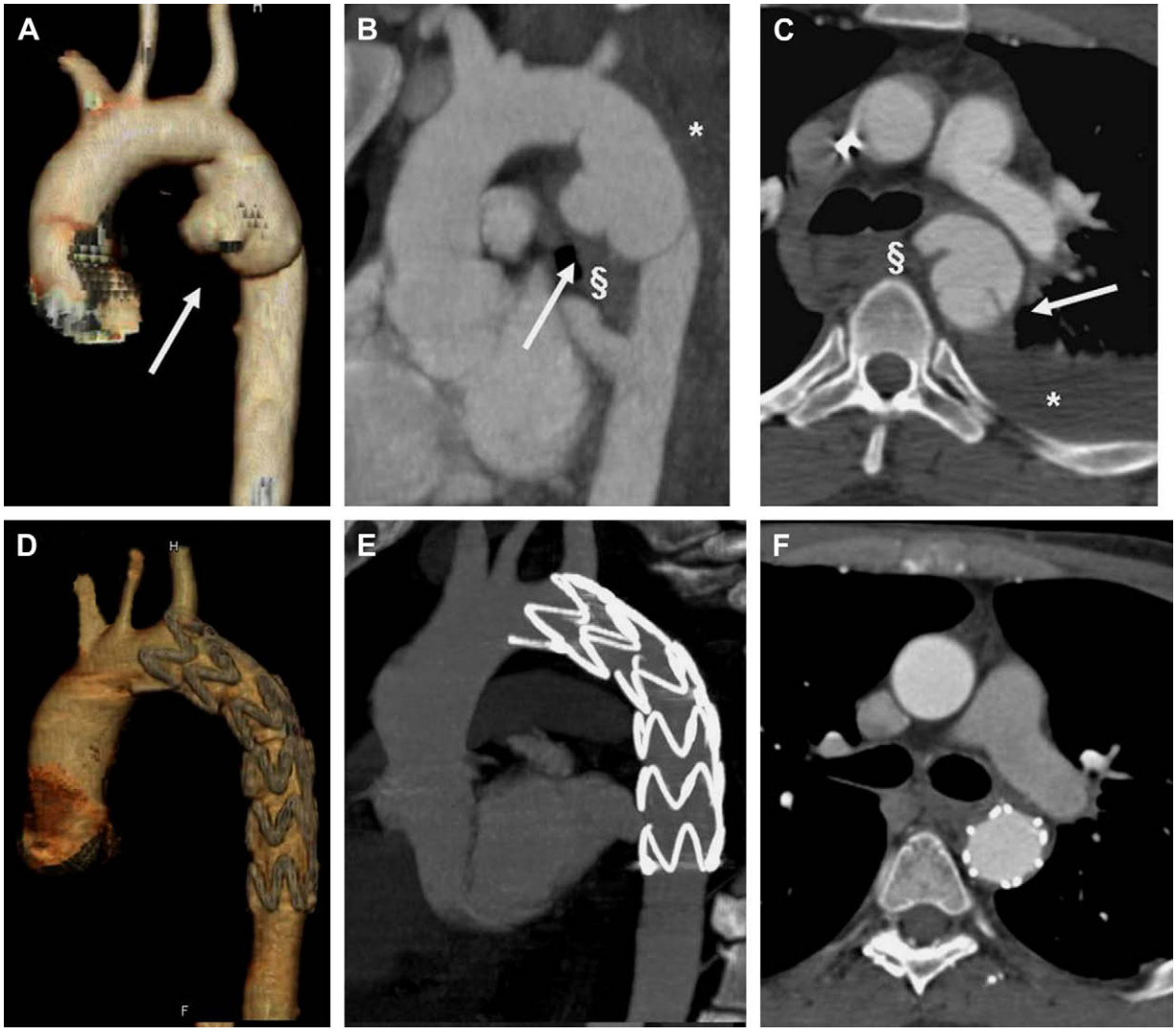

Figure 3 Multidetector CT images of an acute traumatic aortic injury before (A—C) and after (D—F) endovascular treatment: a circumferential aortic injury could be easily detected (arrows), together with periaortic mediastinal hematoma (§) and pleural effusion (*). The lesion is very close to the left subclavian artery. After endovascular treatment, the lesion is completely resolved as well as pleural effusion and mediastinic hematoma. The left subclavian artery is open.

post-traumatic aneurysms ranges from 0% to 10% and paraplegia accounts for 5% of cases.[33,34] The risk of paraplegia in surgery of chronic post-traumatic aneurysm is very low as compared to atherosclerotic ones, because of the limited extension of the pseudo-aneurysm that usually does not extend beyond the first pair of intercostal arteries (Fig. 2). However, patients with chronic asymptomatic aneurysm are not always prone to accept a major thoracotomy and the risk of dreadful complications. Endovascular treatment may play an important role in chronic post-traumatic aneurysms management allowing a low invasive option with limited risk of complications.

Assessment of proper anatomy for stent graft

The endovascular treatment needs some peculiar anatomical conditions to be performed so that not all the patients can be treated. At least a proper peripheral vascular access (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 anatomical characteristic of any lesion allowing endovascular treatment is the presence of an adequate proximal neck. The aortic isthmus is usually very close to the left subclavian artery and sometimes the lesion in contiguity or with a limited distance from the vessel. Several studies reported the artificial creation of an aortic neck, covering the left subclavian artery with the stent graft, with or without previous subclavian to carotid transposition or by-pass grafting. However, the risk of vertebral ischemia and cerebellar infarction is reported up to 5% for interventional treatment of intracranial aneurysm treated by vertebral legation, therefore we may expect the same rate of complication for endovascular coverage of the left subclavian and vertebral arteries without previous revascularization.[35] Weigang et al.[36] and Riesenman et al.,[37] respectively, reported the development of central adverse neurological events in 2 out of 20 (10%) and 3 out of 28 (10.7%) patients after overstenting of the LSA without previous revascularization. Flow compensation throughout the circle of Willis is a dynamic process which can be influenced by the diameter of the posterior communicating arteries and vasomotory regulation. Focusing our attention on acute traumatic ruptures of descending aorta, we should also consider that cerebral edema, often associated even with mild head trauma, and general anesthesia may potentially influence vasomotor effects in cerebral circulation.

In young patients, the association of endovascular procedures and carotid with subclavian by-pass (CSBP) could be criticized, because both operations have no long term follow-up regarding integrity and durability. Moreover, the long-life adjacency of the uncovered part of the stent graft to the left carotid artery is a potential source of emboli. Several articles addressed the outcomes of CSBP, reporting a graft patency between 66 and 89% at an FU of maximum 15 years.[38] Unfortunately, these data concern atherosclerotic lesions of supraortic vessels and are not applicable to this cohort of patients. Therefore, if traumatic injuries of the aorta involve young patients with an inadequate landing zone for stent graft deployment, it is also possible to proceed with conventional open surgical treatment as an alternative option to endovascular repair associated with surgical revascularization of LSA.

It is important to remind that the absence of atherosclerotic wall involvement of the aorta in post-traumatic aneurysms relies on endovascular treatment with successful aneurysm sealing even with short aortic neck (4—5 mm), without the need of left subclavian artery coverage (Fig. 3). Preoperative imaging studies are fundamental in order to define the indication to endovascular treatment and to provide precise measurement of such anatomic condition.

The accuracy of measurements then becomes essential also to verify the efficacy of the procedure along the follow-up. Usually the aneurysm sac in both acute and chronic cases presents a high rate of total shrinkage. Both Multidetector CT and MR imagings represent excellent modalities for evaluation of traumatic aortic lesion, displaying the extent of the disease without partial volume errors and providing accurate details of the aortic wall structure and proximal aortic neck.

Conclusions

For many years traumatic aortic injury has been considered a highly lethal lesion and a potential cause of death in blunt chest trauma. Despite evidence in the literature of lower morbidity and mortality, initial medical management of uncomplicated aortic injury and subsequent delayed surgery has not been easily accepted in the clinical practice. The use of endovascular techniques in patients with suitable anatomy represents a viable alternative with very low risk and limited impact on trauma destabilization.

References

1 Strassman G. Traumatic rupture of the aorta. *Am Heart J* 1947; **33**:508—15.
2 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—8.
3 Bertrand S, Cuny S, Petit P, Trosseille X, Page Y, Guillemot H, Drazetic P. Traumatic rupture of thoracic aorta in real-world motor vehicle crashes. *Traffic Inj Prev* 2008;**9**:153—61.
4 Hunt JP, Baker CC, Lentz CW, Rutledge RR, Oller DW, Flowe KM, Nayduch DA, Smith C, Clancy TV, Thomason MH, Meredith JW. Thoracic aorta injuries: management and outcome of 144 patients. *J Trauma* 1996;**40**:547—56.
5 Greendyke RM. Traumatic rupture of the aorta. Special reference to automobile accidents. *JAMA* 1966;**195**:527—30.
6 Arajarvi E, Santarvirta S, Tolonen J. Aortic ruptures in seat belt wearers. *J Thorac Cardiovasc Surg* 1989;**98**:355—61.
7 Ben-Menachem Y. Rupture of the thoracic aorta by broadside impacts in road traffic and other collisions: further angiographic observations and preliminary autopsy findings. *Journal of Trauma* 1993;**35**:363—7.
8 Shorr RM, Crittenden M, Indeck M, Hartunian SL, Rodriguez A. Blunt thoracic trauma. Analysis of 515 patients. *Ann Surg* 1987; **206**:200—5.
9 Parmley LF, Mattingly TW, Manion WC, Jahnke Jr EJ. Nonpenetrating traumatic injury of the aorta. *Circulation* 1958;**17**: 1086—101.

10 Pate JW, Fabian TC, Walker W. Traumatic rupture of the aortic isthmus: an emergency? *World J Surg* 1995;**19**:119—26.
11 Fabian TC, Richardson JD, Croce MA, Smith Jr JS, Rodman Jr 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.
12 Fattori R, Celletti F, Bertaccini P, Galli R, Pacini D, Pierangeli A, Gavelli G. Delayed surgery of traumatic aortic rupture: role of Magnetic Resonance Imaging. *Circulation* 1996;**94**:2865—70.
13 Maggisano R, Nathens A, Alexandrova NA, Cina C, Boulanger B, Mc Kenzie R, Harrison AW. Ruptures traumatiques de l'aorte thoracique: l'intervention d'urgence est-elle toujours indiquee? *Ann Chir Vasc* 1995;**9**:44—52.
14 Jahromi AS, Kazemi K, Safar HA, Doobay B, Cinà C. Traumatic rupture of the thoracic aorta: cohort study and systematic review. *J Vasc Surg* 2001;**34**:1029—34.
15 Kipfer B, Leupi F, Schuepbach P, Friedli D, Althaus U. Acute traumatic rupture of the thoracic aorta: immediate or delayed surgical repair? *Eur J Cardiothorac Surg* 1994;**8**:30—3.
16 Blegvad S, Lippert H, Lund O, Hansen OK, Christensen T. Acute or delayed surgical treatment of traumatic rupture of the descending aorta. *J Cardiovasc Surg* 1989;**30**:559—64.
17 Pacini D, Angeli E, Fattori R, Lovato L, Rocchi G, Di Marco L, et al. Traumatic rupture of the thoracic aorta: 10 years of delayed management. *J Thorac Cardiovasc Surg* 2005;**129**(4):880—4.
18 Fattori R, Celletti F, Descovich B, Napoli G, Bertaccini P, Galli R, et al. Evolution of post traumatic aneurysm in the subacute phase: magnetic resonance imaging follow-up as a support of the surgical timing. *Eur J Cardiothorac Surg* 1998;**13**:582—7.
19 Finkelmeyer BA, Mentzer RM, Kaiser DL, Tegtmeyer CJ, Nolan SP. Chronic traumatic thoracic aneurysm. Influence of operative treatment on natural history: an analysis of reported cases, 1950-1980. *J Thorac Cardiovasc Surg* 1982;**84**:257—66.
20 Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Diagnosis and treatment of blunt thoracic aortic injuries: changing perspectives. *J Trauma* 2008; **64**:1415—8.
21 Mirvis SE, Shanmuganathan K, Miller BH, White CS, Turney SZ. Traumatic aortic injury: diagnosis with contrast-enhanced thoracic CT- five-year experience at a major trauma center. *Radiology* 1996;**200**:413—22.
22 Gavant ML. Helical CT grading of traumatic aortic injuries. Impact on clinical guidelines for medical and surgical management. *Rad Clin North Am* 1999;**37**:553—74.
23 Downing SW, Sperling JS, Mirvis SE, Cardarelli MG, Gilbert TB, Scalea TM, et al. Experience with spiral computed tomography as the sole diagnostic method for traumatic aortic rupture. *Ann Thorac Surg* 2001;**72**:495—501.
24 Rousseau H, Soula P, Perreault P, Bui B, Janne d'Othee B, Massabuau P, et al. Delayed treatment of traumatic rupture of the thoracic aorta with endoluminal covered stent. *Circulation* 1999;**99**:498—504.
25 Lachat M, Pfammatter T, Witzke H, Bernard E, Wolfensberger U, Kunzly A, Turina M. Acute traumatic aortic rupture: early stent-graft repair. *Eur J Cardiothorac Surg* 2002;**21**:959—63.
26 Fattori R, Napoli G, Lovato L, Russo V, Pacini D, Pierangeli A, et al. Indications for, timing of and results after treatment of catheter based treatment of the injury of aorta. *AJR Am J Roentgenol* 2002;**179**:603—9.
27 Doss M, Balzer J, Martens S, Wood JP, Wimmer-Greinecker G, Fieguth HG, et al. Surgical versus endovascular treatment of acute thoracic aortic rupture: a single-center experience. *Ann Thorac Surg* 2003;**76**:1465—9.
28 Andrassy J, Weidenhagen R, Meimarakis G, Lauterjung L, Jauch KW, Kopp R. Stent versus open surgery for acute and chronic traumatic injury of the thoracic aorta: a single-center experience. *J Trauma* 2006;**60**:765—71.
29 Rousseau H, Dambrin C, Marcheix B, Richeux L, Mazerolles M, Cron C, et al. Acute traumatic aortic rupture: a comparison of surgical and stent-graft repair. *J Thorac Cardiovasc Surg* 2005; **129**:1050—5.
30 Schumacher H, Bockler D, von Tengg-Kobligk H, Allenberg JR. Acute traumatic aortic tear: open versus stent-graft repair. *Semin Vasc Surg* 2006;**19**(1):48—59.
31 Fattori R, Nienaber CA, Rousseau H, Beregi JP, Heijmen R, Grabenwöger M, et al. Results of endovascular repair of the thoracic aorta with the Talent Thoracic Stent-Graft: the T.T.R. registry. *J Thorac Cardiovasc Surg* 2006;**132**:332—9.
32 Malm JR, Deterling RH. Traumatic aneurysm of the thoracic aorta simulating coarctation. *J Thoracic Cardiovasc Surg* 1960; **40**:271—8.
33 Hess PJ, Howe HR, Robicsek F, Daugherty HK, Cook JW, Selle JG, Stiegel RM. Traumatic tears of the thoracic aorta: improved results using the Bio-Medicus Pump. *Ann Thorac Surg* 1989;**48**:6—9.
34 Safi HJ, Bartoli S, Hess KR, Shenaq SS, Viets JR, Butt GR, et al. Neurologic deficit in patients at high risk with thoracoabdominal aortic aneurysms: the role of cerebral spinal fluid drainage and distal aortic perfusion. *J Vasc Surg* 1994;**20**: 434—43.
35 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—73.
36 Weigang E, Luehr M, Harloff A, Euringer W, Etz CD, Szabó G, et al. Incidence of neurological complications following overstenting of the left subclavian artery. *Eur J Cardiothorac Surg* 2007;**31**:628—36.
37 Riesenman PJ, Farber MA, Mendes RR, Marston WA, Fulton JJ, Keagy BA. Coverage of the left subclavian artery during thoracic endovascular aortic repair. *J Vasc Surg* 2007;**45**:90—4.
38 Cina CS, Safar HA, Lagana A, Arena G, Clase CM. Subclavian carotid transposition and bypass grafting: consecutive cohort study and systematic review. *J Vasc Surg* 2002;**35**:422—9.