Rom J Leg Med [26] 235-240 [2018]
DOI: 10.4323/rjlm.2018.235

Post traumatic rupture of the isthmic aorta in multitrauma patients involved in car accidents: Our initial experience with emergency endovascular repair

Ovidiu Stiru[1,], Rasvan Vasile[1], Roxana Carmen Geana[1], Diana Ioana Sorostinean[1], Platon Pavel[1], Marian Croitoru[1], Lucian Predescu[1], Liana Valeanu[1], Serban Bubenek[1], Vlad Anton Iliescu[1]*

Abstract: Emergency endovascular treatment of traumatic aortic rupture of the isthmus (TARI) after crush accidents is an alternative to open repair (OR). Two patients, both victims of car accidents with multiple trauma and TARI were presented after 48 hours, respectively 10 hours after accident to our institution. Diagnosis was based on computed tomography angiography (CTA) on both cases, initial management and stabilization of the patients were performed in other hospitals and included resuscitation protocols, blood pressure control, as well as treatment of other associated injuries. Both patients had stable rupture of the aortic isthmus with tear associated with aortic isthmic hematoma and left hemothorax. Initial conservative treatment allowed management of the major associated lesions, and after analysing the CTA images the patients were considered suitable for emergency thoracic endovascular aortic repair (TEVAR) and transferred to our hospital. In emergency, both patients had a Valiant Thoracic endograft with the Captivia Delivery System (Medtronic Vascular, Santa Rosa, California) implanted. The postoperative evolution was clinically uneventful, and the patients were discharged from the hospital on the 9th day, respectively 12th day after TEVAR procedure. The purpose of the present study is to report our first two successful cases of emergency TEVAR in patients with multiple trauma and TARI after car accidents and to discuss some important issues related to this approach.
Key Words: Traumatic aortic rupture of the isthmus, thoracic endovascular aortic repair.

INTRODUCTION

TARI is one of the most devastating injuries. The incidence of TARI is unknown, however, estimates of 7500-8000 deaths per year have been reported in USA and Canada [1]. A 10-years meta-analysis published by Smith 50 years ago found that an overall mortality of 84 % was associated with TARI and only 71% of patients reached the emergency department with stable vital signs [2]. An article published by Parmley *et al.* in 1958 reported an 85% prehospital mortality in patients with TARI [3]. The majority of TARI result from automobile crashes and is the second most common cause of death [2]. The UK-based Cooperative Crash Injury Study indicating that blunt aortic trauma accounts for approximately 20% of vehicle-related deaths, death at the site of trauma is approximately 5% [4]. The aorta ruptures as a result of rapid deceleration, most commonly appear in the isthmus portion of the aorta (TARI) at 1 cm from the origin of the left subclavian artery at the insertion of the embryonic ductus arteriosus (duct of Botallo), most injuries at this site are complete transection [5]. A transection of the aorta is defined as a complete disruption of the intima and media layers and a tear is defined as partial disruption of the same two layers, the adventitia remained intact in both cases. Not all trauma victims with aortic injury die as a direct result of the injury to the aorta, in patients who reach the hospital alive, the aortic adventitia and surrounding aortic structures remain intact, preventing acute exsanguination into the thorax. An estimated 1/3 [6] to 2/3 [7] of the persons with TAI actually die of associated causes. A small number of

1) "Prof. Dr. C. C. Iliescu" Emergency Institute for Cardiovascular Diseases, Bucharest, Romania
** Corresponding author: "Prof. Dr. C. C. Iliescu" Emergency Institute for Cardiovascular Diseases, 258 Fundeni Rd., 2nd District, 022328 Bucharest, Romania, Phone/Fax: +400722207286, E-mail: ovidiu_stiru@yahoo.com*

victims survive, they suffer a non-lethal lesion (usually tears) of the aortic isthmus, their condition allows transfer to hospital and definitive treatment. Survival depends on the extent of accompanying injuries and the speed with which an aortic injury can be identified and treated. There are a number of variables difficult to quantify that influence aortic injury during a vehicle impact. This variables that need to be considered include: the history of a force impact magnitude, position of the victim in the car, whether safety systems were employed/ deployed, head-on impact, multiple rib fractures, sternal or thoracic spine fracture; the presence of associated injuries [5]. Despite these multiple impact variables, some researchers have tried to draw a series of conclusions about the link between the impact and the initiation of TARI. Arajarvi *et al.* found that for TARI, there was no proportional difference in the onset of this injury for either belted or unbelted occupants [8]. The effect of air bag systems on the incidence and severity of TARI still remains uncertain. Also the study demonstrated that TARI seemed to be equally frequent if impacts are frontal or lateral [8]. Several theories have been issued to explain the mechanism of TARI. The most plausible theory explaining the mechanism of the isthmic lesion was issued by Coermann and associates and completed by Lundevall. In Coermann theory deceleration is the main factor because the descending aorta remains fixed to the posterior thoracic wall by the intercostal vessels and the ductus arteriosus, while the heart, ascending aorta and arch pushed forward, resulting in a shearing force at the isthmus [9]. In addition to Coermann's theory, Bowen and Keen postulated that a combination of deceleration and direct impact is necessary to rupture the aorta [10, 11]. In Lundenvall's theory, described as “water hammer” effect, the aortic rupture is caused by hydrostatic pressure which rapidly elevates at impact, and the aorta bursts and tears at its weakest points [12]. Therefore these forces can be generated by an auto crash in which the vehicle is going approximately 40 mph, where the deceleration occurs in milliseconds [12]. Concomitant with chest compression the pressures could be sufficient and displacement of the heart, ascending aorta and aortic arch and produce a “water hammer” effect and a shearing force. Crass and later Symbas propose that the rupture of the aorta is due to an ‘osseous pinch’, they state that the rupture of the aorta is due to entrapment of the aorta between fixed bone structures: manubrium, ribs, clavicles, sternum and the vertebral column [13, 14]. Traditional open surgical repair (OR) has been associated with high morbidity and mortality rates. OR was for many decades the gold standard in TARI, the result depends on quick diagnosis, and aggressive management with an immediate surgical repair. Despite the fast reach of a specialized team at the site of trauma and quick transportation to a specialized hospital, fast recognition of a TARI and advances in surgical and reanimation techniques OR for TARI are still associated with significant morbidity and mortality rates with a range from 23.5% to up to 50% [1]. A 20-years meta-analysis published in 1994 re-ported a mortality and paraplegia rate of 32% [15]. TEVAR is a strategy introduced for treatment of chronic aortic diseases and it offers several advantages compared to OR for the management of TARI but little is known about endovascular repair performed in an acute or subacute setting. Several meta-analyses have documented significantly improved outcomes with TEVAR compared to OR [16-18]. We report our initial experience with integration of emergency TEVAR for management of TARI.

CASE SERIES

Case 1

A 20-years-old male driver (1.65 m and 54 kg) who was involved in a collision with another vehicle in January 2018. The impact happened on the side of the driver, who was wearing his seat belt. He was referred as a stable patient to our hospital from an emergency hospital after stabilizing vital functions, 2 days after a car crash. CTA scan of the brain, thorax, abdomen and pelvis, performed at the referring center described 2.8/ 3.8 cm aortic isthmus aneurysm, periaortic isthmus haematoma (Fig. 1), bilateral pulmonary contusion with minimum pleural effusion in the left pleural cavity and bilateral minimum pneumothorax, fractured ribs, liver contusion and right kidney contusion. Also, he was diagnosed with a stable pelvic fracture with minimal displacement. No brain injuries were reported at CT brain scan. The patient does not have a history of pathological disease, and did not require any surgical intervention for the traumatic

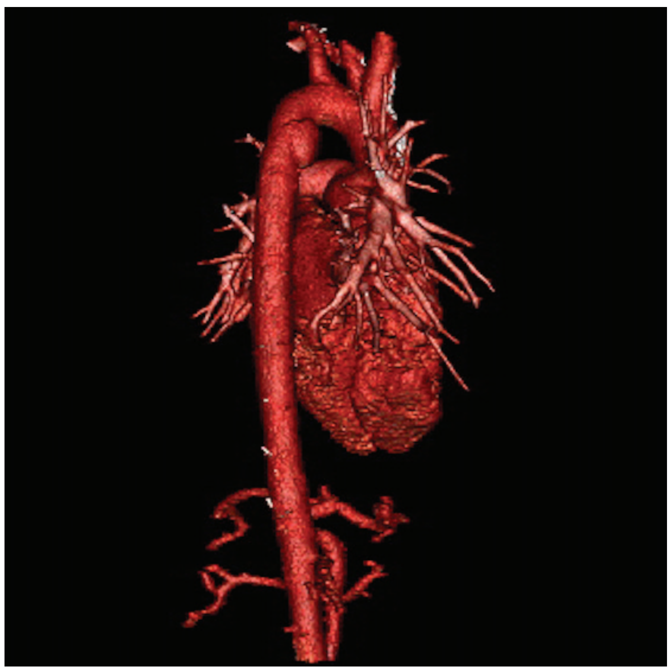

Figure 1. First case: CTA 3D-reconstruction showing the aortic aneurysm at the isthmus level.

lesions above mentioned. On admission, the patient was haemodynamically stable, awake and responsive. He presented pain at the fractured ribs and both ischiopubic ramus as well as left back pain. He was transferred to intensive care unit (ICU) stable. Laboratory test results displayed haemoglobin level of 12.8 g/dL, and a creatinine of 0.89 mg/dL. Transthoracic echocardiography found normal cardiac function without any valvular pathology, without signs of pericardial effusion. Doppler echography of peripheral arteries found a 6.8 mm (left) and 7.2 mm diameter (right) femoral arteries without any associated lesions. Emergency OR was considered to have more risks compared to emergency TEVAR mainly because of the patient's associated internal injuries. However, it was taken into consideration that TEVAR could be compromised by the patient's small diameter of femoral arteries. Preoperative cerebrospinal fluid (CSF) drainage was not performed due to the fact that the isthmus aneurysmal lesion covered by the endograft was up to 40mm and the risk of intercepting the Adamkievici artery was very small. Under general anaesthesia, a surgical access of right femoral artery was performed, which was found to be as expected, a very small and spastic artery. After flushing the artery with papaverine solution, a 8 Fr. sheath was placed in the common femoral artery under direct vision. Systemic heparin (3000 UI) was administered. The contralateral femoral artery was catheterized for angiographic control. We initially performed a control arteriography of the aortic arch, aortic isthmus and descendent thoracic aorta (Fig. 2) which showed the aneurysm located in the isthmus aorta and a 20 mm healthy proximal landing zone enough for a correct positioning of the proximal end of the endograft. Under fluoroscopy a floppy wire was guided into the aortic arch and when it was in the proper position the floppy wire was extracted and exchanged with a soft catheter. A stiffer guide-wire was inserted through this soft catheter [19]. The 8 Fr. sheath was then exchanged with the appropriate 24 Fr. (8mm) device sheath. A stiff guide wire was advanced through the right external iliac artery into the ascending aorta. Under fluoroscopic guidance, a 24mm diameter by 150 mm long thoracic endoprosthesis Valiant Thoracic Endograft with the Captivia Delivery System (Med-tronic Vascular, Santa Rosa, California), was loaded, positioned and deployed in the first portion of the descending thoracic aorta covering the isthmic aneurysm very close to the distal border of origin of the left subclavian artery. The proximal and distal landing zones were expanded with a Reliant stent graft balloon catheter. The complete angiography demonstrated total exclusion of the isthmus descending thoracic aortic aneurysm, correct position on the endograft in relation to the left subclavian artery with no evidence of any endoleaks (Fig. 3). The postoperative evolution was clinically uneventful, and the patient was discharged from the hospital on the 9th day after TEVAR procedure. CTA control at three months after the procedure revealed a good evolution, with no evidence of aneurysm expansion or endoleaks (Fig. 4).

Case 2

A 32-year-old male pedestrian (1.74m and 82kg) who was hit by a vehicle in May 2018. The crash happened on the pedestrian cross the street through a signalling pedestrian crossing. The car hit the pedestrian with the right wing, this being thrown on the hood and then on the sidewalk. The circumstances of the accident are not known and the police unit reports show that there was a

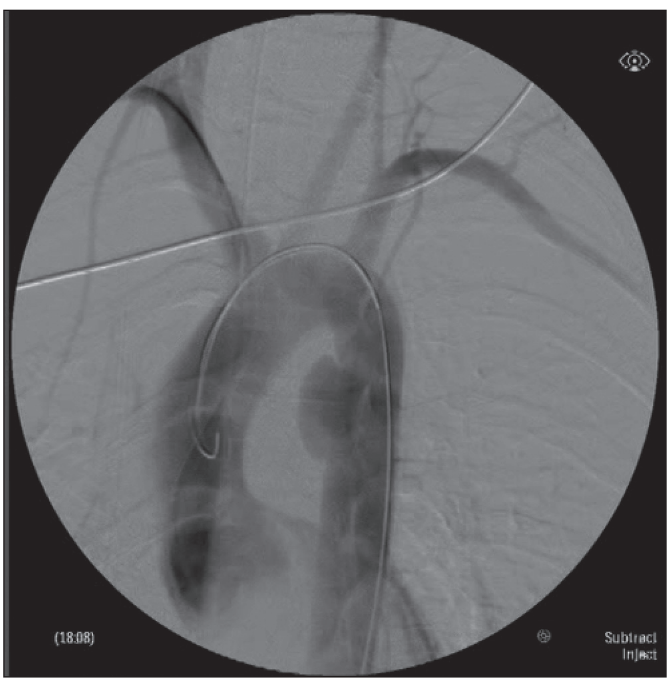

Figure 2. First case: Intraoperative aortography with evidence of the ishmic aortic aneurysm.

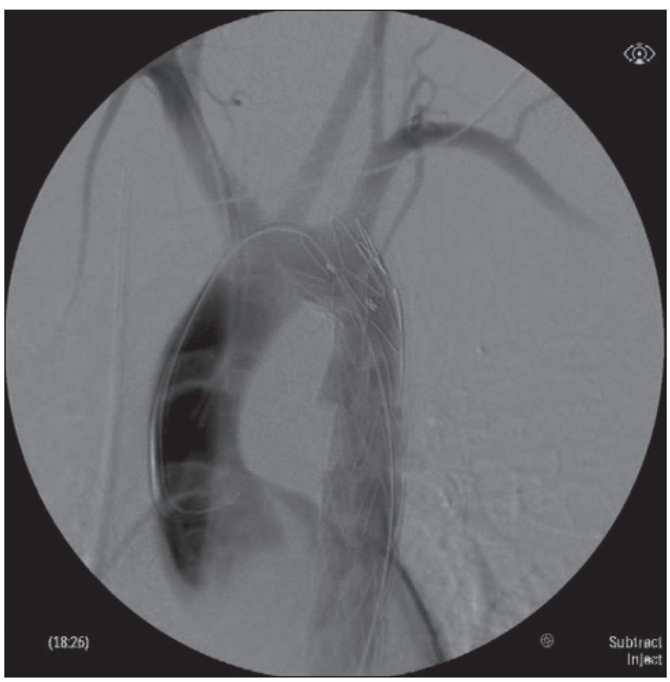

Figure 3. First case: Postoperative arteriography showing correctly positioned stent graft.

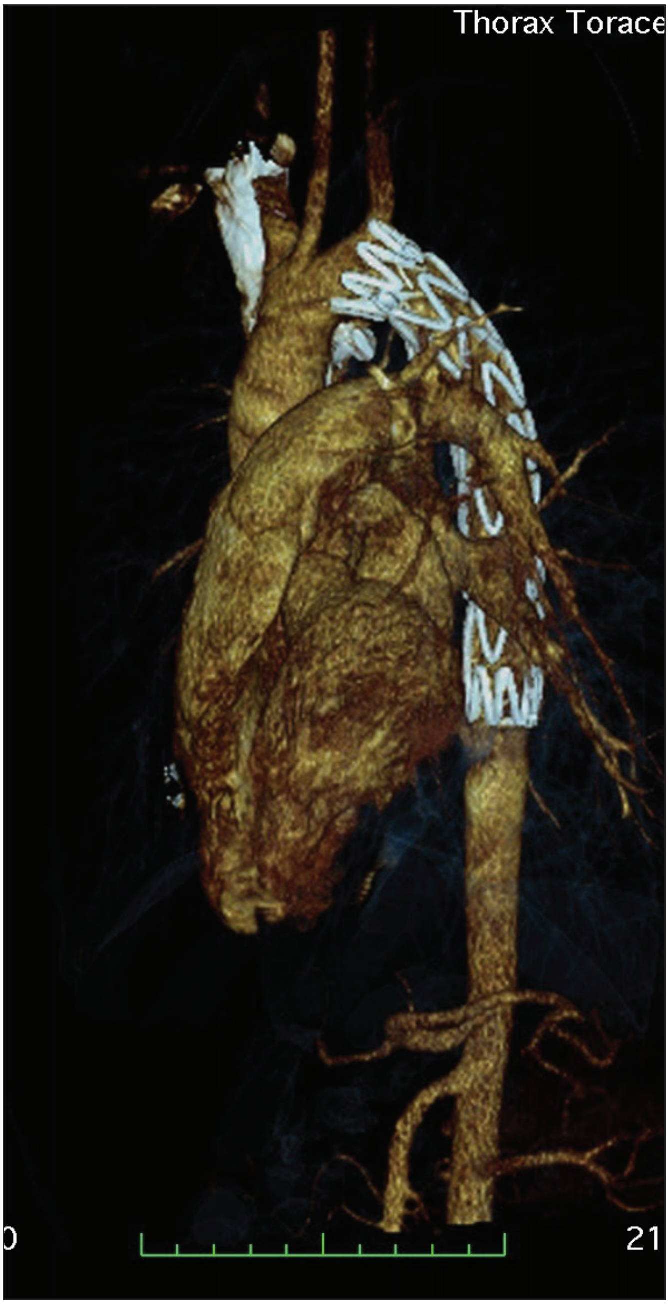

Figure 4. First case: Folow up CTA 3D-reconstruction at 3 months showing correct positionof the stent graft.

Figure 5. Second case: CTA showing dissection and rupture of the aortic isthmus and left pleural effusion.

Figure 6. Second case: Postoperative arteriography showing correctly positioned stent graft.

7m long braking trace before to impact. He received initial treatment at a emergency hospital and he was transferred to our hospital after stabilizing vital functions, at 10 hours after accident. CTA scan of the brain, thorax, abdomen and pelvis performed at the referring center described acute aortic dissection localized at aortic isthmus level, periaortic isthmus haematoma (Fig. 5), right shoulder contusion with acromioclavicular contusion, right pulmonary contusion with 15 mm right pleural effusions, 35 mm left pleural effusion, fractured first and second anterior arch right ribs, right kidney contusion and splenic contusion with two splenic collections of 45/21mm and 39/21mm. Rib fractures were associated with minimal displacement. No brain injuries were reported at CT brain scan. The patient has a history of asthmatic bronchitis, and did not require any surgical intervention for the traumatic lesions mentioned.

On admission, the patient was haemodynamically stable, he presented pain at the fractured ribs, mild dyspnoea and left back pain. Accordingly with our institution's protocol he was transferred to ICU stable. Laboratory test results showed haemoglobin level of 13.9 g/dL, and creatinine of 0.79 mg/dL. Transthoracic echocardiography found normal cardiac function without any valvular pathology, without signs of pericardial effusion. Doppler echography of peripheral arteries found 7.8 mm (left) and 8.2 mm diameter (right) femoral arteries. The patient was considered suitable for emergency TEVAR mainly because of the patient's associated internal injuries. Preoperative cerebrospinal fluid (CSF) drainage was not performed. As in the previous case under general anaesthesia, a surgical access of right femoral artery was performed and a 8 Fr. sheath was placed in the common femoral artery under direct vision. Systemic heparin (5000 UI) was ad-ministered. The contralateral femoral artery was catheterized for angiographic control. We per-formed a control arteriography of the aorta (Fig. 5) which showed the localized dissection on the descending aorta and a small aneurysm of the isthmus. The difficulty of the case was that proximal landing zone was dissected there was a small challenge in establishing future stabilization of the endograft. The technique was the same as in the first case , a 34 mm diameter by 150 mm long thoracic endoprosthesis Valiant Thoracic Endograft with the Captivia Delivery System (Medtronic Vascular, Santa Rosa, California), was loaded, positioned and deployed in the first portion of the descending thoracic aorta covering the isthmic dissected portion very close to the distal border of origin of the left subclavian artery. As in the previous case the proximal and distal landing zones were expanded with a Reliant stent graft balloon catheter. The post-procedural complete angiography demonstrated total exclusion of the isthmus descending thoracic aortic aneurysm, correct position on the endograft in relation to the left subclavian artery with no evidence of any endoleaks (Fig. 6). The postoperative evolution was clinically uneventful, and the patient was discharged from the hospital on the 12^{th} day after TEVAR.

DISCUSSION

Many years the success of the treatment of traumatic rupture of the aortic isthmus depended on how quickly the lesion was diagnosed and how quickly patients were on the table for OR, associated multiple trauma complicating the postoperative results. With the appearance of endoluminal technique, these concepts are changed. Endovascular treatment of TARI is achieved by trans luminal placement of the stent-grafts devices which close the aortic isthmic rupture. The periaortic hematoma has no longer communication with the lumen of the aorta and thus the periaortic thrombus is cure by organized. In both our patients the CTA diagnosed the stable rupture of the aortic isthmus and periaortic isthmic hematoma and minimum left hemothorax with no signs of active bleeding. All of the associated traumatic lesions have been previously stabilized, the patients were hemodynamically stable, did not have any traumatic brain injuries and there were no contra indications for the administration of anticoagulant treatment required in order to perform TEVAR. Nowadays TEVAR is a rapidly evolving therapy and revolutionizing TARI treatment. For TARI, endovascular stent grafting is less invasive, life-saving, and is an ideal alternative to OR. OR unfortunately is associated with severe complications such as death, acute myocardial infarction, stroke, and spinal cord ischaemia and paraplegia during or shortly after the operation.

The theoretical advantages of TEVAR are multiple: First - stent-graft insertion can be performed with mild anticoagulation and the bleeding complications can be avoided. Second - the risk of medullar ischemia and paraplegia is lower than in OR because only a few proximal branches to the spinal cord might be covered by the implanted stent graft [20]. Third - the blood loss is minimal because in TEVAR the technical aspect involves only a simple femoral arteriotomy for the insertion of the delivery system of the stent graft. Furthermore there is no need to perform thoracotomy, the use of cardiopulmonary by-pass nor intra-operative single-lung ventilation. The post procedural recovery of patients is easier compared with OR and periprocedural stroke rates in TEVAR are from 0% to 7% [21], comparative with 8.7% in OR [22]. This approach has a series of limitations including appropriate aortic diameter at the isthmic level with available size endoprosthesis, the characteristics and extent of TARI, adequate proximal and distal landing zones, anatomic limitations of femoral and iliac arteries access and logistic limitation of the stent graft variable sizes to be available for emergency use [23. There still remain a series of unresolved issues related to TEVAR procedure: anatomical variability of the aortic arch, the proximity of the traumatic aortic isthmic rupture to the origin of the left subclavian artery which frequently requires to cover it with the stent-graft, timing of repair-emergency or elective, impact of intra-operative anticoagulation in the setting of cerebral injuries, long-term follow-up and evolving of devices implanted in growing patients, durability of the stent-graft material and the fixation system which are a subject of permanent improvement. However a few technical limitations of device implantation must be emphasized - at the moment the proximal landing zone is under the left subclavian artery and requires a proximal length of 20 mm prior to the aneurysm, and the aortic inner diameter must be between 23 mm and 37 mm [19], since the calibre of available delivery systems are now in a range between 18F to 24F, which can be problematic in the small, spastic, tortuous or atherosclerotic and rigid arteries. In the past, this devices were not available for emergency use because each device had to be customized for each patient according to CT measurements making them unavailable in such cases. Nowadays these devices are available in our institution for emergency cases. We have five sizes available according to the measurements obtained by computed tomography imaging thus covering the dimensions of the aortic isthmus between 24-36 mm. The proximal landing zone was just below the left subclavian artery origin and in both patients the measurements showed a length of more than 20 mm prior to the aneurysm, and the aortic inner diameter was of 23 mm in first patient, respectively 34 mm in the second case. The distal landing zone was far away above the celiac trunk, and the inner diameter for both patients was the same inner diameter as for the proximal landing zone. We evaluated all these parameters on the CTA examination for each patient and we considered they were eligible for the TEVAR procedure. The difficulty of the first presented case was the femoral arteries size which were smaller than the minimum diameter (8 mm) of the necessary endoprosthesis sheat. After local flushing of the femoral artery with antispastic drugs (papaverine) it was possible to advance the device with the thoracic endoprosthesis to the isthmic aneurysm. The difficulty of the second presented case was the unhealthy dissected proximal landing zone, with major risk after TEVAR procedure of retrograde progression of dissection to the aortic arch and to the ascending aorta or even to free wall rupture. Intra-operative angiography in both patients showed successful deployment, permeability of the left subclavian artery and total exclusion of the aneurysm and dissection.

CONCLUSIONS

Emergency endovascular treatment of traumatic aortic rupture of the isthmus after high impact accidents is challenging in patients with multiple trauma. Initial management, resuscitation, treatment of associated injuries and stabilization of the patients must be done at the same time with CTA analysis of the thoracic isthmic aorta and precise preoperative stent-graft planning. Emergency endovascular treatment can be done with very good results in traumatic lesion of the aortic isthmus (acute dissection or acute aneurysm) and can be a strong alternative to open surgery repair.

Conflict of interest. Authors have declared that no competing interests exist.

Ethical approval. All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

References

1. Fabian TC, Richardson JD, Croce MA, Smith JS, Rodman G, Kearney PA, Flynn W, Ney AL, Cone JB, Luchette FA, Wisner DH, Scholten DJ, Beaver BL, Conn AK, Coscia R, Hoyt DB, Morris JA, Harviel JD, Peitzman AB, Bynoe RP, Diamond DL, Wall M, Gates JD, Asensio JA, McCarthy MC, Girotti MJ, VanWijngaarden M, Cogbill TH, Levison MA, Aprahamian C, Sutton JE, Allen CF, Hirsch EF, Nagy K, Bachulis BL, Bales CR, Shapiro MJ, Metzler MH, Conti VR, Baker CC, Bannon MP, Ochsner MG, Thomason MH, Hiatt JR, Omalley K, Obeid FN, Gray P, Bankey PE, Knudson MM, Dyess DL, Enderson BL. Prospective study of blunt aortic injury: multicenter trial of the American Association for the Surgery of Trauma. J Trauma Inj Infect Crit Care 1997; 42(3):374-80.
2. Smith RS, Chang FC.Traumatic rupture of the aorta: still a lethal injury. Am J Surg 1986; 152:660-663.
3. Parmley LF, Mattingly TW, Manion WC, Jahnke EJ. Non-penetrating traumatic injury of the aorta. Circulation 1958;17:1001-1086.
4. 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 Cooperative Crash Injury Study. Eur J Cardiothorac Surg 2003;23:143-148.
5. Williams JS, Graff JA, Uku JM, Steinig JP. Steinig Aortic Injury in Vehicular Trauma. Ann Thorac Surg 1994; 57:726-730.
6. Bodily K, Perry JF, Strate RG, Fischer RP. The salvageability of patients with post-traumatic rupture of the descending thoracic aorta in a primary trauma center. J Trauma 1977;17:754-760.
7. Greendyke RM. Traumatic rupture of aorta. Special reference to automobile accidents. JAMA 1966;195:119-22.
8. Arajarvi E, Santavirta S, Tolonen J. Aortic ruptures in seat belt wearers. J Thorac Cardiovasc Surg 1989;98:355-361.
9. Coermann R, Dotzauer G, Lange W, Voigt GE. The effects of the design of the steering assembly and the instrument panel on injuries (especially aortic rupture) sustained by car drivers in head-on collision. J Trauma 1972;12:715-724.
10. Bowen DAL, Teare RD. Delayed traumatic rupture of aorta. Thorax 1962;1715&3.
11. Keen G. Closed injuries of the thoracic aorta. Ann R Coll Surg Engl 1972;51:137-156.
12. Lundevall J. The mechanism of traumatic rupture of the aorta. Acta Pathol Microbiol Scand 1964;62:34-46.
13. Crass JR, Cohen AM, Motta AO, Tomashefski JF, Wiesen EJ. A proposed new mechanism of traumatic aortic rupture: the osseous pinch. Radiology 1990; 176:645-649.
14. Symbas PN. Fundamentals of clinical cardiology 96 Great vessels injury. Am Heart J 1977;93:518-522.
15. Kouchoukos NT, Dougenis D. Surgery of the thoracic aorta. N Engl J Med. 1997;336:1876-1888.
16. Akins CW, Buckley MJ, Dagget W. Acute traumatic disruption of the thoracic aorta: a ten year experience. Ann Thorac Surg. 1981;31:305-9.
17. 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; 8:30-23.
18. Pate JW, Fabian TC, Walker W. Traumatic rupture of the aortic isthmus: an emergency? World J Surg. 1995;19:119-126.
19. Ovidiu Stiru, Roxana Carmen Geana, Platon Pavel, Marian Croitoru, Cristian Boros, Ion Iovu, Vlad Anton Iliescu Descending thoracic aortic aneurysm rupture treated with thoracic endovascular aortic repair in a patient with peripheral artery disease The Heart Surgery 2018; 2017-1895 21.
20. Kato N, Dake MD, Miller DC, Semba CP, Mitchell RS, Razavi MK, Kee ST. Traumatic thoracic aortic aneurysm: treatment with endovascular stent-grafts. Radiology. 1997; 205:657-662.
21. Botsios S, Frömke J, Walterbusch G, Schuermann K, Reinstadler J, Dohmen G. Endovascular treatment for nontraumatic rupture of the descending thoracic aorta: long-term results. J Card Surg. 2014; 29:353-358.
22. Jonker FH, Verhagen HJ, Lin PH, Heijmen RH, Trimarchi S, Lee WA, Moll FL, Atamneh H, Rampoldi V, Muhs BE. Open surgery versus endovascular repair of ruptured thoracic aortic aneurysms. J Vasc Surg. 2011;53(5):1210-1216.
23. Conrad MF, Ergul EA, Patel VI, Paruchuri V, Kwolek CJ, Cambria RP. Management of diseases of the descending thoracic aorta in the endovascular era: a medicare population study. Ann Surg. 2010;252(4):603-610.