Surgical conversion for intragraft thrombosis following endovascular repair of traumatic aortic injury

Enrico Maria Marone, MD, Andrea Kahlberg, MD, Yamume Tshomba, MD, Davide Logaldo, MD, and **Roberto Chiesa, MD,** *Milan, Italy*

We report the case of a 32-year-old man with severe polytrauma, submitted to urgent endovascular exclusion of a posttraumatic thoracic aortic pseudoaneurysm. Two years later, computed tomography scan showed asymptomatic mural atherothrombosis of the aortic stent graft in its middle-distal portion, and the patient was placed on oral anticoagulants. As subsequent computed tomography scan showed progression of the thrombosis, the patient underwent surgical conversion, with stent graft explantation and in situ aortic replacement. Gross examination revealed mural organized thrombosis and a significant infolding of the distal end of the stent graft. (J Vasc Surg 2012;55:538-41.)

Thoracic endovascular aortic repair (TEVAR) for blunt traumatic aortic injuries (BTAIs) has been considered in the last decade a promising alternative to open surgical treatment, with lower perioperative mortality and neurologic morbidity.[1,2] Even if specific stent grafts designed for the treatment of BTAIs are under investigation, and availability can be anticipated in the near future, currently commercially available devices are suboptimal for use in young trauma victims, and both early and late graft-related complications have been described.[3]

We report the case of a patient who developed late thrombosis after TEVAR for BTAI and was offered surgical conversion 30 months after the primary procedure.

CASE REPORT

A 32-year-old man sustained a violent injury during a high-speed motor vehicle collision, resulting in polytrauma and hemorrhagic shock. At arrival, first evaluation demonstrated multiple bone fractures, right pneumothorax, and hemoperitoneum. After initial resuscitation, the patient underwent primary repair of multiple liver lacerations and splenectomy. Due to disseminated intravascular coagulopathy (platelet count: 26 $\times$ 10^9/L), perihepatic packing was performed. A computed tomography angiography (CTA) of the chest showed a posttraumatic 2.5-cm pseudoaneurysm of the aortic isthmus with concomitant hemomediastinum (Fig 1, *A*). Of note, the left common carotid artery originated from the brachiocephalic trunk. Measured aortic diameters were 21 mm proximal to the lesion and 19 mm in the descending tract. The patient was transferred again to the operating room, where an 8 Fr sheath was inserted into the right common femoral artery surgically exposed. An aortogram revealed an aortic pseudoaneurysm on the lesser curve of the proximal descending thoracic aorta (DTA), 12-mm distal to the left subclavian artery (LSA). A 24-mm $\times$ 104-mm Relay Thoracic stent graft (Bolton Medical Inc, Sunrise, Fla) was deployed in the DTA, with the uncovered proximal stent across the origin of the LSA, obtaining complete exclusion of the pseudoaneurysm. Two days later, coagulopathy resolved, and perihepatic packing was removed. CTA performed on postoperative day (POD) 9 demonstrated regular patency of the aortic stent graft and pseudoaneurysm exclusion (Fig 1, *B*). No evidence of malapposition or infolding of the the stent graft was noted at completion angiography nor at postprocedural CTA. The latter discovered an asymptomatic thrombosis of the left internal jugular, treated with low-molecular-weight heparin. Standard thrombophilic screening revealed no abnormalities. The patient was discharged on POD 29 to an orthopedic rehabilitation hospital. After 2 months, since duplex scan showed resolution of the left jugular vein thrombosis, heparin was stopped and replaced by acetylsalicylic acid (100 mg daily), indefinitely.

Six- and 12-month CTAs showed regular patency of the thoracic stent graft, without signs of mural atherothrombosis. Twenty-four-month CTA demonstrated intragraft atherothrombosis, predominantly involving the middle and distal third of the aortic stent graft (Fig 2, *A*), without evidence of endoleak, migration, collapse, or structural device alterations. Clinical examination and blood sampling did not reveal any sign of systemic inflammation or sepsis, and a more accurate thrombophilic screening excluded previously unidentified coagulopathy. The patient was placed on anticoagulant therapy with warfarin, with a target International Normalized Ratio range of 2.5 to 3.5. He remained asymptomatic, and a CTA performed after 6 months (Fig 2, *B* and *C*) showed a significant increase of mural atherothrombosis, also evolving into multiple intraluminal septa within the aortic stent graft. Thus, the patient was readmitted to undergo thoracic surgical conversion. The distal aortic arch and the DTA were exposed through a left posterolateral thoracotomy at the fourth intercostal space. Spinal

From the Scientific Institute H. San Raffaele, "Vita-Salute" University School of Medicine.

Competition of interest: none.

Reprint requests: Andrea Kahlberg, Chair of Vascular Surgery, Scientific Institute H. San Raffaele, "Vita-Salute" University School of Medicine, Via Olgettina 60, 20132, Milan, Italy (e-mail: kahlberg.andrea@hsr.it).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a competition of interest.

0741-5214/$36.00

doi:10.1016/j.jvs.2011.07.075

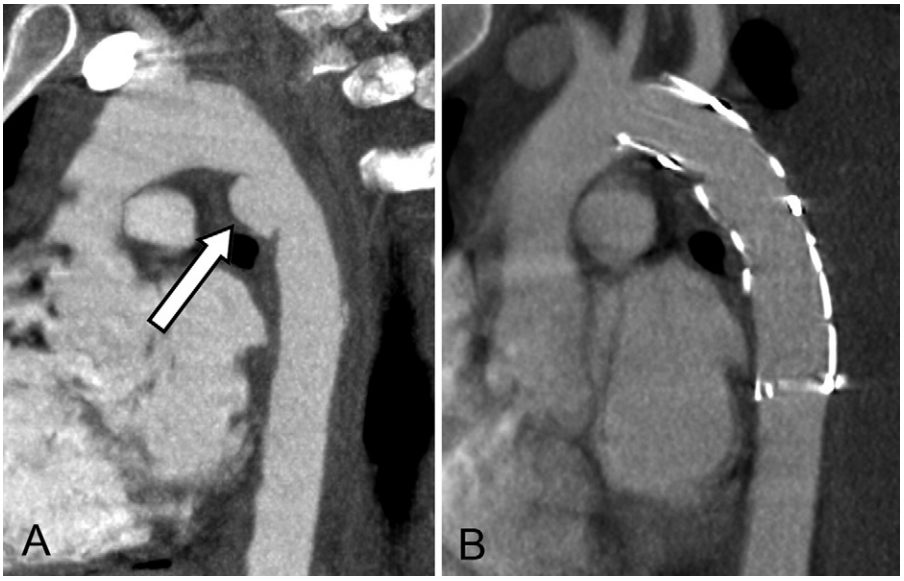

Fig 1. A, Computed tomography angiography (CTA) showing an acute posttraumatic pseudoaneurysm (*arrow*) of the thoracic aorta at isthmus. **B**, Complete exclusion of the pseudoaneurysm by a 24-mm × 104-mm Relay Thoracic stent graft, with bare-metal overstenting of the left subclavian artery (LSA).

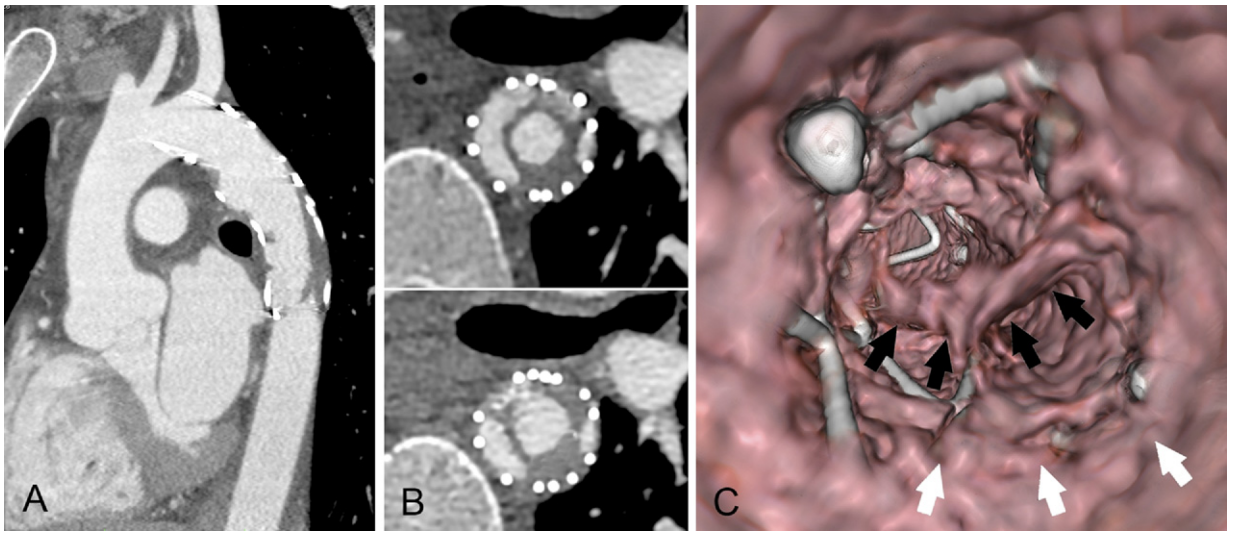

Fig 2. A, Twenty-four-month computed tomography angiography (CTA) showing mural thrombosis within the middle-distal portion of the stent graft. **B**, At 30 months, the amount of intragraft thrombus is increased, with appearance of endoluminal septa. **C**, Virtual angioscopy of the intragraft lumen confirming the presence of mural thrombus (*white arrows*) and intraluminal septa (*black arrows*).

cord fluid drainage was instituted preoperatively, and left heart bypass (Biomedicus pump; Medtronic, Inc, Minneapolis, Minn) was used during aortic cross-clamping. The proximal aortic clamp was placed between the brachiocephalic trunk and the LSA (Fig 3, *A*). The DTA was then opened, and the stent graft was easily manually removed. No tight incorporation of the proximal stent into the aortic wall was noted during this maneuver. Aortic reconstruction was performed (Fig 3, *B*) with a tubular 18-mm Dacron graft (Hemashield, Boston Scientific, Boston, Mass). The presence of mural organized thrombus and multiple intimal septa within the middle and distal thirds of the stent graft was found at gross examination. Histopathologic analysis demonstrated a fibrous tissue separated by a number of neovessels, with focal lympho-granulocyte infiltration, consistent with human organized thrombus. Also, infolding of the distal end of the stent graft was noted (Fig 3, *C*). The postoperative course was uneventful, and the patient was discharged on POD 10. CTA performed at 1 month showed regular patency of the aortic graft. The patient is well at 3-month follow-up.

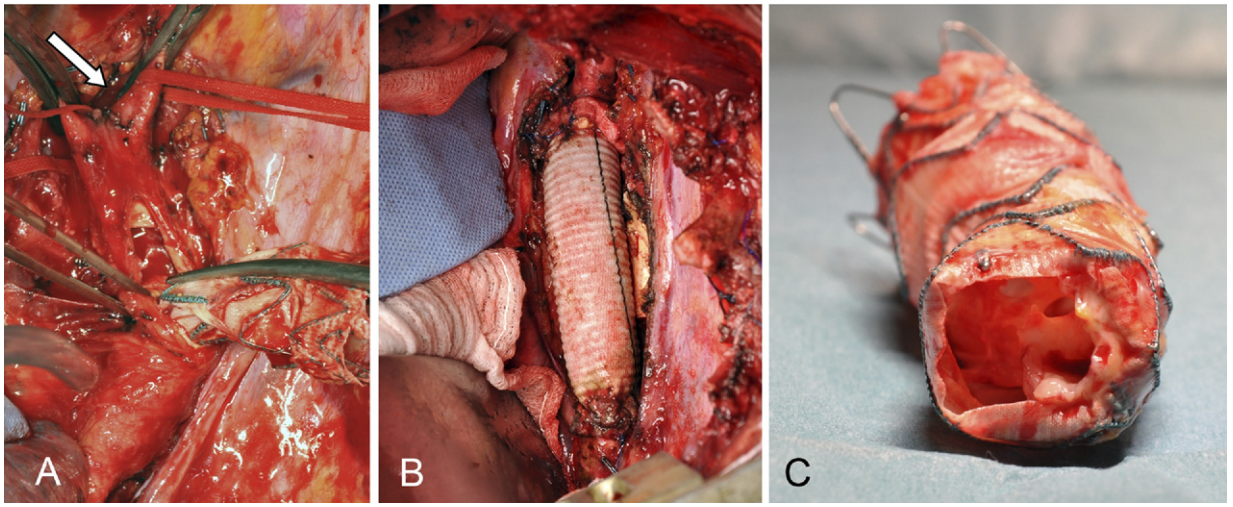

Fig 3. A, Intraoperative photograph during open conversion, detailing aortic cross-clamping proximal to the origin of the left subclavian artery (LSA) (*arrow*) and explantation of the endovascular graft. **B**, Reconstruction of the descending aorta with an 18-mm tubular Dacron graft. **C**, Photograph of the explanted stent graft, demonstrating significant intragraft thrombosis associated with infolding of the distal end of the device.

DISCUSSION

Since TEVAR has become the most used technique to treat acute BTAIs, several case studies have reported late device-related complications.[4,5] Perhaps the most severe stent graft collapse has been proposed to be related to strict aortic arch angulation, small distal aortic diameter, and minimum intragraft aortic diameter.[6]

As far as we know, only the Vall d'Hebron Hospital group of Barcelona has previously described intragraft mural thrombus formation following TEVAR for BTAI. In 2009, they reported the case of a 17-year-old patient treated with a Zenith TX2 stent graft, who developed a nearly occlusive intragraft thrombosis 1 year after TEVAR.[7] Of note, the stent graft was intentionally oversized 30% compared with the measured aortic diameter. Emergency axillofemoral bypass was performed due to acute heart failure, followed by definitive repair consisting of an extra-anatomic bypass arising from the ascending aorta.

In 2010,[3] the same group reported the results of their entire series of TEVAR for BTAI (mean follow-up, 43 months). They used a mean proximal stent graft oversizing of 23.2% (range, 12%-40%). Asymptomatic parietal thrombosis was found at CTA in seven of 20 patients (35%), but no major complications developed at subsequent follow-up.

Our case presents some similar features to that described by Alvarez et al.[7] In both, structural alterations of the device were absent, and the thrombosis was partial, appeared after a period of regular stent graft patency, and involved mainly the distal portion of the stent graft.

Although we could not really identify a cause for this complication, we excluded both the presence of structural device defects and diseases predisposing to thrombosis such as sepsis or thrombophilia. Based on CTA and intraoperative findings, we can speculate that stent graft oversizing in its distal portion (24 vs 19 mm = 26% oversizing) has led to infolding of the stent graft fabric, with reduction of the intragraft lumen diameter and increase of blood flow turbulence, finally contributing to thrombus apposition. We strongly believe that stent graft oversizing should always remain within the recommended range (usually 10%-20%) also in case of BTAI treatment, and that oversizing calculation should also consider the distal landing zone. In this respect, custom-made and tapered devices may be used, if available in a timely fashion.

Even in retrospect, we could not find any initial sign of stent graft distal malapposition or infolding in both procedural imaging and postoperative CTAs, suggesting that these techniques may be not accurate in detecting subtle fabric infolding. Since intravascular ultrasound has been described as a useful and accurate diagnostic tool in case of TEVAR for BTAI,[8] it may have helped us to detect the distal graft infolding during follow-up, perhaps allowing early endovascular reintervention.

The best therapeutic strategy in case of asymptomatic partial thrombosis of a thoracic stent graft is still unclear, due to scant evidence derived from the literature. Conservative management, including anticoagulant therapy and observation, has been reported as the first-line treatment to prevent clot progression or mobilization in case of partial thrombosis of nonaneurysmatic descending thoracic aortas.[9] However, in our experience, this approach was not effective after 6 months. Endovascular relining may be considered as a less invasive option compared with surgical conversion,[10] but the risk of clot mobilization during crossing of guidewires and balloon dilatations might be expected. Moreover, we should wonder whether endovascu-

lar approaches are to be intended only as a "bridge" to a later open conversion in many young multitrauma patients.

Surgical conversion is probably the most definitive option in case of thoracic stent graft thrombosis. Especially in young healthy patients, we prefer a direct approach to the DTA via left thoracotomy, allowing stent graft explantation and aortic in situ reconstruction.[11] All adjunctive strategies to prevent visceral and spinal cord ischemia should be used. The extra-anatomic approach, as previously described by some authors,[7,12] is a viable option that may reduce the risks associated with total aortic cross-clamping, but it leaves the potential for progression of the thrombosis, distal embolization, stent graft infection, and fistulization.[13]

REFERENCES

1. Tang GL, Tehrani HY, Usman A, Katariya K, Otero C, Perez E, et al. Reduced mortality, paraplegia, and stroke with stent graft repair of blunt aortic transections: a modern meta-analysis. J Vasc Surg 2008;47: 671-5.
2. Xenos ES, Abedi NN, Davenport DL, Minion DJ, Hamdallah O, Sorial EE, et al. Meta-analysis of endovascular vs open repair for traumatic descending thoracic aortic rupture. J Vasc Surg 2008;48:1343-51.
3. Fernandez V, Mestres G, Maeso J, Domínguez JM, Aloy MC, Matas M. Endovascular treatment of traumatic thoracic aortic injuries: short- and medium-term Follow-up. Ann Vasc Surg 2010;24:160-6.
4. Idu MM, Reekers JA, Balm R, Ponsen KJ, de Mol BA, Legemate DA. Collapse of a stent-graft following treatment of a traumatic thoracic aortic rupture. J Endovasc Ther 2005;12:503-7.
5. Lazar HL, Varma PK, Shapira OM, Soto J, Shaw P. Endograft collapse after thoracic stent-graft repair for traumatic rupture. Ann Thorac Surg 2009;87:1582-3.
6. Muhs BE, Balm R, White GH, Verhagen HJ. Anatomic factors associated with acute endograft collapse after Gore TAG treatment of thoracic aortic dissection or traumatic rupture. J Vasc Surg 2007;45:655-61.
7. Alvarez B, Constenla I, Maeso J, Matas M. Late thrombosis of a thoracic aorta stent graft: therapeutic management. J Vasc Surg 2009;49:774-7.
8. Azizzadeh A, Valdes J, Miller CC 3rd, Nguyen LL, Estrera AL, Charlton-Ouw K, et al. The utility of intravascular ultrasound compared to angiography in the diagnosis of blunt traumatic aortic injury. J Vasc Surg 2011;53:608-14.
9. Tsilimparis N, Hanack U, Pisimisis G, Yousefi S, Wintzer C, Rückert RI. Thrombus in the non-aneurysmal, non-atherosclerotic descending thoracic aorta–an unusual source of arterial embolism. Eur J Vasc Endovasc Surg 2011;41:450-7.
10. Fueglistaler P, Wolff T, Guerke L, Stierli P, Eugster T. Endovascular stent graft for symptomatic mobile thrombus of the thoracic aorta. J Vasc Surg 2005;42:781-3.
11. Langer S, Mommertz G, Koeppel TA, Schurink GW, Autschbach R, Jacobs MJ. Surgical correction of failed thoracic endovascular aortic repair. J Vasc Surg 2008;47:1195-202.
12. Almeida de Oliveira S, Lisboa LA, Dallan LA, Abreu F CA, Rochitte CE, de Souza JM. Extraanatomic aortic bypass for repair of aortic arch coarctation via sternotomy: midterm clinical and magnetic resonance imaging results. Ann Thorac Surg 2003;76:1962-6.
13. Chiesa R, Melissano G, Marone EM, Marrocco-Trischitta MM, Kahlberg A. Aorto-oesophageal and aortobronchial fistulae following thoracic endovascular aortic repair: a national survey. Eur J Vasc Endovasc Surg 2010;39:273-9.

Submitted May 5, 2011; accepted July 15, 2011.