

# Endograft collapse following endovascular repair of traumatic aortic injury

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## ABSTRACT

The advent of endovascular treatment of traumatic thoracic aortic injuries offers a valuable, minimally invasive alternative to open surgical repair. However, there are limitations of the current endovascular stent graft technology for this group of patients. After endovascular repair meticulous follow-up is required with a high index of suspicion for potential complications including the lethal complication of endograft collapse.

**Key words:** • traumatic aortic injury • endovascular repair • endograft collapse

**T**raumatic thoracic aortic injury is a life threatening surgical emergency. These patients commonly have other accompanying severe injuries including head and neck injuries, blunt chest and abdominal injuries and multiple bone fractures. These injuries may delay surgery and significantly increase the morbidity and mortality associated with open thoracic aortic repair. The advent of endovascular treatment of traumatic aortic injury offers a valuable, minimally invasive alternative to surgical repair. Since the first reports of successful endovascular treatment in 1997 (1), several small series have been published with encouraging results (2–6).

We present a case of a fatal complication after endovascular repair of a traumatic aortic injury, resulting from collapse of a Zenith TX2 Thoracic TAA Endovascular Graft (Cook Inc., Bloomington, Indiana, USA) causing severe visceral and lower limb ischemia. To our knowledge, this complication with this particular type of device has not been described in the literature previously.

## Case report

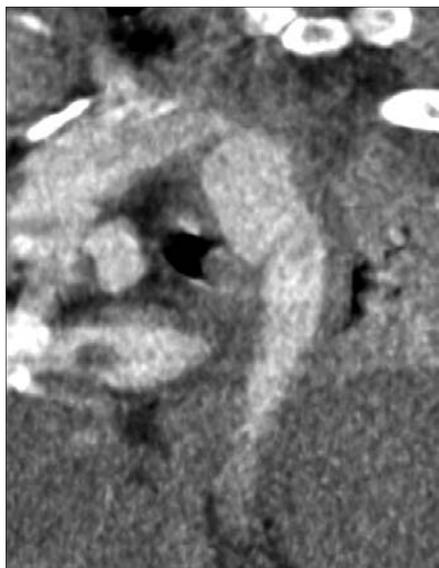
A 31-year-old male pedestrian was struck by a motor vehicle traveling at high speed and was thrown fifty feet away. His companion was killed at the scene. He sustained multiple rib and scapular fractures, left pneumothorax, left diaphragmatic rupture, liver and renal lacerations with hemoperitoneum, pelvic and compound left tibia and fibular fractures and a traumatic thoracic aortic injury. Computed tomographic angiography (CTA) demonstrated aortic tear with pseudoaneurysm and small surrounding hematoma (Fig. 1) located at the isthmus, just distal to the origin of the left subclavian artery. Right vertebral artery was patent with an intact circle of Willis.

He was hemodynamically unstable with respiratory distress and proceeded to the operating room for laparotomy and repair of the diaphragm. Immediately following stabilization, endovascular stent grafting was performed in the operating room during the same session. The transaxial aortic diameters at the proximal and distal landing zones were measured on the CTA at 22 mm and 20 mm, respectively. A 26 mm diameter by 134 mm length Zenith TX2 (oversizing by 18% with respect to the proximal landing zone) endograft was chosen and inserted via a right femoral artery cutdown. Angiograms were performed using a pigtail catheter inserted percutaneously through the left common femoral artery. The endograft was deployed with the proximal end of the graft just distal to the left carotid artery origin, intentionally covering the left subclavian artery to provide adequate proximal landing zone. The endograft was moulded and fully expanded at the proximal and distal landing zones using a Reliant balloon (Medtronic Inc, Minneapolis, Minnesota, USA). Angiography demonstrated complete exclusion of the

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**Figure 1.** Pre-interventional sagittal reformatted CT image showing aortic injury and pseudoaneurysm at the aortic isthmus.



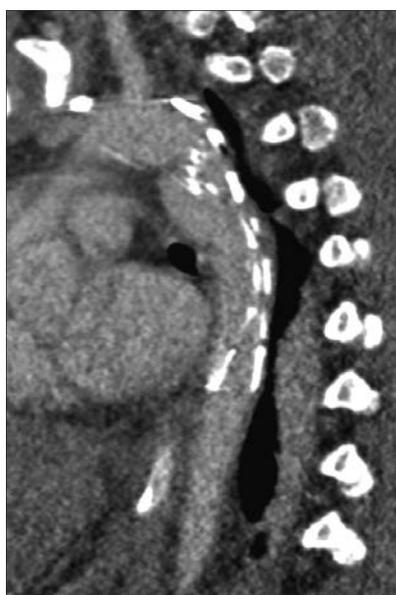
**Figure 3.** Day 5 post-procedural axial CT angiogram demonstrating collapsed endograft.

pseudoaneurysm but the proximal part of the graft was sitting “proud” and not in full contact with the aortic wall along the lesser curvature of the aortic arch. Repeat balloon dilatation was performed but on completion there was suboptimal device apposition at the proximal landing zone (Fig. 2).

The following day he returned to the operating room for open reduction and internal fixation of pelvic fractures, left below knee amputation and right lower leg fasciotomy. CTA was not performed in the early postoperative period due to impaired renal function presumed to be secondary to the renal



**Figure 2.** Angiogram following deployment and post-dilatation of Zenith TX2 endograft demonstrating complete exclusion of the pseudoaneurysm; however, the proximal end of the graft is not in full contact with the aortic wall along the lesser curvature of the aortic arch.



**Figure 4.** Sagittal reformatted CT angiogram showing the collapsed endograft with reperfusion of the pseudoaneurysm. Distal part of the endograft remains expanded, thereby causing aortic obstruction.

injury and hypovolemic insult. He was making satisfactory progress but on the fifth postoperative day following the endovascular procedure he developed increasing lactic acidosis and became markedly septic. CT scan performed at this stage demonstrated that the endograft had collapsed proximally

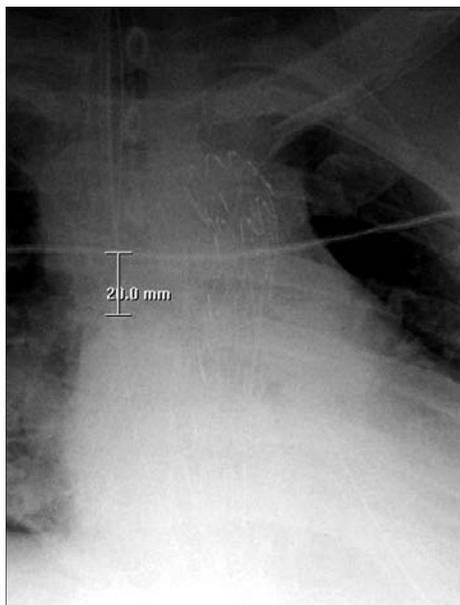
(Fig. 3) with the pseudoaneurysm being reperfused. A small portion of the distal end of the endograft remained fully expanded (Fig. 4). Although contrast was noted within the lumen of the graft and distally within the abdominal aorta, the collapsed endograft was causing a significant obstruction to flow. The patient developed severe hepatic and renal failure and bowel and lower limb ischemia. He rapidly deteriorated and died before any aortic intervention could be performed.

In retrospect, gradual collapse of the proximal end of the endograft was evident on daily frontal chest radiographs (Figs. 5 and 6) beginning two days prior to the CT scan. This finding was, unfortunately, overlooked and an opportunity to address this complication earlier missed.

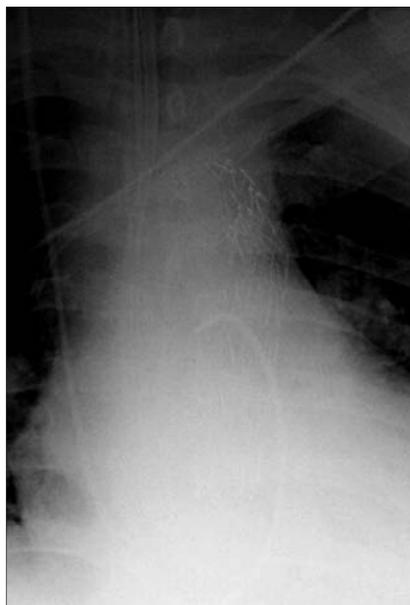
### Discussion

Emergency surgical repair of traumatic thoracic aortic injury is associated with a high morbidity and mortality. In a systematic review of the literature, the hospital mortality following open repair ranged from 8% to 15% depending on type of surgical technique and the paraplegia rate was up to 7% (7). Thirty-day mortality rates of 15% to 30% and paraplegia rates of 2% to 20% have been reported following surgical treatment of traumatic thoracic aortic ruptures (3). Patients with traumatic aortic injuries often have significant pulmonary contusions and thoracotomy increases the risk of respiratory complications. Open surgical repair requires high dose systemic heparinization of a multitrauma patient and therefore is associated with increased risk of hemorrhagic complications, including intracranial bleeds. If the aortic rupture is contained, the presence of other life-threatening conditions may result in the aortic repair being postponed until the patient is in a more stable condition, but 4% of patients die from a ruptured aorta whilst awaiting surgery usually within one week of the trauma (2).

Endovascular treatment is minimally invasive with low operative mortality (6%) and paraplegia (3). It can be per-



**Figure 5.** Day 1 post-procedural chest radiograph showing a fully expanded proximal endograft.



**Figure 6.** Day 3 post-procedural chest radiograph showing a partially collapsed proximal endograft.

formed immediately after treatment of other acute life threatening conditions (intracranial or intra-abdominal hemorrhage) during the same operating session. Thoracotomy, aortic cross clamping and systemic heparinization are not required and the procedure time is considerably shorter with less blood loss. Therefore, endovascular treatment of traumatic thoracic aortic injuries is becoming established as the preferable alternative to open surgical repair in these severely ill patients (3, 6, 8, 9). Although promising short term data have been published, the durability of the endovascular technique for the treatment of traumatic aortic injuries is currently unknown and long term follow-up data is required. Most of the published data is from small case series with limited follow-up. Melnitchouk et al. (9) reported 15 patients with traumatic aortic injuries treated with endograft. Only one device related event (type III endoleak) occurred over a mean follow-up of 32 months.

Melissano et al. (10) reported a patient with distal aortic arch aneurysm who required surgical conversion at two weeks because of total collapse of an EndoMed EndoFit stent graft with fracture of three stents. Idu et al. (11) described a case of asymptomatic Gore TAG endograft collapse noted on routine follow-up CTA at 3 months, which was successfully treated with

a Medtronic Talent proximal extension. Two further cases of TAG endograft collapse have been reported, one occurring within 24 hours and the other presenting with chest pain at 2 months. These were also successfully repaired using TAG proximal extension and Palmaz stent respectively (12). No reports of this complication have been described in the literature with the use of the Zenith TX2 device.

Collapse of an endograft can be a fatal complication as demonstrated in our case. It causes repressurization of the traumatic pseudoaneurysm, which may lead to total aortic rupture. A partially collapsed prosthesis can cause high thoracic aortic obstruction with lethal complications of visceral and lower limb ischemia.

Patients presenting with traumatic aortic injuries are often young with non-atherosclerotic aortas. The configuration of the aortas is often different in these patients than those with aneurysms. They usually have smaller diameter vessels and considerably smaller radius of the lesser curvature of the aortic arch. Currently available endografts are designed to treat atherosclerotic aneurysmal disease, and may be less suitable for the treatment of traumatic aortic injury due to both the need for excessive oversizing and limited conformability and longitudinal flexibility of the grafts. The three reported TAG

collapsed endografts were oversized by 12%, 22% and 37% (11, 12). Excessive oversizing, especially if by greater than 25%, may cause wrinkling of the graft and make it susceptible to collapse. The smallest diameter Zenith endograft available is 26 mm and was used in our patient. Therefore, it was not excessively oversized in our case (18%). In a retrospective review of 50 thoracic aortograms with aortic injury, Borsa et al. (13) noted that the mean aortic diameter adjacent to the injury was 19.3 mm. Therefore, smaller diameter endografts are required to treat this group of patients. Due to the limited flexibility and inability of the graft to adequately conform to the configuration of the aortic arch, there may be inadequate device apposition to the aortic wall at the proximal landing zone even when the left subclavian artery is covered and the graft fully expanded using a balloon. The arterial pressure and pulsations may then push the endograft away from the aortic wall and make it collapse. Although oversizing may have played a role in some of the other cases, the lack of adequate proximal attachment along the lesser curve is likely to have been the main contributing factor to the endograft collapse in our case and the previously reported cases. All these cases highlight the importance of the proximal landing zone.

Meticulous follow-up imaging should be performed, particularly if there has been marked oversizing or if the proximal edge of the endograft is not in full contact with the aortic wall on the completion angiogram. Day 1 post-procedural CTA and daily chest radiographs (including lateral and oblique projections) should be performed and carefully scrutinized by an interventional radiologist for this potentially lethal complication. If there is any evidence of stent collapse the patient should be urgently treated with ballooning followed by further stenting to provide buttressing and better fixation with either a proximal extension or Palmaz stent.

In conclusion, given the shortcomings of the current endovascular stent graft technology, when endovascular therapy is employed in the treatment of thoracic aortic injury, meticulous follow-up is required with a high index of suspicion for potential complications. Problems such as endograft collapse should be detected early and treated appropriately and rapidly.

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