Type B aortic dissection after endovascular abdominal aortic aneurysm repair causing endograft collapse and severe malperfusion

Vikram Iyer, MD,[a] **Mark Rigby, MD,**[b] and **George Vrabec Sr, MD,**[a]
New Westminster, British Columbia, Canada

We report a unique case of type B aortic dissection occurring 11 weeks after endovascular repair of an abdominal aortic aneurysm. This resulted in severe organ and limb malperfusion with collapse and occlusion of the endograft. Successful endovascular salvage is described along with a brief review of the literature. (J Vasc Surg 2009;50:413-6.)

Acute aortic dissection is rarely seen in the patient with a previously repaired abdominal aortic aneurysm (AAA). There are few reports of this in the literature after open aneurysmectomy,[1,2] and to our knowledge, only one report after endovascular AAA repair (EVAR).[3] Retrograde dissection was thought to be the cause in this latter case, and this has also been reported after angioplasty or stenting of the thoracic and abdominal aortas, renal arteries, and iliac arteries.[4-9]

We report a patient with acute Stanford type B aortic dissection 11 weeks after EVAR that resulted in limb and visceral malperfusion. This was treated successfully with endovascular techniques. To our knowledge, this is the only reported case of its kind.

CASE REPORT

An active 77-year-old man was treated with EVAR for a 5.6-cm infrarenal AAA at our institution. He had a medical history of coronary artery disease and asthma. His preoperative renal function was normal. The infrarenal neck was 25 mm long, measuring 22 mm proximally and 25 mm distally; however, it was irregular, with a 18-mm midportion. There was mild calcification at the level of the renal arteries and no significant angulation. Moderate noncircumferential thrombus was present within the neck. There was no abnormality in the thoracic aorta.

A Zenith Flex AAA bifurcated graft (Cook Medical Inc, Bloomington, IN) was used to treat the AAA through femoral artery cutdowns. The main body proximal diameter was 28 mm. After cannulation of the main body, the pigtail test was performed. The pigtail catheter was then straightened using a soft glidewire within the endograft. The catheter was advanced to the aortic arch, after which the wire was exchanged to a stiff wire. No intraoperative endoleaks were demonstrated, and both renal arteries were patent. A 2-week postoperative duplex ultrasound examination did not document any endoleaks. The patient's postoperative renal function remained normal.

He presented to the emergency department 11 weeks after surgery with upper abdominal and back pain and bilateral lower extremity ischemia. Laboratory analysis showed serum levels of lactic acid were 7.5 mMol/L, aspartate aminotransferase was 326 U/L, amylase was 522 U/L, and creatinine was 1.80 mg/dL. He was hemodynamically stable. His abdomen was soft but tender to palpation in the lower quadrants. All infrainguinal pulses were nonpalpable, and Doppler signals could not be obtained from either foot. He had complete sensory loss in both feet, with complete motor loss of both ankles.

A computed tomography (CT) angiogram of the thorax and abdomen showed a new type B aortic dissection (Fig 1). Within the abdomen, the true lumen was compressed, compromising flow to the celiac axis and superior mesenteric artery. The right renal artery and kidney, arising from the false lumen were nonenhancing. Contiguous with the narrowed true lumen, the upper half of the stent graft was collapsed and occluded, with distal thrombus extending into both legs.

The patient was transferred urgently to the operating room and, under general anesthesia, underwent bilateral femoral artery cutdowns. Both femoral arteries were pulseless and clot-filled. Guidewire and sheath access was obtained in both common femoral arteries and a hydrophilic wire was advanced up the right limb of the endograft to the aortic arch and subsequently exchanged for a stiff wire. Access through the left common femoral artery was achieved in a similar manner, and after passage of a wire up the iliac limb of the endograft to the aortic arch, a pigtail catheter was positioned in the arch to perform serial angiograms. The site of the tear was right at the distal aspect of the origin of the left subclavian artery. The stiff wire was felt to be in the true lumen, but intravascular ultrasound scanning was not available to confirm this.

We deployed a Valiant Free-flo stent graft (Medtronic Vascular, Santa Rosa, Calif) with proximal bare metal stents over the tear, with deliberate partial coverage of the left subclavian artery. A subsequent angiogram showed flow through the true lumen with a patent celiac, superior mesenteric, and left renal artery (Fig 2). The right renal artery was occluded. A dissection flap was present in the left renal

From the [a]Division of Vascular Surgery, and the [b]Department of Radiology, Royal Columbian Hospital.
Competition of interest: none.
Correspondence: Vikram Iyer, MD, Division of Vascular Surgery, McMaster University, Hamilton General Hospital, 237 Barton Street East, Hamilton, Ontario, L8L 2X2 Canada (e-mail: iyer@hhsc.ca).
0741-5214/$36.00
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doi:10.1016/j.jvs.2009.03.060

Fig 1. Coronal computed tomography slice shows **(a)** aortic dissection just distal to left subclavian artery with distal progression. Axial slices at the level of the **(b)** celiac axis and **(c)** superior mesenteric artery show severe compression of true lumen. Coronal slice in the abdomen shows **(d)** compression and occlusion of the endovascular stent graft.

artery, but this did not limit blood flow and was not treated. A Reliant aortic balloon (Medtronic Vascular) was passed up over the wires on both sides and, with gentle inflation inside the infrarenal endograft, was used as a thrombectomy catheter. Bilateral femoral thrombectomy with a No. 4 Fogarty catheter was then performed. The total operative time was 147 minutes, which included 24 minutes of fluoroscopy time. The total volume of contrast used was 60 mL.

Postoperatively, the patient's pain improved. He had palpable pedal pulses bilaterally. Transaminases and amylase levels returned to normal. Renal function deteriorated, with a peak creatinine of 4.57 mg/dL 4 days postoperatively. Dialysis was not required.

He had some residual weakness of the left ankle upon discharge at 3 weeks. At 11 months of follow-up he is back to normal activities and has resumed playing recreational badminton. His ankle-brachial indexes are normal. His renal function has improved, with a creatinine of 1.88 mg/dL. An unenhanced CT image at 6 and 11 months showed no significant change in thoracic or abdominal aortic diameters. The abdominal endograft remains expanded (Fig 3).

DISCUSSION

Only one other case of type B aortic dissection after endografting of an infrarenal AAA has been reported.[3] It was speculated in this report that the dissection began in the infrarenal aorta and propagated in a retrograde fashion.

Retrograde dissection after thoracic stent grafts has been reported in up to 2.4% of cases.[9] One report documents a retrograde type A dissection occurring after endovascular treatment of an uncomplicated type B dissection using an endograft with proximal bare springs.[4] On au-

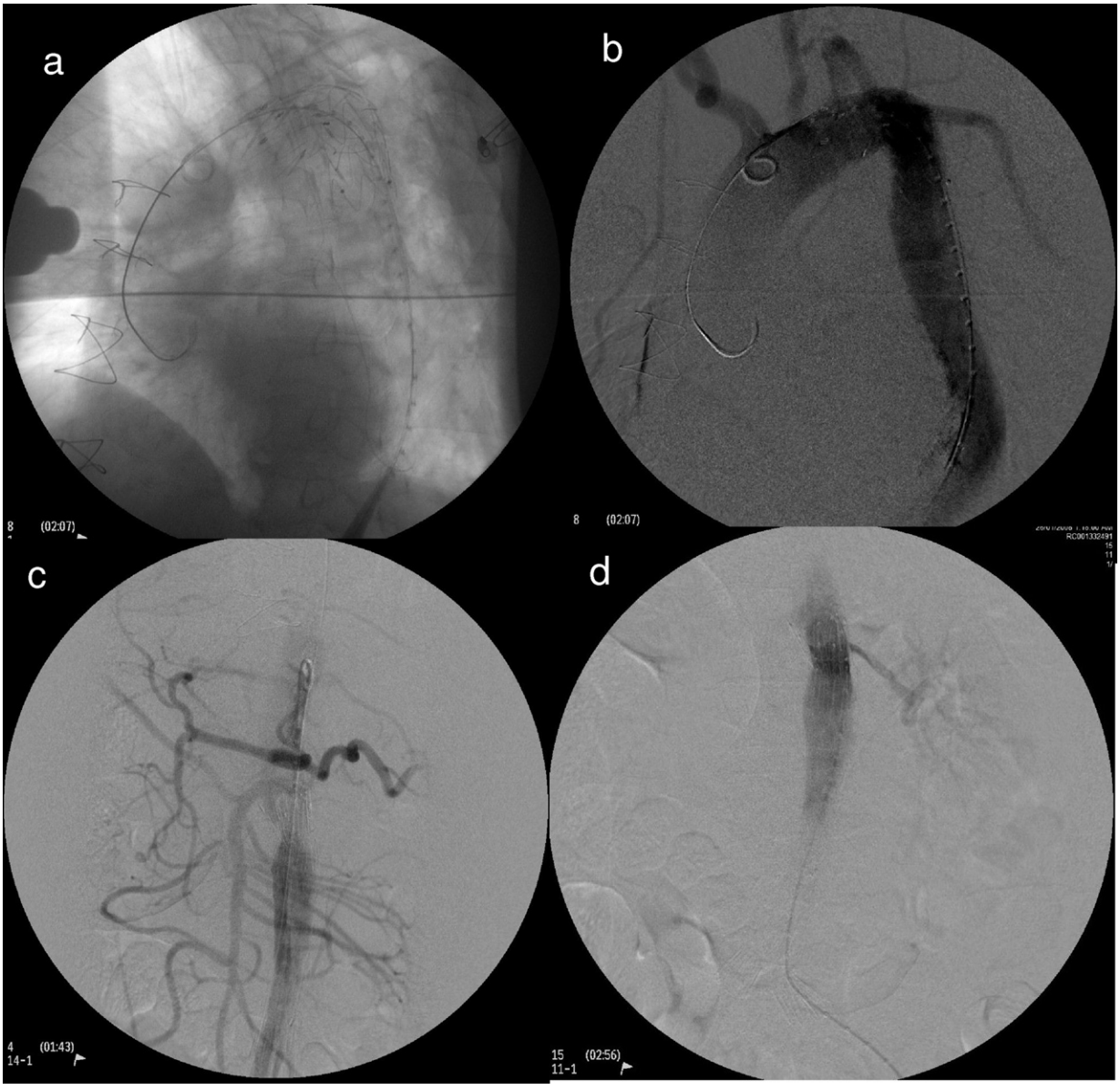

Fig 2. Intraoperative fluoroscopy and angiography shows **(a)** placement of the stent-graft with **(b)** contrast flow in the true lumen. The **(c)** celiac axis, superior mesenteric artery, and **(d)** left renal artery are all patent.

topsy, the bare springs were implicated as the cause of the intimal tear. At least two retrograde aortic dissections after renal artery percutaneous transluminal angioplasty have been reported.[5,6] Both had suboptimal result with initial angioplasty or stent placement leading to repeat percutaneous transluminal angioplasty with a larger balloon in once case and placement of a stent in the other. This suggests the underlying cause of dissection was related to the direct act of vessel expansion, as is also postulated in a case of aortic dissection after balloon angioplasty of an aortic coarctation.[7] In contrast, another mechanism for arterial dissection is direct wire injury, as reported in a patient with retrograde aortic dissection after iliac artery stenting.[8]

Dissection in the present patient has many possible causes. Direct wire injury to the thoracic aorta with subsequent antegrade dissection is one; however, we think this is unlikely because we never pass a stiff wire up to the aortic arch unless it is protected within a catheter. We routinely use a soft, angled glidewire under fluoroscopy, followed by a pigtail catheter and subsequent wire exchange.

Some possible anatomic factors also exist, including the mild calcification of the aorta just above the level of the renal arteries. More importantly, perhaps, was the irregular

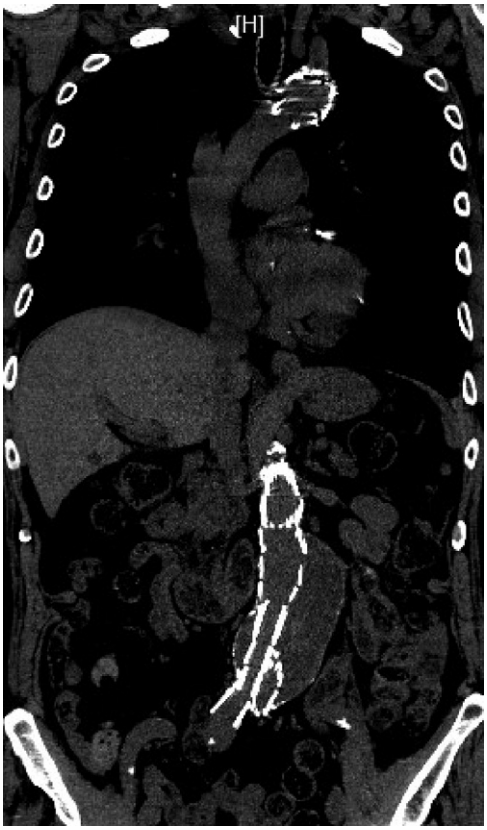

Fig 3. Nonenhanced coronal computed tomography slice shows re-expansion of the abdominal stent graft.

shaped infrarenal neck and substantial oversizing in the narrow-most segment (a 28-mm graft in a 18-mm vessel).

Device-specific factors could also have contributed to the dissection. The abdominal stent graft used has a proximal bare stent with anchoring barbs designed to penetrate the aortic wall. Bare stents were also present in the only previous report of a type B dissection after infrarenal stent graft placement. This presented as postoperative back pain without malperfusion. The dissection in this patient extended from the left subclavian artery down to the abdominal stent graft. This resulted in a thoracoabdominal aortic aneurysm requiring open repair. At the time of surgery, an ulcerated, calcified plaque had apparently been displaced by the stent anchors and was thought to be the origin of the dissection.

Finally, the aortic dissection could have been spontaneous, beginning at the subclavian artery away from the area of previous intervention. The delayed presentation in our patient may support this. In many reported cases of dissection after intervention, the presentation has been immediate.[3-6,8] It is possible that there was a subclinical dissection occurring at the time of the procedure that progressed over 11 weeks. Unfortunately, no postoperative CT scans were performed to confirm or refute this. It is therefore possible that the dissection was purely coincidental.

Whatever the cause, or whether the dissection began in the proximal aorta or at the site of the infrarenal endograft, because of distal false lumen thrombosis, the proximal tear acted as the entry tear causing pressurization of the false lumen and compression of the true lumen, and in this case, the endograft. Fortunately, coverage of this entry tear, as described by Dake et al,[10] led to reperfusion of the true lumen and its branches along with re-expansion of the abdominal stent graft.

CONCLUSION

Acute aortic dissection in the presence of a pre-existing abdominal aortic stent graft can lead to device collapse and subsequent limb malperfusion. A combination of factors, such as infrarenal neck morphology, device oversizing, bare metal stents with barbs, and wire manipulation in the proximal aorta may have caused this complication. Like re-expansion of a collapsed true lumen, re-expansion of a collapsed abdominal stent graft is possible with repressurization of the true lumen by endografting of the proximal entry tear.

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Submitted Dec 31, 2008; accepted Mar 31, 2009.