Retrograde type B aortic dissection after fenestrated-branched endovascular aortic aneurysm repair

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ABSTRACT

Retrograde type B aortic dissection after perivisceral aortic endograft placement is a rare occurrence, not well described in literature. A handful of case reports describe this complication after infrarenal endovascular aortic aneurysm repair (EVAR). We have identified retrograde type B aortic dissection after fenestrated-branched EVAR (F/BEVAR) as a rare, but real complication. We describe diagnosis and management of 4 such occurrences in the context of 344 F/BEVARs. (J Vasc Surg Cases Innov Tech 2024;10:101586.)

Keywords: FEVAR complication; Retrograde dissection; Immediate or delayed

Retrograde type A aortic dissection (RTAAD) is a known, although rare, complication of thoracic endovascular abdominal aortic aneurysm repair (TEVAR), with reported rates in the range of 2.5%.[1,2] Retrograde type B aortic dissection (RTBAD) after perivisceral aortic endograft placement is an even less common occurrence. A Vascular Quality Initiative study noted a rate of 0.7% for non-type A retrograde dissections after TEVAR.[2] There are few existing case reports describing retrograde TBAD after infrarenal EVAR. We have identified retrograde TBAD after fenestrated-branched EVAR (F/BEVAR) is a rare, but real complication.

We describe diagnosis and management of four such occurrences in the context of 344 F/BEVARs (Cook Medical CMD, Bloomington, IN), of which 260 had proximal-most landings in zones 4 or 5. The remainder of the F/BEVARs were landed in zones 0 to 3 and were not considered "at risk" for RTBAD, because the descending thoracic aortas were fully covered at the time of procedure. This results in a RTBAD incidence of 1.5% in our cohort.

The patients described here have consented to publication of all case details and associated images. Consent was obtained from families for patients who are deceased.

CASE 1

A 67-year-old man presented with a type IA endoleak after previous infrarenal EVAR and right iliac-branched graft. He underwent four-vessel F/BEVAR using a custom-made device (CMD, Cook Medical). A 34-mm graft was landed proximally in zone 5, where the 30-mm distal thoracic aorta was uncalcified, without disease. The proximal landing zone was not ballooned. Intraoperatively, flapping of the proximal endograft was noted on completion angiography, suspicious for dissection (Video 1). Postoperative computed tomography angiography (CTA) confirmed dissection extending from the subclavian artery to the top of the endograft with no obvious entry tear (Fig 1). The patient was brought back to the operating room on postoperative day 3 to undergo placement of an aortic cuff and two open dissection stents. One and one-half years later, he was noted to have continued dilation of the proximal thoracic aorta and underwent proximal TEVAR extension to the level of the left subclavian artery. At the 3.5-year follow-up, he was noted to have a type IA endoleak of the TEVAR and is now planned for carotid-subclavian bypass with proximal TEVAR extension. Retrospective review of intraoperative anesthesia records for the initial procedure demonstrated an intraoperative systolic blood pressure (SBP) of <140 mm Hg for most of the case. There was one 40-minute period mid-procedure and a later 10-minute period toward the end of the case where the SBP did rise to the 170 to 180 mm Hg range.

CASE 2

A 77-year-old man presented with type IV thoracoabdominal aortic aneurysm and bilateral common iliac artery aneurysms. He underwent four-vessel F/BEVAR using a CMD (Cook Medical) and bilateral iliac branched graft placement. A 36-mm graft was landed proximally in zone 5, where the 32-mm distal thoracic aorta was healthy appearing and without calcification. The proximal landing

Fig 1. Acute type B aortic dissection (*TBAD*) extending from subclavian artery takeoff to the top of the endograft.

zone was not ballooned. TBAD was noted on completion angiogram and confirmed on intraoperative transesophageal echocardiography (Fig 2, *A*, Video 2). Flapping of the proximal end of the graft was also noted intraoperatively. The patient underwent proximal extension with an uncovered dissection stent intraoperatively, with immediate resolution of the dissection (Fig 2, *B*, Video 3). He did require open thrombectomy to treat an ischemic left leg. Although blood flow was restored, he unfortunately passed on postoperative day 3 owing to persistent acidosis. The source of persistent acidosis remained unclear, but perhaps was an ongoing consequence of delayed diagnosis of an acute limb. A CT scan performed in that time period demonstrated intact F/BEVAR with patent renal and visceral vessels, dissection stent in place, and no evidence of TBAD or bowel ischemia. Retrospective review of intraoperative anesthesia records for the initial procedure demonstrated consistent SBP of <140 mm Hg throughout the entire procedure.

CASE 3

An 82-year-old man presented with juxtarenal abdominal aortic aneurysm after previous open aortobiiliac with a very low proximal anastomosis. He underwent three-vessel F/BEVAR with celiac scallop using a CMD (Cook Medical). A 42-mm graft was landed proximally in 36-mm native, healthy-appearing aorta at the level of the celiac artery. The proximal landing zone was not ballooned. A CTA immediate postoperatively demonstrated intact repair without any abnormalities. At the 2-month clinic visit, he was well. Four months postoperatively, he presented to the emergency room with acute chest, back pain and hypertension to 170/89 mm Hg. CTA demonstrated new TBAD which appeared to originate from the aortic hiatus at the fenestrated endograft and extended retrograde to the proximal descending thoracic aorta 2 to 3 cm distal to the subclavian artery takeoff (Fig 3). Contrast enhancement of the false lumen was best distally and seemed to fade more proximally. He was admitted to the cardiovascular intensive care unit for anti-impulse therapy, where his symptoms resolved. He was transitioned to oral antihypertensives and transferred to the ward. Repeat CTA did demonstrate expansion of the false lumen from 1.1 to 1.7 cm. There was ongoing discussion for timing of further treatment, but unfortunately, 2 days after arriving on the ward, the patient became acutely dyspneic, hypotensive, bradycardic, arrested, and could not be resuscitated.

CASE 4

An 85-year-old man presented with a type IA endoleak after previous infrarenal EVAR. He underwent four-vessel F/BEVAR using a CMD (Cook Medical) and left accessory renal artery coil embolization without issue. A 32-mm graft was landed in zone 5, where the thoracic aorta was healthy appearing and without calcification; completion angiography was without abnormalities. The proximal landing zone was not ballooned. An initial postoperative CTA was performed at 2 months when the patient presented with retrosternal chest pain and hypertension. At this time, he was found to have an intramural hematoma (IMH) extending from the subclavian artery to the top of the endograft with several focal areas of contrast enhancement. He was started on an oral antihypertensive. Repeat CTA another month later demonstrated stable appearance of the IMH. He remains asymptomatic with resolution of the IMH; however, a focal ulceration of 1.2 cm remains that has stabilized in size, he is being managed medically with serial CTAs.

DISCUSSION

Retrograde TBAD after fenestrated/branched endografting in the visceral aorta is a rare event. To our knowledge, there are no reported cases of RTBAD after F/BEVAR. We identified two instances of RTBAD that occurred intraoperatively and two that occurred postoperatively, both in the context of hypertension. The intraoperative RTBADs were treated promptly, while the ones noted postoperatively were treated medically. One of
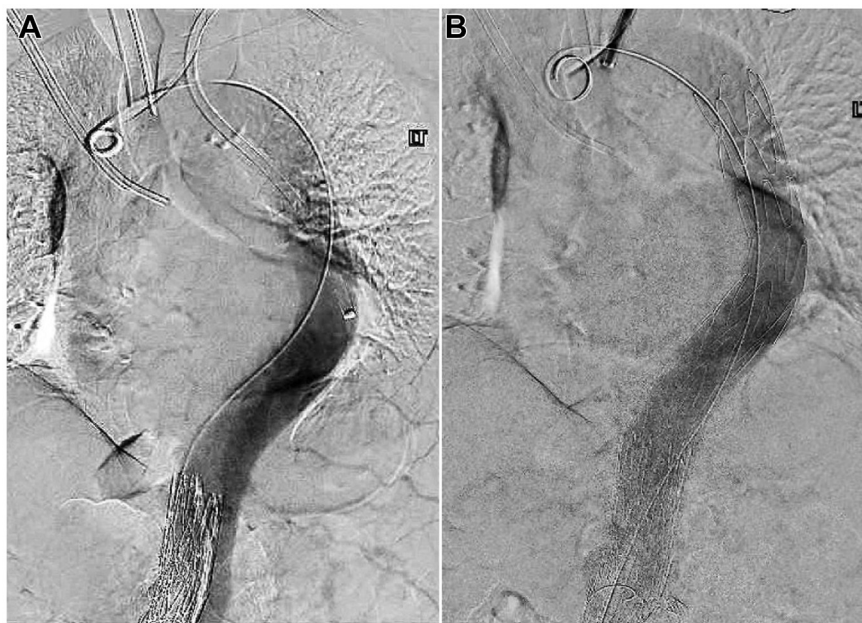
Fig 2. (A) Acute type B aortic dissection (*TBAD*) after fenestrated endovascular abdominal aortic aneurysm repair (*FEVAR*) with contrast outside of the graft lumen. **(B)** Resolution of TBAD after placement of open dissection stent.
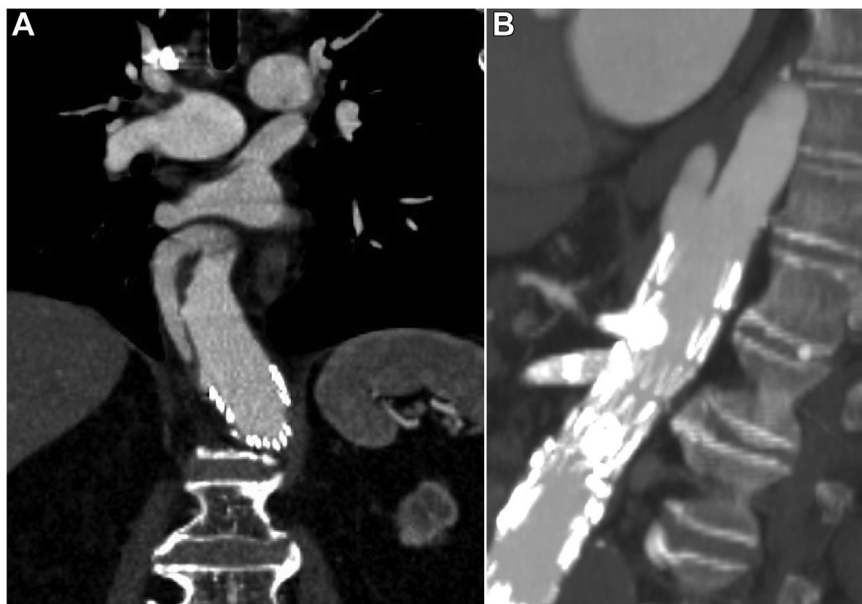

Fig 3. Coronal **(A)** and sagittal **(B)** views of a computed tomography scan 4 months after fenestrated endovascular abdominal aortic aneurysm repair (*FEVAR*) demonstrating acute type B aortic dissection (*TBAD*) originating from top of previously placed endograft.

four (25%) died as a direct result of RTBAD after F/BEVAR. In all patients, CTA demonstrated normal-appearing ascending aorta, aortic arch, and thoracic aorta; endografts were not oversized significantly; endografts were designed with barbs at the proximal end and the proximal end of the F/BEVAR graft did not undergo ballooning.

Unlike RTAAD, which has been well-described in literature, RTBAD has only been described in a few case reports, all of which were in the context of infrarenal EVAR.[3-13] Several factors have been identified as possible causes for RTBAD in the infrarenal EVAR series. One common theory is that landing the anchoring barbs in a diseased infrarenal neck (eg, angulated,

calcified, or ectatic) may cause intimal trauma. The barbs are designed for fixation, but as they penetrate the aortic layers, they have the potential to allow blood into the wall, allowing an entry site for dissection to develop especially if there is coexisting hypertension. In our F/BEVAR series, each proximal graft did have anchoring barbs, but a review of CTAs confirmed that our proximal landing zones were all in healthy distal thoracic aorta without significant calcification or tortuosity.

Additional potential causes include substantial endograft oversizing, iatrogenic trauma from guidewires and catheters, and hypertension. Measurement of proximal landing zone diameter compared to endograft size confirmed that substantial graft oversizing was not of concern in any of our patients. Iatrogenic trauma from guidewires and catheters is frequently identified as a potential cause for RTAAD and may have applicability in RTBAD after F/BEVAR as well.[1] Subclinical entry tears formed in the proximal to mid thoracic aorta may evolve over time and eventually develop into aortic dissections with re-entry tears at the level of the proximal endograft. Hypertension was noted in both the delayed RTBAD presentations, and one of the intraoperative presentations. Given the retrospective nature of this study, however, it is not possible to discern at which point during the procedure hypertension occurred during case 2 (eg whether diameter reducing ties had already been released). It is also worth pointing out that Case #1 had no documented blood pressure fluctuations during the procedure.

Unique to F/BEVAR, small rotational and vertical manipulation of the endograft is common and can be a potential cause for aorta wall trauma in narrow lumens. During graft deployment, small rotational adjustments of the graft are performed to align each fenestration with the target artery before deployment of the diameter reducing ties. After deployment and before cannulation, diameter reducing ties remain on the graft to minimize contact between graft and aortic wall allowing for further adjustments required during cannulations. After cannulation and full deployment of the graft (eg, release of diameter reducing ties), there is no further rotational manipulation of the graft. Significant graft rotation, graft manipulation, or grossly misaligned fenestrations are typically noted in the operative report, though this did not seem to be the case in any of the above reported RTBAD cases.

Additional key differences between the EVAR and F/BEVAR patients are worth noting. The timing of dissection formation in the EVAR cases ranged from 2 days to 108 days, whereas two cases of our F/BEVAR cases were noted intraoperatively. In placement of an infrarenal EVAR endograft, balloon molding of the infrarenal neck is a key maneuver to ensure proximal fixation; this is not the case in F/BEVAR, and none of our patients underwent ballooning in the proximal extent of the stent graft.

Given the rarity of these occurrences and the paucity of literature on RTBAD after F/BEVAR, management of this complication remains in evolution. In retrospect, case 1 could have been treated intraoperatively with bare stents. The 3-day delay between initial dissection and treatment potentially allowed for formation of additional re-entry tears, which decreased the efficacy of the bare stents, resulting in later continued expansion of the false lumen. Immediate treatment after intraoperative recognition seems most effective, as shown in case 2. Further retrospective observations are in management of delayed RTBAD. We hypothesize that the cause of mortality for case 3 was rupture. The false lumen growth, when compared with the patient's presenting CT scan, was attributed initially to aortic changes that potentially occurred during his hypertensive/symptomatic phase while in the cardiovascular intensive care unit. Given that he had been normotensive and without symptoms for several days before the follow-up scan, we did not feel significant urgency in bringing him back to the operating room, although those discussions were underway. In retrospect, more aggressive treatment was warranted, despite the patient's relatively benign appearance. In summary, we would recommend immediate intraoperative treatment if RTBAD is recognize intraoperatively. In delayed presentations, we recommend expeditious treatment if there is any imaging evidence of aneurysm instability, regardless of the patient's symptomatic status.

CONCLUSIONS

RTBAD after F/BEVAR is a rare and potentially lethal complication that can occur up to several months postoperatively. The precise etiology remains unclear, although could be related to proximal barb fixation but hypertension likely plays a key role. Early intraoperative recognition can be confirmed via TEE and treated with uncovered stents. Delayed postoperative RTBAD should be followed closely for progression and treated aggressively if growth is noted.

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