Operative strategy for open surgery after failed thoracic aortic stent grafting

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Data and operative strategy regarding thoracic aortic stent-graft (TASG) explantation are sparse.^{1,2} We herein report our experience with 2 patients who required late TASG removal.

Clinical Summary

PATIENT 1. A 28-year-old woman with Marfan syndrome was found to have a root dilatation with severe aortic and mitral regurgitation. A bileaflet mitral repair, a Bentall procedure, and a hemiarch replacement were performed. Five months later, the patient underwent a proximal descending aortic resection for a dissecting aneurysm. Three months later, a false aneurysm at the distal anastomotic site was diagnosed on a computed tomographic (CT) scan. Considering the recent thoracotomy, the pseudoaneurysm was treated with a 24-mm Valiant stent graft (Medtronic, Inc, Santa Rosa, Calif). On the postoperative CT scan, an aortic dissection distal to the stent was demonstrated. Three months later, the patient was admitted for New York Heart Association class III/IV dyspnea caused by recurrent severe mitral regurgitation. Control CT scanning showed a significant type I endoleak (Figure 1). The patient underwent mitral replacement, TASG explantation with descending aortic replacement, and pectus excavatum repair through a clamshell incision. Because of the difficulty of clamping the proximal descending thoracic graft, the TASG was removed during deep hypothermia and circulatory arrest (DHCA). The aortic perfusion was re-established through the cannula in the arch. An open distal anastomosis with septectomy was performed. The postoperative course was marked by a 3-day intubation because of chest wall instability. The thoracic aorta is free from complications on CT at 8 months (Figure 2).

PATIENT 2. A 61-year-old man with a known type I thoracoabdominal aneurysm was admitted for a contained rupture at the distal third of the descending thoracic aorta. Three 100-mm Talent (Medtronic, Inc) stent grafts were implanted (40 × 40, 44 × 44, and 46 × 46 mm). Control angiography showed no endoleak.

Figure 1. Contrast-enhanced chest computed tomographic scan demonstrating a type 1 endoleak at the distal portion of the thoracic aortic stent graft in a patient with severe pectus excavatum.

Figure 2. Postoperative contrast-enhanced chest computed tomographic scan showing the replaced segment of the descending aorta and the pectus excavatum repair.

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Follow-up CT scans were normal for 24 months until December 2006, when a new distal type I endoleak was observed (Figure 3, A). The patient was judged to be an acceptable candidate to undergo a thoracoabdominal repair. In June 2006, TASG explantation with DHCA was performed. Because of the presence of thrombus around the distal stent, aortic cannulation was performed in the ascending aorta to minimize the risk of retrograde cerebral emboli. The proximal bare springs were embedded in the distal

Figure 3. A, Contrast-enhanced computed tomographic scan showing a type 1 endoleak caused by dilatation of the descending thoracic aorta at the distal portion of the stent graft. B, Embedded proximal bare springs of a thoracic aortic stent graft in the distal arch (white arrow) and adjacent to the left subclavian artery (*). C, Presence of significant thrombus surrounding the endoprosthesis.

arch (Figure 3, B and C). The proximal perfusion was resumed after completion of the proximal anastomosis, and an open distal anastomosis was performed. The celiac and superior mesenteric arteries were perfused selectively and reimplanted separately to the main graft. The postoperative course was uneventful, and the thoracic aorta is free from complications on follow-up CT (Figure 4).

Discussion

TASG implantation implies a lifelong follow-up. Advent of a type I endoleak portends a poor outcome and dictates repair. In both our patients a type I endoleak was diagnosed. An open conversion was the only modality to correct the expanding dissection and type I endoleak in the first case. The second patient showed a distal type I endoleak caused by a remodeling of the aorta. A hybrid approach with revascularization of the visceral arteries with stent-graft extension could have been possible.³ However, both cases had good long-term life expectancy and acceptable open operative risks.

Figure 4. Postoperative contrast-enhanced chest computed tomographic scan showing the replaced aorta at the level of the previous stent-graft endoleak.

Operative strategy has to be tailored to each case and has to take into account the type of endograft, its position and length, and the patient's overall operative risk. The classic surgical principles of the descending thoracic aorta should also be applied. Use of DHCA is dictated by the proximal extent of the stent graft and the ability to safely clamp the aorta above the stent graft. Furthermore, as encountered in patient 2, significant clot burden around the endograft might preclude the safe use of a femoral cannulation for DHCA. In such circumstances, a proximal cannulation either through the ascending aorta, the left carotid artery, or the left axillary artery should be considered to minimize the risk of retrograde cerebral emboli.

The necessity to remove all stent grafts remains controversial. We personally think that when feasible, all stent-graft material should be removed to eliminate the risks of late stent-graft complications. In high-risk patients or in patients in whom complete TASG removal is judged at very high risk, such as proximal arch stents, an incomplete TASG resection might be acceptable. In such circumstances, crossclamping of the TASG is possible because the nitinol stent framework should resume its initial position once the crossclamp is released. If external aortic crossclamping is difficult, endoluminal clamping with an endoclamp might be an alternative.⁴ After crossclamping, the stent graft is transected between two Z stents, and an anastomosis is performed by incorporating the stent-graft material, the aortic wall, and outer reinforcement with felt.⁵

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Postural change causing leg malperfusion resulting from expansion of a patent false lumen in type B aortic dissection

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Leg malperfusion is an important symptom resulting from aortic dissection (AoD).¹ Type B AoD, especially with a patent false lumen, exhibits various and complex clinical manifestations. There are no previous reports regarding leg malperfusion caused by postural change. We report 2 interesting cases of patients with type B AoD and a patent false lumen who presented with intermittent claudication despite an ankle brachial pressure index (ABPI) of 1.0.

Clinical Summary

Case 1. A computed tomographic (CT) scan in a 73-year-old man with acute type B AoD revealed that the patent false lumen was tense. Several months later, the patient was examined again with intermittent claudication, but a follow-up CT revealed no specific change. When the patient was supine, the ABPI was 1.0 in both legs and duplex ultrasound of the popliteal artery showed a normal flow pattern (Figure 1, C). However, the reverse phase flow disappeared and the diastolic phase flow increased when the patient stood up (Figure 1, D). We suspected that the true lumen was further compressed to a paper-thin sliver when the patient stood up (Figure 1, A, B), and intermittent claudication occurred. An axillo-bifemoral bypass was electively performed for extra-anatomic revascularization. Postoperatively, the duplex ultrasound revealed a normal

flow pattern regardless of postural change (Figure 1, E, F), and the symptom resolved.

Case 2. Intermittent claudication with numbness of the left leg developed in a 47-year-old man with acute type B AoD. CT revealed that the false lumen was patent and that a dissection flap extended to the right common iliac artery, where there was a small reentry tear (Figure 2, A). Although the ABPI was 1.0 in both legs when the patient was supine, his left leg lost pulsation and the blood pressure diminished with a symptom of numbness after walking. It appeared that the true lumen was compressed by further expansion of the patent false lumen resulting from the postural change and walking exercise, and that the bloodstream of his left leg was severely disturbed (Figure 2, B, C). Surgical aortic fenestration above the abdominal aortic bifurcation (Figure 2, D) was performed, and the symptom resolved.

Discussion

The treatment strategy for type B AoD is still controversial and is made on an individual basis.² Even in patients who survive until hospital discharge, the mortality is insufficient, despite improved surgical techniques and the advent of new endovascular therapies, such as stenting and fenestration.² Akutsu and colleagues³ demonstrated that false lumen patency is a strong independent prognostic factor for type B AoD. The anatomic position of the intimal flap is important with regard to peripheral malperfusion of branch vessels. Gaxotte and colleagues⁴ reported that evaluation of the intimal flap position was important and useful for endovascular treatment strategies.

In our 2 cases, the symptom of intermittent claudication had some discrepancies from the ABPI. Although the precise mechanism of this unique manifestation is difficult to prove, we propose that postural change is a key factor in the atypical leg malperfusion. A possible mechanism is that the true lumen is compressed by further expansion of the patent false lumen, such that the bloodstream in the true lumen diminishes when a patient changes his or her position to standing. A duplex ultrasound study of the popliteal artery in the standing position in case 1 and an exercise test in case 2 were effective for elucidating the unique mechanisms of the atypical leg malperfusion. After surgi-

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