Heart, Lung and Circulation (2015) **24,** e206–e209
1443-9506/04/$36.00
http://dx.doi.org/10.1016/j.hlc.2015.07.004

CLINICAL SPOTLIGHT

Retrograde Type A Aortic Dissection Treated with Continuous Perfusion ''Branch-First'' Aortic Arch Replacement Technique

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Received 9 October 2014; received in revised form 13 January 2015; accepted 5 July 2015; online published-ahead-of-print 14 August 2015

The development of thoracic endovascular aortic repair in recent years has revolutionised the way aortic disease is treated. However, there are potential complications associated with this which can be life threatening and pose a difficult challenge to manage. We present a case of retrograde ascending aortic dissection complicating thoracic endovascular aortic repair, and its repair using a technique of continuous perfusion ''branch-first'' aortic arch replacement. We discuss the complication of retrograde ascending aortic dissection and the issues that affect its surgical management.

Keywords Retrograde dissection • TEVAR • Thoracic stent-graft • Endograft • Graft

Case Report

A 64-year-old female presented with interscapular pain, radiating through to her chest. She was hypertensive with a blood pressure of 200 systolic. Computed tomography angiography (CTA) revealed a Type B aortic dissection commencing immediately distal to the left subclavian artery and extending into the right common iliac artery. The CTA also showed a 5 cm aneurysm of the ascending aorta. Her uncomplicated Type B aortic dissection was managed with analgesia and blood pressure control. Three days later, she reported intermittent paraesthesia and claudication of the right lower limb. Repeat CTA confirmed dynamic malperfusion of the right common iliac artery. She was managed with left common carotid to left subclavian artery bypass (Omniflow II Vascular Prosthesis x 6 mm, Bio Nova International, Melbourne, VIC, Australia) and stent grafting to the distal arch (Zenith TX2 TAA Endovascular Graft with Proform 42 x 135, Cook, Bloomington, IN, USA) which was deployed immediately distal to the left common carotid artery. A dissection stent was then placed in the descending thoracic aorta (Zenith Dissection Stent 46 x 165, Cook, Bloomington, IN, USA). Passage of the dissection stent delivery system caused excessive friction with the outer wall of the TX2 stent resulting in 2 cm of proximal migration of the TX2 stent and 'shuttering' of the left common carotid artery origin. An unsuccessful attempt to pull the TX2 distally back into position was made with balloon dilatation and distal traction. Despite covering the primary tear, the right common iliac artery remained occluded. Consideration was given to either a fenestration or infrarenal AAA stent grafting, or a femoro-femoral crossover. The EVAR was chosen as it provided definite treatment to a larger area of dissected aorta, it would be protected against delayed aorto-iliac aneurysmal

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degeneration, larger French femoral access had already been obtained with the TEVAR, and the anatomy lent itself to this. An infra-renal bifurcated aorto-iliac stent graft (Zenith Flex AAA Endovascular Graft and Spiral-Z Iliac Leg Graft, Cook, Bloomington, IN, USA) was placed which resulted in reperfusion of her lower limbs. Completion angiography did not reveal a Type A dissection.

She had an uneventful recovery. Two days later, follow-up carotid CTA was performed to evaluate the degree of coverage of the carotid artery ostium by the stent graft. While the patient was asymptomatic, the CTA revealed a retrograde ascending aortic dissection (RAAD) (Figure 1). The patient was taken to the operating theatre immediately for surgical repair. On-table transoesophageal echocardiography showed severe aortic regurgitation secondary to prolapse of the non-coronary valve leaflet. Operative findings revealed a primary tear on the inferior aspect of the mid arch at the proximal margin of the stent graft. The dissection false lumen extended proximally to involve the entire non-coronary and part of the right coronary aortic sinuses. It also involved the proximal half of the innominate artery and left common carotid artery. The stent graft covered the distal half of the ostium of the left common carotid.

The aortic arch was replaced using the continuous perfusion ''branch-first'' aortic arch replacement technique described by Matalanis [1]. Cardiopulmonary bypass (CPB) was established with cannulation of the right femoral vessels. Cooling to 25 degrees centigrade was commenced. The aortic arch was debranched by sequentially anastomosing the innominate and left common carotid arteries to the 12 and 8 mm limbs of a trifurcation arch graft (Vascutek 12/10/8 x 8 mm, Renfrewshire, Scotland, UK) thereby excluding the dissected portions of both vessels. Antegrade cerebral perfusion via the perfusion limb of the trifurcation arch graft was used for the remainder of the procedure. The mid-ascending aorta was cross-clamped and the proximal ascending aorta transected above the sinotubular junction. The heart was arrested using custodial cardioplegia. Lower body circulatory arrest was instituted. The ascending aorta was excised from the sinotubular junction to the distal aortic arch, 10 mm from the TEVAR graft. A straight Dacron graft with an 8 mm side arm (Vascutek Ante-flo 30 x 8 mm, Renfrewshire, Scotland, UK) was anastomosed to the stent graft and surrounding aortic wall, using a series of pledgetted interrupted horizontal mattress 2/0 polyester braided sutures, creating an everting suture line. The suture line was reinforced with a running 3/0 polypropylene suture. Antegrade lower body perfusion was recommenced via the arch graft 10 mm side-arm and rewarming commenced.

The aortic valve commissures were resuspended, the dissected aortic root repaired and the surgical graft anastomosed to the sinotubular junction. The trifurcation graft was then connected to the ascending aortic graft (Figure 2).

The patient was weaned off CPB. Bypass time, cross clamp time and lower body circulatory arrest were 236, 135, and 46 minutes, respectively. She made an uneventful recovery and was discharged home 12 days later.

The patient remains well at six-month follow-up. Transthoracic echocardiography at three months showed normal aortic valve cusp movement with a mild central jet of aortic regurgitation.

Discussion

Acute type B aortic dissections represent approximately 25-40% of all acute aortic dissections [2]. They have traditionally been managed medically if stable with a mortality of 6.4% and only surgically if complicated, due to the high mortality and morbidity of surgical repair [3]. Recent advances in thoracic endovascular aortic repair (TEVAR) techniques have changed this approach and TEVAR has become the treatment of choice for complicated type B aortic dissections with a mortality of 10.2% [3]. It may also have a role to play in uncomplicated cases that are at high risk of longer-term complications [2–4]. The increasing use of TEVAR has highlighted a number of complications, one of which is RAAD.

Retrograde Type A Aortic Dissection after endovascular repair has several possible aetiologies related to the procedure, the device or disease progression [5]. These include aortic injury from the catheter, wire, delivery device or stent graft manipulation, aggressive balloon dilatation, inadequate perioperative blood pressure control and inappropriate patient and device selection, particularly oversizing in patients with fragile aortic walls [6,7]. In our case it is likely that deployment of the dissection stents or the attempt at repositioning the stent graft caused the RAAD. We would thus recommend abandoning dissection stent placement if its delivery system fails to track easily through the previously placed stent graft.

The incidence of RAAD following TEVAR for all causes is 1.33-1.9%, 4.4% following TEVAR for dissection, and 8.4% after TEVAR for acute Type B dissections [5,6,8]. Although most complications occur during or early after TEVAR, RAAD may occur up to 36 months following repair [9]. In such cases it may be related to progression of underlying aortic disease rather than the procedure itself [9,10]. In patients with a dissection, ascending aortic diameter > 4 cm and a TEVAR landing zone 0, are risk factors for RAAD. When all factors are present the incidence may be as high as 25% [5]. There must be a high index of suspicion for RAAD following TEVAR, as the presentation can be ambiguous and can be rapidly life threatening with 19-25% of patients experiencing sudden death. It is associated with a 30-day mortality of 33-42% despite repair [5,6,8]. These alarming figures have led some authors to recommend that any patient undergoing TEVAR be assessed for RAAD with intravascular ultrasound or transoesophageal echocardiography at the conclusion of the case, in addition to undergoing lifelong aortic surveillance [2,3].

Surgical repair of RAAD must follow the principles of excising or excluding the primary tear, stabilising the proximal aspect of the existing stent graft to prevent type 1 endoleak, and treating all residual dissected or aneurysmal aorta