Endovascular treatment of thoracic aortic disease

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Endoluminal repair is now a realistic alternative to open surgery for the treatment of thoracic aortic disease

Since Volodos and colleagues performed the first endoluminal repair of a thoracic aneurysm, the technique has been used to treat descending thoracic aneurysms, type B (Stanford) aortic dissection, false aneurysms, penetrating ulcers, and aortic transection. This minimally invasive approach has many advantages over conventional surgery as it avoids open thoracotomy, single lung ventilation, and aortic cross clamping.

The main criticism of endoluminal repair is poor durability of the stent grafts. The first published series from Stanford used home made stent grafts and reported a primary success rate of 73%. However, there were problems associated with the large introducer catheters and rigidity of these devices. The now commercially available stent grafts are more flexible with smaller delivery systems and have improved deployment mechanisms. Notably, stent graft failure has been reported with the early home made grafts and more recently the Gore Excluder (WL Gore Associates, Inc, Flagstaff, Arizona, USA) has been withdrawn for redesigning following reports of frictions in the nitinol frame. The longer experience from the infrarenal devices has shown that lifelong surveillance is essential.

INDICATIONS FOR TREATMENT

Descending thoracic aneurysms are life threatening, with an estimated incidence of 6 cases per 100 000 person years. The number of patients with thoracic aneurysms is increasing presumably because of better diagnostic modalities and longer life expectancy. We advise treatment for symptomatic aneurysms and for asymptomatic aneurysms greater than 6 cm in diameter. Several reports have reported the risk of rupture in patients with untreated aneurysms to range from 46–74%, with five year survival rates estimated at 9–13%. The contraindications for endovascular repair of thoracic aneurysms are absence of an aneurysm neck, an excessively large neck, or insufficient normal aorta to fix the stent graft either proximally or distally. For aneurysms involving the proximal descending aorta it is sometimes necessary to intentionally cover the origin of the left subclavian artery. This can be done without the need for carotid subclavian transposition. Currently all patients have a computed tomographic (CT) scan and calibration angiography as a planning procedure. The procedure can be carried out in the operating theatre or the angiography suite, but good quality imaging is essential. We prefer to use regional or local anaesthesia as this allows monitoring of distal neurological function throughout the procedure.

A major indication for endoluminal treatment in the thoracic aorta is type B (Stanford) aortic dissection. The majority of patients with acute type B aortic dissection are treated medically with antihypertensive drugs and β blockers. Endovascular intervention is reserved for ongoing pain, refractory hypertension, localised false aneurysm, end organ ischaemia, and rupture, which occur in 30–40% of patients. Open surgery in this group of patients is associated with a very high morbidity and mortality (35–50%). Endovascular treatment has also been recommended for penetrating ulcers and intramural haematomas of the descending aorta, which have been identified as precursors of dissection.

Endovascular treatment of complicated type B dissection includes covering the primary entry tear with a stent graft, percutaneous fenestration of the intimal flap, and stenting of obstructed aortic side branches. The primary endovascular technique is placement of a stent graft across the primary entry tear which will decompress the false lumen and can relieve both static and dynamic obstruction of aortic branches. Experiments have shown that decreasing the false lumen inflow by placing a stent across the primary entry tear is the most effective treatment for true lumen collapse. In addition, endoluminal exclusion promotes thrombosis of the false lumen, which has been shown to decrease the risk of aneurysm formation. Some authorities have recommended stenting all patients presenting with acute type B dissection to reduce the incidence of late aneurysm formation. However, as only about a third of patients will develop aneurysms this should be assessed against best medical treatment in a randomised controlled trial.

The Stanford group were the first to report their experiences with home made grafts and acute aortic dissection. They reported that revascularisation of obstructed aortic branches occurred in 76%. Long term follow up has shown 79% (15/19) patients had complete thrombosis of the false lumen. Nienaber and colleagues reported a 0% mortality and morbidity in a small series of 12 patients treated by endoluminal repair, which compared very favourably with their own surgical experience. They have since published results on a further 82 patients with excellent results. However, it is difficult to interpret this data as it does not specify the distribution of acute and chronic cases, or the number with rupture or end organ ischaemia. It appears that stent graft placement in the acute situation can prevent late aneurysm formation by facilitating complete thrombosis of the thoracic aortic false lumen.
However, there are still problems with rigid stent grafts, which can erode through the intimal flap and cause pressurisation of the false lumen leading to rupture. There is a place for a stent graft that is specifically designed for the treatment of aortic dissection, which is blunt with a tapered end. Despite these problems, endoluminal repair is currently the treatment of choice for acute and chronic type B dissection, penetrating ulcers, and intramural haematomas.

COMPLICATIONS

The avoidance of aortic cross clamping reduces the risk of end organ damage from ischaemia and ischaemia–reperfusion. Cardiac (1% v 10%), respiratory (8% v 28%), and renal complications are significantly lower than for open surgery. The most common complication of endoluminal repair is damage to the access artery, which is caused by a combination of the large calibre delivery system and pre-existing iliofemoral atheromatous disease. For patients with small arteries an iliofemoral bypass can be inserted to allow safe passage of the introducer. An incidence of stroke has been reported as 4–7% and is usually caused by manipulation of the guidewire or device in the aortic arch causing cerebral embolisation.

As with open surgery, paraplegia is the most devastating complication of endoluminal repair of thoracic aortic disease. The incidence of paraplegia following endoluminal repair for thoracic aneurysmal disease is comparable to open surgery (2–5%). However, for aortic dissection the incidence of paraplegia is notably reduced following endoluminal repair when compared with open surgery (0% v 19%).

It is well recognised that in patients with thoracic aneurysmal disease the spinal cord circulation is disturbed and is often reliant on collaterals. Jacobs and colleagues have shown that the main blood supply of the spinal cord arises from lumbar and pelvic collaterals in 25%. Various techniques have been instituted in an attempt to reduce the incidence of neurological deficit following open surgery: cerebrospinal fluid (CSF) drainage, epidural cooling, pharmacotherapy, left heart bypass, motor evoked potentials (MEPs), and intercostal reimplantation. In contrast, adjunctive techniques are not used routinely during endoluminal repair of thoracic aortic disease. Simultaneous or previous aortic surgery, long segment coverage, and perioperative hypotension have been identified as risk factors for the development of paraplegia following endoluminal repair. Some groups advocate the use of prophylactic CSF drainage in high risk patients. CSF drainage for open surgery is used to reduce raised CSF pressure, which occurs on aortic cross clamping and consequently improves spinal cord perfusion. There have also been reports of successful reversal of delayed onset paraplegia with CSF drainage for both open surgery and endoluminal repair. There have been a total of 13 reported cases of paraplegia following endoluminal repair, of which 5/13 (38%) recovered with CSF drainage. Other groups have attempted to identify patients at risk of neurological complications by using retrievable stent grafts and balloon occlusion of the aorta in association with MEPs. Unfortunately both techniques increase the risk of embolism and, if positive, would mean abandoning the procedure in favour of open surgery. In patients who are fit for open surgery MEPs could be used to monitor motor function after deployment of thoracic stent grafts. If critical MEPs develop there is the option of converting to an open procedure to allow intercostals or lumbar reimplantation.

Current opinion is that it would be unethical to perform a randomised controlled trial comparing endoluminal repair with open surgery, given the notable difference in results in favour of endoluminal treatment. Hence, endoluminal repair should be offered as first line treatment for thoracic aortic disease if technically feasible. To allow further evaluation of the technique it is important that all cases are entered into the national thoracic registry.

In conclusion endoluminal repair appears to be a safe alternative to open surgery for descending thoracic aneurysms, acute and chronic type B dissection, and traumatic aortic rupture. It also has the additional benefits of being a less invasive procedure that significantly reduces the length of hospital stay. However, the durability of the stent grafts remains unknown in the long term.

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