Review Article

Strategies in the Management of Extensive Descending and Thoracoabdominal Aortic Aneurysms

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The surgical repair of descending and thoracoabdominal aortic aneurysms began in the early 1950s, and has been traditionally associated with a high mortality rate and a significant risk of paraplegia and multiple organ complications.[1] Crawford classified thoracoabdominal aortic aneurysms into four categories based on the extent of the aneurysm (Figure 1). According to this classification, extent I aneurysms originate distal to the left subclavian artery and involve the visceral arteries. Extent II aneurysms involve the entire aorta distal to the left subclavian. Extent III aneurysms involve the distal half of the descending thoracic aorta and the entire abdominal aorta, and extent IV involve the infradiaphragmatic aorta. Extents I and II are associated with the highest mortality and paraplegia rates.[2]

Treatment decisions require balancing the risk of death caused by rupture of the aorta with the risk of mortality and complications from the operation itself. Surgical results have improved significantly over the past 10 years, whereas on the other hand rupture of these aneurysms is invariably fatal.[3,4] Accurate prediction of the probability of rupture would improve the selection of patients for elective surgery, because rupture rates in patients not treated surgically are high, ranging from 21% to 74%.[5,6]

Specific risk factors that increase the likelihood of rupture have been identified from studies of the natural history of thoracic and thoracoabdominal aortic aneurysms. An individualized yearly risk of rupture can now be calculated, based on specific, easily obtainable dimensional and nondimensional variables. Selected patients whose operative risk is significantly lower than their calculated risk of rupture should be offered elective surgery.[7]

Risk factors for rupture: dimensional variables

Size and expansion rate

The aneurysm size is a major predictor of rupture. In a review of the natural history of thoracoabdominal aortic aneurysms, rupture was much more likely to occur when an aneurysm exceeded 5 cm in diameter, and the risk increased as the aneurysm increased further in size. The probability of rupture of a thoracoabdominal aortic aneurysm is 40% when the aneurysm exceeds 7 cm in diameter, and 46% of patients with large aneurysms will die within 5 years. For counseling purposes, a patient with an aneurysm exceeding 6 cm in diameter can expect a yearly rate of rupture or dissection of at least 6.9% and a yearly death rate of 15.6%. The rate of expansion of aneurysms is of

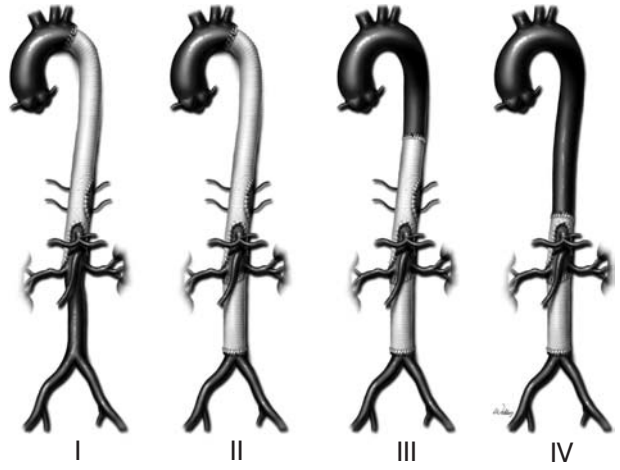

Figure 1. Drawing illustrates the Crawford classification of thoracoabdominal aortic aneurysm repairs, according to the location and extent of aorta replaced in continuity.

great importance, since a fast growth rate (greater than 0.5 cm per year) is an independent risk factor for thoracic aneurysm rupture. Elective operation eliminates the risk of rupture and restores survival to near normal.[8]

Risk factors for rupture: non-dimensional variables

Smoking/chronic obstructive pulmonary disease

In our multivariate analysis of non-dissecting thoracic and thoracoabdominal aortic aneurysms, a history of chronic obstructive pulmonary disease (COPD) increased the odds of rupture by a factor of 3.6 (p=0.04).[6]

Smoking history is also a risk factor for rupture, but COPD has eclipsed smoking in several studies in which the two were looked at in the same multivariate analysis.[6,7] Significantly more rapid growth was seen in smokers, justifying our continuing to recommend cessation of smoking in all patients with aneurysmal disease.[9]

Age

The risk of an aneurysm rupture increases with age.[1] The proportion of women with thoracic aneurysms also increases with age, almost equaling male incidence in the elderly. According to our studies the relative risk of rupture increased by a factor of 2.6 for every decade of age (p=0.02).[6]

Pain

The presence of even vague and uncharacteristic pain at the time of non-operative follow up in patients with thoracic or thoracoabdominal aortic aneurysms was significantly associated with subsequent rupture, with an odds ratio of 2.3 in our multivariate analysis (p=0.04).[6]

Hypertension

A history of hypertension is present in most patients with aneurysms, and it is widely recognized that hypertension, especially diastolic hypertension, is very highly correlated with the initial development of aneurysms.[7] Treatment of patients who have aneurysms with beta-adrenergic blocking agents and other antihypertensive agents is recommended in order to minimize the progression of their disease.[10]

Renal failure

The presence of renal failure has been identified as a risk factor for rupture of thoracic, abdominal, and thoracoabdominal aortic aneurysms.[2,5,11] In our own studies, however, renal failure has not emerged as a significant risk factor for aneurysm rupture.

Etiology: degenerative aneurysms vs. chronic dissections

Patients with known disorders of connective tissue, particularly those with Marfan or Ehlers Danlos syndrome, are likely to experience rupture more readily than other patients with aneurysms. Patients with a family history of early dissection and rupture may be particularly at risk.

In addition, evaluation of patients who experienced rupture revealed that those with chronic type B dissection had smaller maximal diameters in the descending aorta before rupture than patients with degenerative aneurysms: a median of 5.4 cm in dissections vs. 5.8 cm in patients with degenerative aneurysms (p=0.05).[6,11]

In Mount Sinai's study of the natural history of descending aortic and thoracoabdominal aortic aneurysms an equation for the probability of rupture within 1 year for each patient was generated.[7] The equation incorporates the patient's age, history of COPD, presence of pain, and the maximal thoracic and abdominal diameters of the aneurysm (Figure 2).[7] For patients with degenerative aneurysms, our current practice is to recommend operation if the calculated risk of rupture within 1 year exceeds the estimate of operative risk.

Surgical technique

All patients are placed in the standard thoracoabdomi-

Degenerative Aneurysms

$$\text{Ln } \lambda = -21.055 + 0.0093 \text{ (age)} + 0.841 \text{ (pain)} + 1.282 \text{ (COPD)} + 0.643 \text{ (desc. dia.)} + 0.405 \text{ (abd. dia)}$$

$$\text{Propability of rupture within 1 year} = 1\text{-}e^{-l(365)}.$$

Figure 2. The formula for calculating the probability of rupture of a degenerative thoracic aortic aneurysm: age is entered in years; maximal descending (desc. dia.) and abdominal diameter (abd. dia.) are entered in cm, and pain and chronic obstructive pulmonary disease (COPD) are scored as 0 if absent and 1 if present.

nal position. A double-lumen endotracheal tube is used to isolate the left lung. A right radial arterial line, a right common femoral line, and a pulmonary artery catheter are inserted routinely. Intraoperative transesophageal echocardiography is used in all patients. A spinal catheter is placed, and cerebrospinal fluid (CSF) pressure is monitored during the operation and for the subsequent 72 hours. Somatosensory-evoked potential and motor-evoked potential monitoring is utilized intraoperatively, and somatosensory-evoked potential monitoring is continued for the first 12 hours postoperatively. Distal aortic perfusion is used in the majority of the patients. Full cardiopulmonary bypass and deep hypothermic circulatory arrest is required when placement of the proximal clamp is deemed impossible. The aorta is accessed via a left thoracotomy or thoracoabdominal incision. The diaphragm is divided circumferentially. The infradiaphragmatic aorta is exposed via a retroperitoneal approach. The diseased aorta is replaced with a Dacron Hemashield graft using the sequential clamping technique. If necessary, the visceral arteries are reimplanted using a separate trifurcation graft.

Spinal cord protection: experimental model

One of the most devastating complications of surgery of the thoracoabdominal aortic aneurysm is paraplegia. In 1993, Svensson et al reported on a series of 1509 patients who underwent operations in the thoracoabdominal aorta.[13]

The overall incidence of paraplegia or paraparesis was 16% (234/1509). Significant predictors ($p<0.05$) of paraplegia or paraparesis were total aortic clamp time, extent of aorta repaired, aortic rupture, patient age, proximal aortic aneurysm, and history of renal dysfunction.

However, major improvements have been made over the past 20 years in mortality and paraplegia rates. The clinical approach to thoracoabdominal aortic aneurysm resection in our institution has been dictated by the hypothesis that spinal cord perfusion depends on an integrated collateral network that gets input from the subclavian, hypogastric and intercostal arteries, rather than the presence of a single important major spinal artery.[14] In experimental studies in pigs, changes in spinal cord blood pressure and flow, as a result of thoracic and lumbar segmental artery ligation, have been directly measured. Also, the influence of hypothermia in decreasing the metabolic demands of the spinal cord has been assessed. It is clear that extensive ligation of segmental arteries can be undertaken in the pig using spinal cord monitoring and a moderate degree of hypothermia. It has become evident that careful attention to perfusion variables is important to provide adequate blood flow to the spinal cord via the collateral network. In addition, reduction of resistance to flow in the spinal cord is optimized with drainage of CSF to maintain an intrathecal pressure of less than 10 cm H_2O.[15]

Current practice

Operative strategies such as moderate systemic hypothermia, distal aortic perfusion, CSF drainage, somatosensory- and motor-evoked potentials (SSEP/MEP), guided segmental artery ligation, and continuous spinal cord perfusion pressure measurements are key factors for spinal cord protection during these operations. Also, the utilization of SSEP and MEP monitoring allows for immediate interventions if there are perioperative SSEP and MEP changes.[16]

Distal aortic perfusion

During cross clamping of the thoracoabdominal aorta, blood can be directed into the distal aorta with the use of a Biomedicus pump. Inflow into the bypass circuit is usually via the right atrium. Outflow into the distal aorta is achieved most frequently via the left common femoral artery. Distal aortic perfusion is necessary, because the hypogastric arteries are an important source of collateral blood supply to the spinal cord. Monitoring of the femoral artery pressure is necessary to optimize distal aortic perfusion and hence cord perfusion. Generally, flow rates between 2.0 to 3.0 L are necessary to achieve a distal mean arterial pressure between 60 and 80 mm Hg. Several publications have documented the efficacy of distal perfusion in reducing paraplegia fol-

lowing resection of extensive thoracoabdominal aortic aneurysms.[17,18]

At the same time, maintaining selective perfusion of the visceral arteries may have a beneficial effect in preventing renal insufficiency, as well as liver and intestinal injury, which can potentially lead to severe coagulopathy and multisystem organ failure.[19,20]

Drug use

Almost all clinical techniques for resection of thoracic and thoracoabdominal aneurysms involve some unavoidable intervals of cord ischemia. It appears that the tolerance of the spinal cord to ischemia can be increased by the perioperative use of corticosteroids. Mannitol and naloxone may have a beneficial effect.[21]

Passive hypothermia

Moderate hypothermia (32˚) has been shown to increase the tolerance of the spinal cord to ischemia from 20 to 50 minutes in pigs,[22] by decreasing the metabolic demands of the spinal cord. Longer ischemic intervals can be tolerated at lower temperatures, as evidenced by the protective effect of profound hypothermia on the spinal cord during repair of extensive thoracoabdominal aortic aneurysms.[23,24]

CSF drainage

Invariably, we drain 10 cc of CSF every hour, in an effort to keep the CSF pressure below 10 mmHg. The use of CSF drainage has been shown to decrease postoperative spinal cord ischemia. On the other hand, excessive CSF drainage may create significant central nervous system complications.

Spinal cord monitoring

Changes in somatosensory- and motor-evoked potentials (SSEP/MEP) can be a sensitive indicator of spinal cord ischemia during repair of descending and thoracoabdominal aortic aneurysms.[25] Continuous monitoring of the spinal cord function during these operations and in the immediate postoperative period is essential in reducing the risk of neurologic injury.[26] In most cases, cord ischemia, detected by MEPs or SSEPs or both, during the surgical procedure can be corrected by raising the arterial pressure or decreasing the CSF pressure. In selective cases reimplantation of intercostal arteries may be needed.

Also, monitoring of the spinal cord perfusion pressure by directly cannulating the L1 segmental artery allows for continuous perioperative assessment of the adequacy of the collateral network.

Segmental vessel reimplantation

Most aortic surgeons advocate reimplantion of segmental vessels, aiming to reduce the incidence of spinal cord injury. This appears to be physiologically sensible. However, in a recent series of 100 cases of thoracic and thoracoabdominal aortic aneurysm resection, in which we ligated all segmental vessels originating from the aneurysm sac before opening the aorta, the incidence of paraplegia was only 2%.[16] The perioperative use of SSEP/MEP monitoring can identify the few patients that are very dependent on the segmental arteries for spinal cord blood flow. In these cases, reimplantation of T8-L1 segmental arteries using a separate graft can be accomplished very easily.

According to our recent experimental data, metabolic and hemodynamic manipulation should enable routine sacrifice of all segmental arteries without spinal cord injury.[27] This is very pertinent in view of the use of stent grafts in the treatment of thoracic aortic aneurysms.

Postoperative hemodynamic stability

Perfusion of the spinal cord through the collateral network is enhanced by high perfusion pressures. We insist that the mean arterial pressure is maintained between 85-95 mmHg during the immediate postoperative period. Oxygen delivery is maximized, with maintenance of normal hematocrit levels and careful attention to ventilation and oxygenation of the patient. During descending and thoracoabdominal aortic aneurysm repair, large volumes of fluid are routinely administered in order to maintain an adequate preload and avert possible spinal cord ischemia.[28]

Spinal cord injury can occur not only during the aneurysm repair but also in the postoperative period, as a result of inadequate spinal cord perfusion caused by systemic hypotension. Delayed paraplegia can perhaps be prevented with better hemodynamic and fluid management.

If it is detected early, salvage of the spinal cord can be achieved by raising the systemic pressure and placing a spinal catheter for prompt CSF drainage.

Implications for endovascular treatment

Endovascular stent-grafting is a new treatment option for patients with thoracic aortic pathology for whom standard surgical reconstruction is prohibited because of many comorbidities or is associated with expected significant risks. Rapidly expanding experience during the last decade has allowed the method to evolve, and many details concerning optimal approaches and technical strategies have been published in the literature. Numerous reports have described the use of these techniques for a variety of conditions, including degenerative aneurysms, traumatic injuries, acute and chronic type B dissections, aortoesophageal and aortobronchial fistulas, mycotic aneurysms, and penetrating aortic ulcers.[29] Successful stent-grafting of patients with acute type B aortic dissection was first reported by Dake and colleagues[30] from Stanford in 1999; Nienaber and colleagues[31] simultaneously reported their results in patients with subacute and chronic type B dissection. The main concept of endovascular therapy is that the coverage of the area of the primary intimal tear with a stent graft would promote false lumen thrombosis and subsequent aortic remodeling by eliminating antegrade or retrograde flow into the false lumen. Endovascular treatment of descending and thoracoabdominal aortic aneurysms has the benefits of reducing periprocedural complications while expanding the number of patients who can be offered treatment. The main differences include obviating the need for aortic cross-clamping, avoiding general anesthesia, and avoiding thoracic or thoracoabdominal incisions in this frequently debilitated population with many comorbidities.[32] The feasibility of stent-grafting descending thoracic aortic aneurysms has been established for various pathologies, but the indications for intervention are not yet clear. The indication for stent-grafting of a descending thoracic aortic aneurysm at the present time should be based on a predicted operative risk that must be lower than the risk of either conventional open repair or optimal medical management. Several recent studies have demonstrated that operative mortality is between 2% and 26% and depends largely on urgency, the extent of comorbid conditions and the experience of the operator.[33] Despite reasonably low early operative morbidity and mortality, late complications, including endoleaks, graft migration, stent fractures, and aneurysm-related death, are much more common than those reported for the gold standard procedure, open aortic surgery.[34,35] On the other hand, the long-term durability of endovascular grafts is still unknown.

The Stanford group considers the most suitable pathologic target for successful thoracic aortic stent-grafting to be lesions that were relatively localized, including penetrating aortic ulcers, anastomotic pseudoaneurysms, mycotic aneurysms, and false aneurysms due to chronic aortic transections. The first long-term results that have been published after endovascular stent-graft repair of descending thoracic aortic pathology have raised concern about suboptimal patient survival, freedom from aortic rupture, and the worrisome prevalence of early and late stent-graft–related complications. Late aortic complications occur in a substantial proportion of stent-graft patients, emphasizing the importance of serial imaging surveillance. This risk has been reduced by the introduction of second-generation commercial stent-graft devices and better patient selection criteria, but still represents the leading drawback to endovascular stent-graft treatment.

Endovascular treatment of thoracic and thoracoabdominal aortic aneurysms usually precludes segmental vessels within the stented segment from contributing to spinal cord blood supply. Stent grafts result in sudden complete occlusion of a large number of segmental vessels under normothermic temperatures.

The report by Greenberg et al[36] of neurologic deficit attributable to spinal cord ischemia in 3 out of 25 patients (12%) undergoing endovascular thoracoabdominal aortic exclusion showed long segment thoracic aortic coverage to be a significant risk factor in predicting clinically evident spinal cord ischemia. On the other hand, delayed-onset spinal cord ischemia has also been reported, and has been associated with the presence of periprocedural hypotension. This finding suggests that a marginal collateral supply after endografting may be vulnerable to postoperative hemodynamic compromise.

A recent review found a 2.7% incidence of spinal cord injury after endovascular repair of all or a portion of the descending thoracic aorta, which is certainly no better than current results of open aneurysm resection.[37] Because delays in the onset of the neurologic deficit have been encountered after endovascular thoracoabdominal aorta repair, careful follow-up with neurologic examination and institution of cerebrospinal fluid drainage upon the first signs of deficit may positively affect the outcome.[38]

In conclusion, current advances with stent-graft thoracic intervention must be viewed as exciting new developments that offer hope to many patients with

thoracoabdominal aortic aneurysm and other serious conditions affecting the thoracic aorta. Early and midterm results demonstrate a significant advantage over open surgical repair in strictly selected patients, with many comorbidities that preclude them from open surgical repair and with specific pathologies of the thoracic aorta. Further long-term prospective randomized studies are needed to confirm a potential advantage of these techniques over the traditional gold standard open repair.

Improved results with these techniques

Between June 2002 and June 2005, 219 patients underwent descending (n=79, 36%, 23 elephant trunk completions) and thoracoabdominal aortic (n=140, 64%) aneurysm repair (Table 1). The mean age of the patients was 63.8 years (range, 18 to 88). Preoperative risk factors included a history of hypertension in 212 patients (97%); a history of smoking in 110 patients (50%), with chronic obstructive pulmonary disease (COPD) in 53 (24%); a history of cerebrovascular accident in 34 patients (16%); chronic renal insufficiency in 27 patients (12%), with preoperative need for hemodialysis in 8 (4%); and insulin-dependent diabetes mellitus in 24 patients (11%). In all, 144 patients (66%) underwent elective operation owing to enlargement of their known aneurysm, 34 patients (16%) had urgent operations, and 41 patients (19%) underwent emergent repairs owing to ruptured aneurysms. The classification of the thoracoabdominal aortic aneurysm was as follows: Crawford I (52%), II (10%), III (11%), IV (7%). The overall in-hospital mortality was 5.9% . The mortality for descending thoracic aneurysm repair was 5.1% (4 of 89), and for thoracoabdominal aortic aneurysm repair 6.4% (9 of 140). Only 4 patients developed postoperative paraplegia (1.8%). Postoperative myocardial infarction occurred in 5 patients (2%), and thirteen patients (5.9%) suffered a postoperative stroke. Sixty patients (27%) experienced respiratory complications with prolonged postoperative ventilation (longer than 48 hours); 24 required tracheostomy (11%). Eight patients had renal failure requiring postoperative dialysis (3.7%).[39]

Conclusions

Operations in the descending and thoracoabdominal aorta can be performed with acceptable mortality and morbidity. This requires a multidisciplinary approach to the operative and postoperative care of these patients. Organ-specific protective measures should be undertaken to prevent postoperative complications. In addition, calculation of the risk of rupture of the aneurysm

Table 1. Postoperative complications and hospital stay, stratified by institution.

Postoperative complication	Number (%) / Median (interquartile range)		
	Total (n=219)	Mt Sinai (n=130)	Montefiore (n=89)
Respiratory:			
Ventilation >48 hours	27%	23%	33%
Reintubation	15%	7%	27%
Tracheostomy	11%	11%	11%
Vocal cord paralysis	11%	5%	19%
Pneumonia	14%	7%	24%
ARDS	2%	2%	2%
Cardiac:			
Myocardial infarction	2%	1%	4%
Bleeding:			
Requiring rethoracotomy	8%	8%	8%
Renal insufficiency:			
Creatinine >2.5 mg/dL	9%	7%	11%
Requiring temporary dialysis	4%	4%	4%
Neurologic dysfunction:			
Stroke	6%	5%	8%
Paraplegia	2%	2%	2%
Hospital stay (days)	11 (6-35)	11 (6-29)	11 (5-40)

ARDS – adult respiratory distress syndrome.

based on specific variables is very important in order to be able to identify the patients who will benefit the most from these extensive operations.

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