**REVIEW**

**Aortic Dissections: New Perspectives and Treatment Paradigms**

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Aortic dissection is a complex manifestation of disease of the arterial wall. The severity and consequences of a dissection are related to the physical characteristics and anatomic location of the tear as well as the underlying patient physiology. Despite in vitro and in vivo modeling advances, our understanding of the pathophysiology has been limited to evaluations of the success and failure of various treatment modalities. The indications for intervention have historically included rupture, intractable pain or hypertension, distal ischemia and degeneration of the aortic wall causing aneurysm formation. The management decisions for patients with dissections are dependent upon the abnormal anatomy, the acuity of the patient presentation, and physiology. Despite the availability of open surgery as a therapeutic option, acute dissections with evidence of ischemia are now handled using an endovascular approach that is specifically directed at the cause of the ischemia. Endovascular treatments include the placement of a stentgraft into the proximal aorta, branch vessel stenting, uncovered stent placement in the abdominal aorta, and aortic fenestrations. Chronic dissections, in contrast, are still most frequently managed with open surgical techniques. However, a subset of patients that are not candidates for traditional surgical repair of the thoracoabdominal aorta may be managed with a combined open mesenteric revascularization with subsequent endovascular grafting of the thoracoabdominal aorta.

**Key Words:** Aortic dissections; Thoracic aneurysm; Thoracic dissection; Endograft; Endovascular fenestration; Thoracic endovascular repair.

**Introduction**

Stanford type B aortic dissections result in an annual mortality rate in excess of that reported for ruptured aneurysms. Despite the plethora of treatments that have been advocated over the past 80 years, the consequences of this disease remain devastating. Deaths occur as a result of end organ ischemia or aortic rupture. Acute mortality has been most closely associated with ischemic complications, while long-term mortality is traditionally linked to aortic degeneration and late rupture. Ischemic complications have been reported to occur in up to 30% of all cases of type B dissections.1–5 Rupture, on the other hand is less common in the acute setting but still occurs in up to 20% of patients over their life-span. Although a great deal of progress has been made since the inception of the management of aortic dissection clinical sequelae in 1935,6 contemporary reports still harbor exceptionally high mortality rates in the subset of patients that suffer ischemic complications (16–25%).3–5,7

Open surgical techniques include central or focal descending thoracic aortic replacement, diffuse aortic replacement (thoracoabdominal repair), and open fenestration techniques. Interventional techniques include visceral vessel stenting, aortic fenestration, and the use of covered or uncovered stents in the aorta. Although many therapies have attempted to address the acute circumstances the disease creates, the underlying pathophysiology of the disease remains confusing and complicates outcome analysis. Most treatments are viewed as partially effective, and leave clinicians with several questions regarding the appropriate management in the acute and chronic settings.

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Materials and Methods

Papers and studies for this review were identified by Medline and Pubmed literature searches using key-words aortic dissection, type B dissection, visceral ischemia, thoracic endograft, and aortic fenestration. Articles were obtained and evaluated by the authors of this manuscript. Inclusion in this review was dependent on the relevance to the subject, and the ability to discern the acuity of patient presentation, symptoms, and the presence of dissection as opposed to aneurismal disease. In an effort to provide a comprehensive treatment paradigm for this complex patient population, we have included data from our institution as well in this review.

Pathophysiology

An understanding of the pathophysiologic mechanisms of an aortic dissection has the potential to help direct therapeutic options. Unfortunately, despite several studies assessing anatomic and physiologic variants pertaining to dissected aortas including tear depth,8 local wall stress,9 the status of the vaso vasorum,10 and the angulation of the initial entry tear.11 We have been largely unsuccessful in defining specific anatomic parameters predictive of acute or long-term complications from aortic dissections,8,9,12 – 14 Attention to physiologic details, pulse examinations, subtle symptoms, and serum markers indicative of low grade ischemia will allow physicians an opportunity to intervene prior to hemodynamic decompensation and the potential for end organ tissue loss. However, certain generalizations can be made about dissections. Anatomic categorization coupled with physiologic information is most helpful in determining treatment options and predicting outcomes. Patients with acute dissection presenting with ischemic complications have fared worse than those with relatively minimal symptoms such as back pain or hypertension. Chronic dissections typically have complex true and false lumen relationships; most frequently include extensive distal aortic or iliac involvement. Untreated type A dissections have been universally associated with dismal outcomes, while only a subset of type B dissections result in complications that mandate acute treatments. Contemporary reports must be viewed in the context of the indication for treatment, the pathophysiology of the involved patients, the type of treatment performed and the endpoints assessed. For example, a patient in extreme duress with mesenteric ischemia may be considered successfully managed if they survive to hospital discharge, while a patient treated for a chronic dissection in the setting of an enlarging aorta would be deemed successful if growth was arrested or the aneurysm excluded with false lumen thrombosis.

Acute dissections

Acute dissections originating distal to the left subclavian artery without arch involvement have been historically treated medically unless specific symptoms, such as ischemia, rupture or rapid growth are detected. In the absence of such, antihypertensive therapy with beta blockade or combination alpha and beta blockade has been the primary method of minimizing the long-term complications. The development of an ischemic syndrome is the most frequently encountered morbidity and requires rapid treatment to prevent mortality. Unfortunately, conventional treatments in these circumstances have been associated with mortality rates up to 80%. Newer endovascular methods have been noted to markedly diminish the mortality rates for this condition, but lack long-term results and have been reported in series with small numbers of patients.

Mechanisms of ischemia

The lack of an early marker or indicator of which patients with aortic dissections will suffer a catastrophic complication such as visceral ischemia has thwarted our ability to treat patients prior to the development of extreme physiologic duress. Two pathophysiologic mechanisms of ischemia have been proposed.12,13,15,16 The first involves compression of the true lumen within the descending thoracic aorta. This process occurs, most commonly, in the setting of a deep proximal tear with an absence of a large distal fenestration. The mean false lumen pressure increases rapidly, as a result of poor outflow, which in turn results in true lumen compression that ultimately inhibits flow to the viscera and lower extremities. If a large distal fenestration is present, the false lumen acts in a manner similar to that of a shunt, thus distal ischemia is rarely noted because flow is not compromised. If the dissection plane propagates into a visceral or lower extremity branch, individual branch ischemia may be noted. Although many branch vessel dissections are without sequelae, two types of symptomatic branch vessel obstructions can develop. The obstruction can result from a false lumen that has occluded where thrombus resides at the ostium of the branch vessel, or the flow impingement can occur if
the dissection flap functions similar to a mechanical valve oscillating with the pulsatile aortic flow, occluding the visceral vessel ostium during systole, and allowing flow during diastole.\(^{15}\)

**Interventional strategies**

The hemodynamics of aortic dissections must be viewed in the context of cardiovascular physiology and the material properties of the aorta. The inability of an uncovered stent to direct flow away from the false lumen relegates any benefit of such a stent to presence of radial force. The amount of radial force required to collapse the false lumen, that typically has a mean arterial pressure in excess of the true lumen, may be greater than the aortic wall strength. It is furthermore difficult to distribute the force equally along the aortic adventitia in the setting of a dissection with irregular flap morphology or tortuosity that compounds the problem. Animal studies,\(^{17-19}\) as well as early clinical experiences have reinforced this concept. Aortic fenestration techniques have a role in select cases. This method of therapy functions by equalizing blood pressure differentials between the true and false lumen, allowing passive equalization of flow channels. Short-term success with observed resolution of ischemia of the mesentery or lower extremities has been reported.\(^3,20\) However, it is unlikely that this technique will aid in the prevention of long-term aortic degeneration, given the persistence of flow within both lumens and the obligatory diminished wall thickness of each portion of the aorta. It appears that the best management strategy would be to minimize flow and pressurization of the false lumen to treat proximal obstruction and also to prevent aortic enlargement over time.

**Management of acute dissections with ischemic complications**

The assessment of the patient suffering complications from an acute aortic dissection must entail a careful evaluation of both the true and false lumens. Treatment must be directed in a manner that accommodates the complex hemodynamics, luminal relationships, and anatomic points of luminal communication as well as branch vessel flow. The choice of therapy must center on the degree of true lumen compression and the patency of the false lumen (Fig. 1). In the setting of true lumen compression with dissection into branch vessels, critical branches may be protected, if time permits, by the placement of self-expanding stents within them to ensure true lumen supply of blood to the end organ. This would be followed by the placement of an endovascular stent graft proximally over the major fenestration in an effort to block false lumen inflow, allowing for passive contraction of the false lumen, resulting in true lumen expansion. In the absence of branch vessel dissection, placement of the aortic prosthesis usually suffices. These techniques carry the potential to provide the majority of end organs with flow derived from the true lumen. Endovascular fenestration of the aorta is performed only when definitive access to the true lumen cannot be achieved or one is unable to ensure adequate flow to branch vessels following placement of an endovascular prosthesis. However, passive expansion of the true lumen and contraction of the false lumen will occur only in the setting of a patent false lumen. When false lumen occlusion is observed and confirmed, true lumen expansion can be accomplished with the use of flexible uncovered stents, allowing stent placement across visceral ostia. This can be accomplished with the use of a relatively small amount of radial force, as compression of the thrombosed false lumen is quite simple in the absence of the relatively high mean arterial pressure typically found in a patent false lumen. This must be carefully assessed because if there is false lumen perfusion, the pulsatile forces induced by the false lumen transmitted to any stent through the dissection membrane are likely to overcome any amount of opposing radial force, and severely challenge the devices from the standpoint of material fatigue.

**Chronic dissections**

Unlike the emergent situation encountered when intervening on acute dissections, the indication for intervention in chronic dissection generally allows for a well-planned, elective approach. Given the invasiveness of traditional thoracoabdominal repair resulting from chronic dissections, there is a strong desire to develop other treatment methods. However, it is rare that the dissected aorta has anatomy allowing for a proximal and distal seal with an endovascular prosthesis. More typically, the dissection extends into the visceral aortic segment where multiple fenestrations between the two lumens exist. These preclude complete exclusion of the false lumen with an endovascular approach and it remains unclear whether partial exclusion of the false lumen will affect aortic growth or rupture in a beneficial fashion. In this light, we treat patients with an open surgical approach if they are acceptable candidates. However, in some
circumstances, such as patients with severe pulmonary disease, cannot tolerate the thoracic portion of the procedure. In these cases a four vessel mesenteric bypass deriving inflow from the left common iliac artery can be performed through a retroperitoneal incision extending to the 12th rib. This avoids a thoracotomy, theoretically diminishing the pulmonary risks and precedes stentgrafting of the entire thoracic and abdominal aorta (Figs. 2 and 3). Obviously, the development of a branch vessel endovascular device would supplant this fairly cumbersome and relatively invasive combined approach. However, the use of a branch vessel device in a dissected aorta may prove to be significantly more challenging than its use within a non-specific thoracoabdominal aneurysm.

**Discussion**

Aortic dissection treatment paradigms have evolved with the development of new endovascular tools and the better understanding of the pathophysiology of this disease. These new paradigms have been reported by experienced centers involved in the treatment of aortic dissections, where surgeons, radiologists, and cardiologists are incorporated in a team approach optimizing the management of these patients. Prior to embarking on interventional treatments for aortic dissections, extensive experience with endovascular techniques, especially aortic endografting, is viewed as mandatory owing to the complex nature of the required interventions and unforgiving aspects of the disease state.

No definitive evidence is available demonstrating the superiority of endovascular treatment in contrast to open surgical techniques for acute dissections presenting with life-threatening complications (ischemia, aortic rupture). However, in this subset of patients, endovascular treatments performed by experienced teams have shown more favorable outcomes than open surgical series that report mortality rates between 50 and 85%. Much of the endovascular success can be attributed to the minimally invasive aspect of the treatments in conjunction with the rapidity by which distal aortic perfusion is restored.
Dake et al. reported a series of 19 acute dissections treated with thoracic stent-grafts (4 type A and 15 type B), 11 with symptomatic branch-vessel obstruction, and three with aortic rupture. There were three early mortalities (16%), and three patients with serious early complications. After stent-graft placement, all branch-vessel obstruction resulting from true lumen compression, and 40% of those obstructions attributed to a combination of true lumen compression and branch involvement were reperfused. Additional uncovered stents were required in the true lumen of the branch vessels with residual obstruction to restore luminal patency. Kato and Shimono published results on a series of patients with thoracic endografts placed for acute dissection (16 with complications, 8 without), and chronic dissections (n = 13). Acute dissection treatments were limited to hemodynamically stable patients with only one early mortality in this group. Palma et al. published a series of 35 acute (without branch vessel ischemia) and 23 chronic type B dissections out of a series of 65 patients treated with thoracic endografts. They reported an early mortality rate of 6%.

Disparate mortality rates likely pertain to patient selection bias and the acuity or pretreatment physiologic status of the patients in the respective series. Acute reperfusion injuries and multisystem organ failure following multiple laparotomies with bowel resections have been responsible for the majority of deaths. The reperfusion injuries we have witnessed have been dramatic and isolated to patients with severely compressed true lumens. Severe problems have arisen from hyperkalemia, and subsequent patients have been treated by maximizing medical prophylaxis in such cases, and in one case, utilized intraoperative hemodialysis prior to endograft deployment.

The need for visceral vessel stenting in conjunction with endovascular grafting has also become somewhat controversial. Some authors have felt that adjunctive stents within dissected branch vessels are unnecessary. However, following three cases where we were unable to regain access to vessels supplied by a false lumen that was collapsed by a proximal endovascular graft, have become more aggressive in the establishment of true lumen inflow to branch vessels supplied by the false lumen. Ultimately, the decision as to whether a branch vessel is stented prior to the placement of an endovascular prosthesis is dependent on the time required to perform such a procedure, the effect of delayed true lumen expansion, and the risk of branch vessel closure. Fig. 1 depicts the treatment paradigm utilized at our institution in the setting of a patient suffering acute dissection with ischemia complications.

Traditionally, asymptomatic dissections are managed with aggressive antihypertensive regimens and careful follow-up. However, long-term data suggest that up to 30–40% of these patients will ultimately succumb to an aortic-related death or require a direct aortic intervention over a 7-year period. Consequently, we must carefully evaluate any mechanism that may diminish the rate of aortic growth. Treatment at the time of acute presentation appears to be most promising. Initially, the dissection flap is free of hypertrophy and extremely mobile. Success in achieving complete false lumen thrombosis may be most probably at this time point. Were this to occur, the acute dissections would be converted into an intramural hematoma, which has the potential to heal and decrease the incidence of long-term aortic degeneration. Proof of this concept would require a prospective randomized trial involving asymptomatic dissections where endovascular grafting of the proximal fenestration would be contrasted with a medically managed arm.

In the setting of a chronic dissection, complete and durable exclusion of the aneurysmal false lumen, and restoration of the blood flow into the true lumen,
should be achieved to consider treatment success. Because of the thickness of the intimal flap, the presence of multiple re-entry tears, and flow to aortic branches provided by the false lumen, endografting in our experience has limited indications. However, good results of endovascular exclusion have been reported in selected patients. Nienaber et al. reported a series of 12 cases of aortic sub-acute or chronic dissection treated with endografts. The indications for treatment were a maximal aortic diameter of 5.5 cm or more, luminal expansion, and recurrent pain. No patients with visceral branch-vessel obstruction were included in this study. At 3 months, complete thrombosis of the false lumen was documented in all patients, with clear evidence of expansion of the true lumen and shrinkage of the false lumen. In Kato and Shimono’s series, 13 chronic dissections with major branch vessels supplied by the true lumen were treated with endografts. During follow-up (27 months), complete obliteration of the false lumen was noted in 38.5% of patients with chronic dissection, compared to 70% of patients with acute dissections. This last series confirms the difficulties to definitively exclude the false lumen in patients with chronic dissection treated with endografts. We therefore utilise a conventional open surgical approach if the patient has acceptable risk factors. In patients perceived to be incapable of tolerating a traditional repair, a combined endovascular and open procedure involving visceral and renal bypass grafting with thoracoabdominal stentgrafting has been performed (Fig. 4).

The treatment paradigms sited in this manuscript incorporate the majority of the recently published series combined with our own experience. The use of endovascular techniques in the setting of acute aortic dissections associated with peripheral ischemia or
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Chronic Dissection

Aortic Growth
- >5.5cm maximum diameter
- >5mm annual growth
- or Rupture or Ischemia

Yes

Non-surgical Candidate

Surgical Candidate

Anatomy not Amenable to Endovascular Repair

Anatomy Amenable to Endovascular Repair

Endovascular Graft

Medical Rx

Combination Open Endovascular

Serial Imaging

Fig. 4. This treatment paradigm represents our approach to patients with chronic dissections. In contrast to patients with acute ischemic syndromes resulting from aortic dissections, chronic dissections are more frequently managed with an open surgical approach unless they are deemed to be unfit for open surgery. In those select circumstances, a combined open mesenteric or brachiocephalic bypass procedure is performed prior to endovascular grafting, or the patients are simply managed medically.

References


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