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Acute complicated and uncomplicated Type III Aortic Dissection: An endovascular perspective

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Abstract

Type III aortic dissection is associated with high morbidity and mortality. There is a shifting paradigm in the treatment of complicated and uncomplicated acute Type III aortic dissection towards earlier endovascular repair. In this review, the authors present the current perspective on the endovascular management of acute complicated and uncomplicated Type III aortic dissection.

Keywords

Endovascular; Acute Type III Aortic Dissection; Aneurysm; Complicated; Uncomplicated; Review

INTRODUCTION

The first report of an aortic dissection was in the 1800s by Shekelton when he noted an obliterated false lumen of a healed dissection.¹ The misnomer *aneurysme dissequant* or ‘dissecting aneurysm’ was subsequently coined by Laennec in 1826.² Thereafter, the term *acute aortic dissection* has undergone numerous refinements in definition and is a commonly recognized catastrophe of the aorta today. The pathognomonic lesion is an intimal tear resulting in blood dissecting into the medial layer of the aortic wall. Aortic dissections are thought to begin where shear force ($\Delta P/\Delta T_{\max}$) and pressure fluctuations along the aortic wall are the greatest. The original description of a distal (Type III) aortic dissection according to DeBakey originated at level of the left subclavian artery.³ Type IIIa aortic dissections are confined to the descending thoracic aorta, while Type IIIb aortic dissections extend into the abdominal aorta. This classification was further refined by Daily with the introduction of the Stanford schema in 1970 (Figure 1).⁴ Distal aortic dissections that limit blood flow to the viscera and the lower extremities are frequently fatal. According to the International Registry of Acute Aortic Dissection (IRAD), the mortality from distal (Type III) dissections is 27.4%.⁵ Patients with “complicated” dissections who have evidence of malperfusion or rupture had a 31.4% mortality, while those who had “uncomplicated” dissections treated medically had 10.7% in-hospital mortality.⁵

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An *acute aortic dissection* has long been classified as one that occurs within 2 weeks of symptom onset, while chronic dissections are those that persist greater than 2 months following symptoms. These long accepted definitions, however, have been called into question as our understanding of this disease is evolving. Recently, Kasirajan and colleagues suggested that the term *acute aortic dissections* include the first 3 months of disease from the onset of symptoms, *sub-acute aortic dissections* have symptoms persist from 3 through 12 months, and *chronic aortic dissections* have symptoms persist for >1 year.⁶ This suggested classification is based on the behavior of the aortic septum as the disease progresses, from dynamic (acute), to compliant (sub-acute), and eventually non-compliant (chronic).

EPIDEMIOLOGY

Aortic dissections affect 5 to 30 cases per million people each year.^{7, 8} Men typically are affected 5 times more frequently than women.⁵ The incidence of Type III aortic dissections peaks in the 6th and 7th decade of life.⁷ Aortic dissections follow a circadian and a seasonal chronobiologic pattern with the highest occurrences noted in the fall, winter, and spring with the most frequent events reported during the early morning hours.⁹ Based on the established risk factors for aortic dissection, Marfan syndrome, hypertension, pre-eclampsia and cocaine abuse are special groups that have been exclusively identified for their unique predisposition to aortic dissection. A comprehensive list of risk factors for aortic dissection is listed in Table 1.

PATHOPHYSIOLOGY

Aortic dissection occurs due to a defect in the intimal and medial layers of the aorta allowing blood to propagate within the arterial wall creating a false lumen. The elasticity and mobility of the aorta is challenged by the force ($\Delta P/\Delta T_{\max}$) at a local area leading to an intimal tear.^{10, 11} The stress on the aortic wall during a cardiac cycle is greatest in the ascending and the proximal descending thoracic aorta. As such, the most common patterns of a Type III aortic dissection begin at a tear within 2 cm of the left subclavian artery, extending through the thoracoabdominal aorta with the right renal artery and visceral trunks originating from the true lumen and the left renal artery rising from the false lumen.^{12–15}

Patients with aortic aneurysms are predisposed to dissections with evidence of medial degeneration. Much of our understanding of the pathophysiology of aneurysms stems from several murine models.¹⁶ Most relevant, mice with a defect in fibrillin (so-called Marfan mouse) develop aortic root dilation and dissection.¹⁷ Rapid expansion in this model and others is believed to progress as a result of proteolytic enzymes and inflammatory mediators that are produced in the diseased aorta.

NATURAL HISTORY

Patients with acute Type III aortic dissection should be classified by their anatomic sequelae from the dissection and are stratified as complicated or uncomplicated. A “complicated” Type III aortic dissection is defined as one that causes malperfusion of the viscera, kidneys or lower extremities, or as evidenced by rupture and occurs in 25–40% of cases.^{18, 19} The majority of patients do not have these complications at presentation, and are designated “uncomplicated.” The main factors that establish the degree of malperfusion are: (I) the proportion of the aortic circumference that is dissected, (II) the extent of distal reentry from the false lumen into the true lumen, and (III) the topography of the ostia of the visceral branches off the true lumen.^{18, 19} Back pain, acute mesenteric ischemia, cerebrovascular incidents, renal failure and spinal cord ischemia are commonly associated with Type III aortic dissections. For those patients who do not undergo immediate surgical intervention, dissected aortas tend to expand at variable rates ranging from 2.1–15.4mm/year.

There is evidence that a subgroup of patients with acute uncomplicated aortic dissection are at higher risk of developing late aortic complications including aortic dilation.^{20, 21} Specifically, one study demonstrated that >50% patients with a patent false lumen and an aortic diameter ≥ 40 mm had a dissection related death, rupture, malperfusion or late aneurysmal dilation within 5 years ($p=0.04$).²² This risk factor was confirmed in another retrospective review of 141 patients, 42% of which had a late aortic event.²³ In this study, false lumen ≥ 40 mm was highly predictive of late complications ($OR=3.18$).²³ In another study of 100 patients, aortic diameter was not associated with late complications but rather false lumen diameter ≥ 22 mm at the proximal descending aorta.²⁴ Patients with false lumen diameter ≥ 22 mm had higher mortality (17% vs. 5%, $p=0.09$) and late aneurysmal formation (42% vs. 5%, $p<0.001$) compared to patients with a <22mm initial false lumen.²⁴ Based on these retrospective studies, there may be a subgroup of patients at highest risk of complications for dissection.

DIAGNOSIS

Computed tomographic aortography (CTA) is the gold standard for diagnosing aortic dissections and has supplanted other imaging tests.^{5, 25} CTA is rapid, widely available, and is highly sensitive (98–100%) and specific (95–98%).²⁶ A dedicated CTA protocol with 2.5–5.0mm section collimation and 1.5–2.5mm spacing during 120–200ml iodinated contrast administration produces optimum aortic definition, and allows for high resolution anatomic detail and reconstructions.²⁷ Transesophageal echocardiography (TEE) administered by experienced operators can be a rapid and highly sensitive test to diagnose an aortic dissection, but is limited by poor visualization of the descending aorta distal to the diaphragm. In spite of these limitations, TEE is the second most utilized diagnostic modality, especially given the ability to diagnose an acute dissection rapidly in an unstable patient.²⁵ Additionally, magnetic resonance angiography (MRA) with a sensitivity of 95–100% and specificity of 95–98% is an alternative diagnostic imaging modality, but requires availability and patient stability.²⁵ Currently, most diagnostic algorithms de-emphasize the role of catheter based aortography due to the lower sensitivity and catheter related risks. Intravascular ultrasound (IVUS), an evolving technology, has demonstrated 100% sensitivity and specificity during endovascular diagnosis and treatment of complicated acute Type III aortic dissection.²⁸

TREATMENT OPTIONS

Complicated acute aortic dissection requires urgent or emergent intervention by an open surgical or endovascular approach (Figure 2). Patients with uncomplicated acute aortic dissection have typically been treated with medical therapy. The advent of endovascular therapy is challenging the management of complicated and uncomplicated acute aortic dissection, and offers an opportunity for improved outcomes and definitive treatment of this disease.

Medical management of uncomplicated acute Type III aortic dissection

In the absence of radiographic deterioration of the aorta or evidence of malperfusion, intense antihypertensive therapy has long been accepted as the initial management of patients with Type III uncomplicated aortic dissection (Table 2).²⁹ Preexisting hypertension is seen in 70% of patients with Type III aortic dissection compared to only 25–35% in patients with Type I or II aortic dissections. The overall goal in the acute management of dissection is blood pressure reduction and minimizing $\Delta P/\Delta T_{\max}$ on the aortic wall. Close observation should be performed in an intensive care unit with systolic blood pressure regulation <140mmHg, minimizing the risk of a progressive dissection while preventing low flow states to the central nervous system and viscera.³⁰ Ideally, the systolic blood pressure should be titrated to 90–110mmHg as tolerated by the patient. Beta-blockade, with esmolol or labetalol, should be the initial therapy to directly decrease $\Delta P/\Delta T_{\max}$. Once maximal beta-blockade has been achieved, secondary

agents like nitroprusside, nitroglycerin, fenoldopam, and/or calcium channel antagonists may be administered for enhanced control of hypertension. Management of patients with uncomplicated Type III aortic dissection remains contentious. Despite aggressive medical management patients remain at a risk for late aneurysmal dilatation, with 30-day mortality ranging from 1.6% in some series to 30% in others (Table 2). Late rates of aortic dilation requiring intervention exceed 40% in some series.²⁴ Collectively, these studies highlight the high long term mortality and morbidity rates for medical therapy, despite its accepted practice, for uncomplicated Type III dissections.

Open surgery for complicated acute Type III aortic dissection

Indications for an operation or intervention in acute Type III aortic dissection include: (I) a rapidly enlarging aortic diameter (aneurysmal degeneration), (II) progression of the dissection, (III) aortic rupture, (IV) malperfusion syndrome, and (V) persistent or recurrent intractable pain.^{21, 31, 32} Many centers argue that pain is a soft indication for an operation in Type III aortic dissection, with only 4% of patients with recurrent pain developing complications.³³ The method of open surgical repair typically involves identifying the entry tear and replacing the involved dissected aorta with a graft sewn to the true and false lumens. Pledgets and/or biologic glues are often used to reinforce the suture lines to help obliterate the false lumen. Open surgery in the acute setting is not without significant risk even at experienced centers. Baylor University (Houston, TX) reported a 22% operative mortality in their series of patients who underwent early surgical intervention.³⁴ Similarly, the University of Texas (Houston, TX) reported a 17% operative mortality for aortic dissection with organ or limb ischemia.³⁵ At the Massachusetts General Hospital (Boston, MA), the mortality in patients with acute complicated Type III aortic dissection improved from 37% from 1965–1986 to 18% from 1990–1999.³² This reduction was attributed to earlier diagnosis, aggressive medical care, and adherence to complication-specific-therapy. Moreover, while open surgery addresses the life-threatening event, 25–50% of patients still have persistent flow in the false lumen.³⁶ With residual dissected aorta, patients remain at risk for late aneurysmal degeneration, requiring serial imaging surveillance, close blood pressure monitoring, and medical therapy.³⁶ Clearly, open surgery has significant risks and carries a high morbidity.

Endovascular management of acute Type III aortic dissection

Endovascular repair is rapidly becoming a preferred approach by many centers for treatment of distal dissections. The anatomic goal with endovascular repair is to cover the entry tear preventing blood from entering the proximal dissection resulting in false lumen thrombosis and aortic remodeling (Figure 3). In the setting of malperfusion (complicated dissection), reestablishing flow in the true lumen allows blood flow to enter the visceral vessels and often treats the malperfusion. There is further evidence that this approach may reduce the potential for early and late complications of aortic dissections.³⁷ The long term aim of endovascular repair of dissections is to prevent: (I) early rupture of the dissection, (II) progression of the dissection, and (III) late aneurysmal formation. The precise timing of endovascular therapy remains unclear and often is dependent upon the patient's premorbid condition and dissection anatomy.

Two recent large meta-analyses support endovascular therapy in the management of acute Type III aortic dissection. In a report by Parker and Golledge, 942 patients from 29 studies had an in-hospital mortality of 9% with reintervention rate of 10.4%.³⁸ Emergency surgical conversion was rare (0.6%), and major complications were uncommon, although periprocedural stroke occurred in 3.1% of patients.³⁸ Retrograde Type I aortic dissection developed in 2% of patients, while 1.9% developed paraplegia.³⁸ Survival was an impressive 88% at mean follow-up of 20 months. Endovascular re-intervention was performed in 7.6% of patients, while surgical re-intervention was necessary in 2.8% of patients.³⁸ The authors

suggested that endovascular treatment for acute Type III aortic dissection as favorable given their initial outcomes.³⁸

A second meta-analysis by Xiong et al. was just published in 2009, studied 1304 patients (n=39 studies) from China who underwent endovascular repair of acute complicated Type III aortic dissections. A technical success rate of $\geq 99\%$, with 30-day mortality of 2.6% was reported. ³⁹ At late follow-up, false lumen thrombosis occurred in 92.9% of patients, and surgical conversion was required in 0.8% of cases, with endovascular re-interventions performed in 1.6% of patients.³⁹ In this report, retrograde extension into the ascending aorta (0.4%) and neurologic complications (0.6%) were both rare.³⁹

The significance of these two studies is that they were performed in two different patient populations (Western centers versus Chinese centers), had excellent technical success, low complication rates, and high success rates of false lumen thrombosis. Collectively, these findings support the use of endovascular management in acute Type III aortic dissection.

Adjunct procedures

Typically, dynamic obstruction responds to aortic endografting while static obstruction may require additional branch artery stent grafting or aortic fenestration.¹¹ Thus, additional endovascular procedures may be required in patients with malperfusion on presentation. Patients with a dominant left vertebral artery or patent left internal mammary artery bypass should undergo carotid to subclavian bypass, and carotid-carotid bypass if necessary, if coverage of the origin of the left subclavian or left common carotid arteries is necessary.^{16, 40} Additionally, carotid to subclavian bypass should be considered when extensive coverage of the descending thoracic aorta is required in patients with concomitant abdominal aortic aneurysm due to the loss of the intercostal artery supply to the spinal cord. This results in preservation of the vertebral artery and the thyrocervical trunk collaterals to the anterior and posterior spinal arteries that may be critical to decreasing the risk of paraplegia.¹¹ Similarly, a planned laparotomy either immediately or 48 hours after acute complicated Type III aortic dissection stabilization, to ensure adequate visceral perfusion has been suggested. Finally, femoral to femoral cross-over bypass may be required if the dissection flap occludes flow unilaterally.

Technical Considerations

Currently, there are no devices approved by the United States (US) Food and Drug Administration (FDA) for use in aortic dissections. There are several trials scheduled to begin enrolling patients in early 2010 to evaluate the use of newer devices on the horizon in the management of aortic dissection. Today, devices approved for thoracic aneurysm repair are used off-label for aortic dissections. Wall stents are preferred since the goal is to cover the entry tear. Uncovered stents that are available at present are less ideal due to higher radial forces on the aortic wall. Aortic dissection anatomy (tortuosity and involvement of branch vessels), adequate peripheral access, and assessment for extent of coverage necessary are important for technical success. Preoperative carotid subclavian bypass should be considered in the setting of dominant left vertebral artery or patent left internal mammary artery bypass, if coverage of the left subclavian orifice is likely. In addition, extensive coverage of the thoracic aorta should warrant preoperative lumbar drain insertion. Femoral artery access is the most common approach for stent delivery. Through-and-through access via the brachial and femoral arteries is often useful as well. The intimal tear location must be clearly identified both on preoperative imaging and prior to device deployment.⁴¹ The utility and benefit of IVUS to localize the entry tear is well established. Confirming wire placement in the true lumen by IVUS is paramount, as endograft deployment in the false lumen is catastrophic. Adequate proximal and distal landing zones are required, often necessitating coverage of the left

subclavian artery. Pharmacologic hypotension is used to minimize migration during device deployment. A properly positioned stent graft occludes the proximal tear, results in decompression and thrombosis of the false lumen (typically within 3 months), and re-expands the true lumen (Figure 4). Residual malperfusion following coverage of the proximal entry tear may require further fenestration of the dissection flap and direct stenting of branch ostia.

RESULTS

Complicated acute Type III aortic dissection

Although there are no completed randomized studies in the treatment of complicated aortic dissection, a number of small series have lent to the collective experience (Table 3). Slonim et al. were one of the first groups to report endovascular treatment of ischemic complications caused by true lumen obliteration in aortic dissection, and found stent placement to be effective with a 9.1% 30-day mortality.⁴² Dake et al. and Nienaber et al. in two landmark papers presented the concept of endovascular stenting for acute Type III aortic dissection. Dake and colleagues reported the Stanford experience with their first generation homemade device deployed in 19 patients with 79% rate of false lumen thrombosis at 13 months follow-up.⁴³ Nienaber and colleagues reported superior outcomes and more rapid functional recovery in 12 consecutive endovascular patients with acute Type III aortic dissection compared to matched open surgical cases.⁴⁴ These two early series marked the shift in endovascular treatment paradigms of acute Type III aortic dissections.

In 2001, an Austrian group was one of the first to report successful false lumen obliteration in four patients with complicated Type III aortic dissection with a *commercially available device*.⁴⁵ In a subsequent study, a group from Tulane (New Orleans, LA) reported false lumen thrombosis in nine out of ten patients with Type III aortic dissections; however, 30% of these patients still developed late abdominal aortic aneurysm.⁴⁶ In 2005, Dialeto et al. compared 28 complicated acute Type III aortic dissection patients to a similarly matched uncomplicated medically managed cohort.⁴⁷ The stented group had a 10.7% in-hospital mortality, compared to none in-house deaths in the medical group ($p=0.24$).⁴⁷ However, mid-term mortality was equivalent: 10.7% in the endovascular group versus 14.3% in the medically managed group ($p=0.71$).⁴⁷ Importantly, follow-up imaging demonstrated complete thrombosis of the false lumen in 75% of cases in the endovascular group compared to only 10.7% in the medical group ($p=0.0001$).⁴⁷ These initial trials supported stent grafting in complicated acute Type III aortic dissection. Then in 2006, Resch et al. presented their results from a multi-center Swedish trial and included 115 patients who underwent stent placement for acute complicated Type III aortic dissection.⁴⁸ Thrombosis of the false lumen at the level of the stent graft occurred in 80% of patients while 50% of patients still had persistent filling of the false lumen in the abdominal aorta.⁴⁸

Analysis in many of these studies is limited due to the inclusion of both acute and chronic aortic dissection patients. In one of the largest series in the US, Szeto et al. reported the experience from the University of Pennsylvania (Philadelphia, PA). In this series of 35 patients all of whom underwent thoracic stenting for acute complicated Type III aortic dissection, a 97.1% technical success rate and 93.4% one-year survival rate was achieved.⁴⁹ Similarly, the group from Stanford (Stanford, CA) reported their experience in 16 patients with acute complicated dissection who underwent endovascular treatment with custom made grafts within 48 hours of presentation.⁵⁰ The study reported an actuarial survival of 73% and 67% at 1 and 5 years, respectively.⁵⁰ A recent study from Malmö University Hospital, Sweden reported perioperative morbidity and mortality of 18% and 11%, and a re-intervention rate of 22% in 52 patients who underwent stent repair for complicated acute Type III aortic dissection.⁵¹

Endovascular stent graft repair appears to be a feasible approach in patients with acute complicated aortic dissection. In the setting of malperfusion, some centers advocate percutaneous fenestration of the dissection flap by a number of approaches including the *scissor technique*.^{42, 52} With this approach, stiff guide wires are placed in each lumen from a single femoral access, then by advancing a single long sheath over the two wires, the dissection flap is fenestrated.^{53–55} In 2009, Patel and Williams reported their experience from the University of Michigan (Ann Arbor, MI) in 69 patients with acute malperfusion.⁵⁶ Fenestration of the dissection flap followed by expectant management resulted in freedom from aortic rupture or open repair at 1, 5, and 8 years of 80.2%, 67.7%, and 54.2%, respectively.⁵⁶ It should be noted that these outstanding results reported in this series are center-specific, attributable to their endovascular/interventional radiology technical expertise in aortic fenestration, and have not been replicated by other centers.

Cumulatively, these studies support the concept of aggressive endovascular management of acute complicated Type III aortic dissection to address the acute ischemia and/or rupture and minimize the risk of long term complications. Currently trials are underway to evaluate this therapy prospectively. For example, the Zenith® Dissection Endovascular System (William Cook Inc., Europe) trial is enrolling patients who have “complicated” dissections with branch vessel compromise, peri-aortic effusion, transaortic growth ≥ 5 mm within 3 months, or transaortic diameter ≥ 40 mm and is expected to be completed by 2013. The outcomes of this trial will include mortality and complications at 30 days and 1 year. Similarly, the VIRTUE trial and registry recently completed enrollment to evaluate the Valiant™ Endoluminal Stent-Graft and Xcelerator® Delivery System (Medtronic Inc., Santa Rosa, CA) in complicated Type III aortic dissection patients. One hundred patients at 14 European centers will participate in this registry to collect health economic, and clinical performance data on Valiant™ for the treatment of acute dissections, complicated or expanding sub-acute dissections, and expanding chronic dissections. Other clinical trials to evaluate the role of endovascular therapy in complicated acute Type III aortic dissection are awaiting FDA approval.

Uncomplicated acute Type III aortic dissection

While the majority of data investigating the efficacy of stent grafts for uncomplicated aortic dissections is retrospective, one prospective randomized trial has been completed. The INvestigation of STent Grafts in Patients With Type B Aortic Dissection (INSTEAD) is a well known European trial comparing endograft and adjunctive medical therapy, versus optimal medical therapy alone in patients with uncomplicated Type III aortic dissections. A total of 136 patients with uncomplicated Type III aortic dissections and a patent false lumen between 2 and 52 weeks after diagnosis were enrolled, and followed at 3, 12, and 24 month intervals. This trial exclusively used the Talent™ graft (Medtronic Inc., Santa Rosa, CA). Though final results from the INSTEAD trial have not been published, preliminary results demonstrated 7% one-year mortality in the stent arm, and 3% one-year mortality in the medical arm ($p=NS$). Importantly, medically treated patients had a 10% cross-over to the surgical arm in this intent-to-treat analysis.⁵⁷ The INSTEAD trial has been criticized due to: (I) the inclusion of all patients with Type III aortic dissection rather than a subgroup of “high risk” patients, (II) the inclusion of patients who received stent graft therapy from 2 weeks to 1 year following the acute presentation, potentially resulting in a selection bias of survivors of acute events in the medical arm, and (III) a high mortality in the stent graft arm compared to other reports.

An additional trial entitled the Acute Dissection Stent-grafting Or Best Medical Treatment (ADSORB) trial is currently enrolling patients in Europe. This trial will randomize 270 patients with acute (<2 weeks) uncomplicated Type III aortic dissection to stent graft (Gore TAG®, WL Gore, Flagstaff, AZ) versus best medical therapy (strict blood pressure goal <125/80mmHg). No cross-over is planned in this multicenter prospective trial. This crucial

study is expected to be completed in late 2012 and will evaluate false lumen thrombosis, aortic rupture, and aortic dilatation as the primary end points.

Additional studies evaluating acute Type III aortic dissection

As the collective experience of endovascular repair of aortic dissections grows, there are many reports that include patients with both uncomplicated and complicated presentations. A review of the studies reporting their experience in both complicated and uncomplicated patients is presented in Table 4 with notable studies discussed below.

Between 1996 and 2001, Palma et al. found that 58 of 70 patients treated for an aortic dissection underwent elective stent graft placement for Type III dissection.⁵⁸ Custom stents were used and no paraplegia occurred despite the use of multiple stents in many patients.⁵⁸ Subsequently, in 2002 Kato et al. reported outcomes from 38 patients stented for uncomplicated and complicated Type III aortic dissection.⁵⁹ Compared to the uncomplicated group, patients with complicated dissection had higher 30-day mortality and postoperative complication rates ($p < 0.05$).

In 2002, Bortone et al. described outcomes of acute and chronic Type III (electively treated) aortic dissections, laying the foundation for the aforementioned INSTEAD trial.⁶⁰ The group reported that immediate endovascular management avoided the potential of high-risk surgical procedures, had lower postoperative complications with a shorter hospitalization, when performed within 2 weeks of the initial diagnosis of aortic dissection. These findings contrast the 2005 study from the IRAD database by Tsai et al., suggesting patients with uncomplicated Type III aortic dissections are best treated by medical therapy given the 10% 30-day mortality.⁶¹

Leurs et al. in 2004 presented the experience with endovascular treatment of thoracic aortic aneurysms ($n=249$) and dissections ($n=131$) collated by the European Collaborators on Stent Graft Techniques for Thoracic Aortic Aneurysm and Dissection Repair (EUROSTAR) and the United Kingdom Thoracic Endograft registries.⁶² They reported 89% technical success in dissection patients, with 6.5% mortality compared to 9.3% all-cause mortality for the study.⁶² Studies that have compared endovascular treatment of acute versus chronic aortic dissections indicate more risk in the acute setting. Eggebrecht et al. performed a meta-analysis compiled from 609 patients, and reported that overall complications were significantly higher in patients undergoing stent-graft placement for acute aortic dissection than in patients with chronic aortic dissection ($p=0.005$).⁶³ The overall 30-day mortality was three-fold higher in patients with acute aortic dissection when compared with the chronic group ($p=0.015$).⁶³ These reports have yielded caution in early manipulation and stenting of dissected aortas with the present devices.

SUMMARY

Acute Type III aortic dissection is associated with high morbidity and mortality. Treatment options include medical management, open surgery and endovascular techniques. There is an accumulating body of evidence suggesting poor long term outcomes with medical therapy for uncomplicated dissection. The growing experience with endovascular management of both complicated and uncomplicated Type III aortic dissection suggests this approach has promising results. As such, there is a shifting paradigm in the management and treatment of acute Type III aortic dissection at many centers towards an endovascular approach. This review provides the cardiovascular surgeon an updated perspective on the management of acute complicated and uncomplicated Type III aortic dissection. While currently there are no FDA approved devices for aortic dissection, newer devices designed to treat this disease are on the horizon. Medical therapy in uncomplicated patients has unacceptable long term mortality and late

complications. Randomized multicenter trials comparing endovascular therapy to medical management and open surgery for the treatment of acute complicated and uncomplicated Type III aortic dissection are necessary.

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Figure 1. Aortic dissection classification systems – DeBakey and Stanford classifications (adopted from Tran et al., 2009).⁴⁰

Figure 2.
Treatment algorithm for acute and chronic Type III aortic dissections.

Figure 3. Endovascular technique for Acute Type III Aortic Dissection (adopted from Atkins et al., 2006). The goal with this technique is coverage of the primary entry tear resulting in false lumen thrombosis.³²

Figure 4. Axial tomography at presentation and 6 months after early stent graft insertion in a patient with acute uncomplicated Type III aortic dissection. The false lumen at presentation is completely healed with evidence of aortic remodeling at 6 month follow-up.

Table 1**Risk Factors for Aortic Dissection**

Anatomic	Vascular Disease
Annulo aortic ectasia	Uncontrolled hypertension
Aortic Wall Abnormalities	Inflammation
Bicuspid Aortic Valve	Aortic Aneurysm
Pheochromocytoma	Aortic arteritis
Chromosomal/Genetic	Artherosclerosis
Marfan Syndrome	Behçet's disease
Ehlers Danlos Syndrome (<i>Type IV</i>)	Giant cell arteritis
Noonan Syndrome	Ormond's disease
Polycystic Kidney Disease	Syphilis
Turner Syndrome	Takayasu arteritis
Congenital	Iatrogenic/Idiopathic
Aortic arch hypoplasia	Aortic cannulation/Cross-clamping
Aortic Coarctation	Endoluminal transcatheter therapy
Congenital Aortic Stenosis	Graft anastomosis
Demographic	Instrumentation
Age (> 60 years)	Sheehan Syndrome
Lifestyle	Valvular surgery
Cocaine	
Dyslipidemia	
Pregnancy (<i>pre-eclampsia</i>)	
Smoking	

Table 2

The evolution of medical management for acute uncomplicated Type III aortic dissection

Author	Publication Year	Sample Size	Study Length	Follow up	All cause mortality	Findings / Conclusions
Fradet ⁶⁴ (meta-analysis)	1990	27	1966–1986	N/A	30% acute (30D)	26 M/M patients required procedure with 65% survival
Glower ²⁹	1990	124	1975–1988	Mean: 13 years (85%)	24% (30D), 13% (5 year)	3 M/M patients required procedure, 2 M/M patients died from rupture
Masuda ⁶⁵	1991	134	1979–1989	Mean: 1.9 years (0–10.5 years)	8% (30D), 56% (10 year)	Risk factors of poor prognosis (acute): rupture, shock, cerebral accident, myocardial infarction, severe aortic regurgitation, renal failure, mesenteric infarction, extremity arterial occlusion; Surgery for aorta >50mm
Ney ⁶⁶	1992	45	1979–1991	Mean: 33.9 months (1–143 months)	9% (30D), 36% (5 year)	Paraplegia 16%; Rupture 16%; 5 year event-free 64±10%; 12% late rupture
Schor ⁶⁷	1996	48	1985–1995	Mean: 31 months (0–112 months)	10% (1 year), 13% (5 year)	Event free 64% (5 year), 15 M/M patients required procedure
Gysi ⁶⁸	1997	187	1980–1995	N/A	8% (30D), 24% (5 year)	Rupture 14; Malperfusion 13; 9 M/M required procedure
Iguchi ⁶⁹	1998	43	1987–1996	Mean: 4.3 years (0.4–8 years)	9.3% ("late")	3 M/M patients required procedure
Elefteriades ⁷⁰	1999	60	1988–1998	N/A	N/A	Complication specific; anti-impulse therapy
Marui ⁷¹	1999	101	1988–1998	Mean: 59 months (2–125 months)	N/A	41 M/M patients required procedure; 3 M/M patients had rupture in follow-up
Genoni ⁷²	2002	78	1988–1997	Mean: 4.2±2.2 years	9% (30D)	Negative effect on survival malperfusion ($p=0.008$), rupture ($p<0.0001$)
Umana ⁷³	2002	122	1963–1999	Mean: 4.5±4.7 years	15% (1 year)	Free from surgery (10 years) 62%
Hata ⁷⁴	2003	62	1994–2002	Mean: 41.2 months (1–90 months)	1.6% (30D)	13 M/M patients had elective procedure;
Suzuki ⁷⁵	2003	282	1996–2000	N/A	9.6% (30D)	Deadly triad: hypotension/shock ($p<0.0001$), chest/back pain ($p=0.01$), malperfusion ($p=0.02$)
Mehra ⁷⁶	2004	279	1996–2001	N/A	9.9% (30D, <70years) 16.3% (30D, ≥70 years)	Patent false lumen 49.5% (total), Aorta ≥ 6cm 16.1% (total)
Onitsuka ²²	2004	76	1990–2000	Mean: 52.4 months (3–121 months)	17% (30D)	6 M/M patients required procedure; 33% experienced "events"
Estrera ³⁵	2007	159	2001–2006	Median: 20 months (0–67)	7.4% (30D)	23 M/M patients required procedure;

<i>Author</i>	<i>Publication Year</i>	<i>Sample Size</i>	<i>Study Length</i>	<i>Follow up</i>	<i>All cause mortality</i>	<i>Findings / Conclusions</i>
				months)		Paraplegia 8%

M/M = Medical management N/A = Not available

Table 3
The evolution of endovascular management for acute complicated Type III aortic dissection

Author	Publication Year	Sample Size	Study Length	Follow up	All cause mortality	Findings / Conclusions	Stent Type
Slonim ⁴²	1996	11	1992–1995	Mean: 10.1 months (2 weeks–39 months)	9.1%	Technique included fenestration and stent placement, 2 of 5 fenestration patients required re-intervention	Palmaz® ¹
Dake ⁴³	1999	22	1996–1998	Mean: 13 months	16% (30D)	False lumen thrombosis 79%; Re-vascularization in 76%	Custom
Nienaber ⁴⁴	1999	12	1997–1998	12 months	0.0%	Technical success in 100%; no endoleak; no morbidity	Talent™ ²
Tiesenhausen ⁴⁵	2001	4	1998	Mean: 11.5 months (2–34 months)	0.0%	False lumen thrombosis 100%; Distal aneurysm in 1 patient	Talent™ ²
Lopera ⁴⁶	2003	10	1999–2001	Mean: 20 months (7–30 months)	10.0%	Technical failure, stroke, deep vein thrombosis in 1 patient each; False lumen thrombosis 60%; Distal aneurysm in 20%	Custom
Vedantham ⁷⁷	2003	11	1998–2002	Mean: 16 months	0.0%	Preferred to open surgery in complicated Type B aortic dissection	Wallstent® ³
Duebener ⁷⁸	2004	10	2000–2003	Mean: 25 months (1–38 months)	20% (30D)	Early mortality reduced by emergency endovascular stent grafting (No mortality in follow-up period)	Talent™ ²
Dialetto ⁴⁷	2005	28	1999–2004	Mean: 18.1 months (1–61 months)	10.7%	Endoleak in 1 patient; False lumen thrombosis 75%; Distal aneurysm 3.5%	Talent™ ² , Gore EXCLUDER® ⁴
Resch ⁴⁸	2006	115	1994–2005	Median: 14 months (1–79 months)	9.3% (30D)	False lumen thrombosis in 80%; Distal aneurysm in 16%	Talent™ ² , Gore EXCLUDER® ⁴ Gore TAG® ⁴ , Zenith® ⁵ Endofit™ ⁶
Chemelli-Steingruber ⁷⁹	2008	38	1996–2008	Mean: 33 months	23.7%	Secondary interventions in 2 patients; Conversion open in 3 patients; Endoleak in 9 patients	Talent™ ² , Gore EXCLUDER® ⁴ Gore TAG® ⁴
Pearce ⁸⁰	2008	15	2005–2007	Mean: 5.8 months (0–12 months)	13.3% (30D), 20%	TEVAR has high technical success, excellent results at resolving malperfusion, acceptably low complications	Gore TAG® ⁴
Szeto ⁴⁹	2008	35	2004–2007	Mean: 13 months (3–47 months)	2.8%	Technical success 97.1%; 93.4% (1-year survival)	Talent™ ² , Gore TAG® ⁴ Zenith ELSE® ⁵
Verhoye ⁵⁰	2008	16	1996–2004	12 months	25% (30D)	Distal aorta dilation 1 patient; Freedom from treatment failure at 5 years 67%	Custom, Gore EXCLUDER® ⁴ Gore TAG® ⁴
Feezor ⁸¹	2009	33	2005–2007	Mean: 5.5 months	21% (30D)	1 major complication in 76%	Gore TAG® ⁴

Author	Publication Year	Sample Size	Study Length	Follow up	All cause mortality	Findings / Conclusions	Stent Type
Albors ⁸²	2009	25	2003–2008	Mean: 30 months (7–53 months)	8% (30D)	patients, mean follow-up 61% alive and 45% living at home Type I Endoleak in 16%; paraparesis in 4%	Talent TM 2, Gore TAG [®] 4 Zenith TX1 [®] 5, Zenith TX2 [®] 5
Khoynezhad ²⁸	2009	28	1999–2007	Mean: 36 months (1–88 months)	10.7%	82% (1-year survival); 78% (5 year survival); complete thrombosis 85%; treatment failure 18%	Talent TM 2, Gore TAG [®] 4
Manning ⁵¹	2009	52	2001–2008	Mean: 31 months	11.0%	1 patient with persistent false lumen perfusion, only Type IIIb needed re-intervention	Talent TM 2, Gore TAG [®] 4 Zenith [®] 5, Endofit TM 6, Relay TM 7
Patel ⁵⁶	2009	69	1997–2008	Mean: 41.9 months	17.4% (30D), 30%	Freedom from aortic rupture or open repair at 1, 5, and 8 years was 80.2%, 67.7%, and 54.2%	Wallstent [®] 3
Park ⁸³	2009	17	1996–2007	Mean: 731 days (3–2719 days)	8.3% (30D)	Malperfusion improved in 90%; Annual growth rate 3.64mm/year thoracic aorta, 2.64mm/year celiac axis, 1.42mm/year infrarenal aorta	Niti-S TM 8, Wallstent [®] 3 Smart [®] Stent ⁹

¹ Cordis Corporation, Johnson & Johnson Inc., Warren, NJ

² World Medical, Medtronic Vascular Inc., Sunrise, FL

³ Boston Scientific Corp., Natick, MA

⁴ Thoracic Endoprosthesis, W.L. Gore & Associates, Sunnyvale, CA

⁵ Cook Inc., Bloomington, IN

⁶ Endomed Inc., London, United Kingdom

⁷ Bolton Medical Inc., Sunrise, FL

⁸ Taewoong Medical Co. Ltd., Seoul, Korea

⁹ Cordis Corp., Miami Lakes, FL

Table 4
The evolution of endovascular management for acute Type III aortic dissection (complicated and uncomplicated)

Author	Publication Year	Sample Size	Study Length	Follow up	All cause mortality	Findings / Conclusions	Stent Type
Czerniak ⁸⁴	2000	7	1996–1999	Mean: 14 months (1–25 months)	14.3%	Technical success in 86%; 1 patient dissection into ascending aorta; False lumen thrombosis in 86%	Talent TM ¹ , Vanguard TM ²
Sailer ⁸⁵	2001	11	1997–2000	Median: 254 days (2–360 days)		Endoleak in 1 patient; Progressive dissection in 1 patient	Talent TM ¹ Vanguard TM ² Gore EXCLUDER [®] ³
Palmer ⁸⁶	2002	14	1999–2001	Mean: 14 months (1–23 months)	7.1%	Endoleak in 4 patients	Talent TM ¹ , Gore EXCLUDER [®] ³
Palma ⁵⁸	2002	70	1996–2001	Mean: 29 months (1–55 months)	8.6%	Success in 93%; 18.9% persistent false lumen; 7.1% conversion surgery	Custom
MacKenzie ⁸⁷	2004	53	1992–2002		0.0%	Mortality (30D) surgery 58.3%, first half vs. 7.1%, second half ($p = 0.019$)	Talent TM ¹
Leurs ⁶²	2004	131	1997–2003	12 months	6.5% (acute dissection)	30D mortality: 12% (Emergency), 6.5% (Elective) with 90% (cumulative 1-year survival) and 1.5% (late death)	Talent TM ¹ , Gore EXCLUDER [®] ³ Zenith [®] ⁴ , Endofit TM ⁵
Esgebrecht ⁶³ (meta-analysis)	2006	248	1999–2004	Mean: 19.5 months	9.8 % (30 D), 12.6% (1-year)	Success >95%, major complications 14–18%; re-intervention rate 11.9%; rupture during follow up 2.3%	Custom
Won ⁸⁸	2006	51	1994–2003	Mean: 33 months (12–107 months)	No procedure related mortality	Type 1 Endoleak 10%; Type 2 Endoleak 3%; 10% with saccular aneurysm; 7% with progressive dilation	Custom
Xu ⁸⁹	2006	63	2001–2005	Mean: 11.7 months (1–47 months)	3.2% (30D)	Incomplete seal in 4.8%; 4 patients re-intervention; 98.4% false lumen thrombosis; 89.4% (4 year survival)	Talent TM ¹ , Vasoflow [®] ⁶ Agis [®] ⁷ , Griking [®] ⁸ Ankura [®] ⁹
Schoder ⁹⁰	2007	28	2000–2005	Mean: 2 years	10.7% (30D)	90% treated successfully; 89.3% (3 year survival); 86% false lumen thrombosis	Talent TM ¹ , Gore EXCLUDER [®] ³
Jing ⁹¹	2008	42	2002–2007	Mean: 18.7 months	38.1%	Complications (higher in "earlier term") 21.4%; 90% (5 year survival)	
Chang ⁹²	2008	47	2001–2006	Median: 28.2 months (6–75 months)	6.1% (30D), 12.8% (acute group)	9 patients lost to follow-up (death); 30D Endoleak 7.3%	
Parker and Grolledge ³⁸ (meta-analysis)	2008	942	1997–2007	Mean: 20 months	9% (in-hospital)	Re-intervention rate 10.4%; Major complications in 8.1%; Rupture 0.8%	
Guangqi ⁹³	2009	72	2001–2006	Mean: 14.4 months	1.4% (30D)	Successful in 88.9%; Endoleak in 22.4%; 75% (3 year survival); 5 patients lost to follow-up	Talent TM ¹ , Zenith [®] ⁴ Agis [®] ⁷ , Ankura [®] ⁹

Author	Publication Year	Sample Size	Study Length	Follow up	All cause mortality	Findings / Conclusions	Stent Type
Kaya ⁹⁴	2009	37	1996–2004	Mean: 15 months (1–69 months)	8.0%	Conversion in 2 patients; Intraoperative mortality 1.8%	Talent TM ¹
Xenos ⁹⁵ (meta-analysis)	2009	13	2003–2007	Mean: 3.4 months (1–10 months)	7.5% (30D)	Stroke in 15%; stent graft failure in 2 patients; 1 patient died due to endovascular grafting	Gore TAG [®] ³ , Gore EXCLUDER [®] ³
Xiong ³⁹ (meta-analysis)	2009	1304	2001–2007	Mean: 27.1 months	3.1% (30D)	Success rate ≥99% in select cohort; 95.2% (5 year survival); reintervention rate 2.4%	Custom, Talent TM ¹ , Zenith [®] ⁴ Endofit TM ⁵ , VasoFlow [®] ⁶ , Agis [®] ⁷ Griking [®] ⁸ , Ankura [®] ⁹ , Powerlink [®] ¹⁰ , Aortech [®] ¹¹
Chemelli-Steingrub ⁹⁶	2009	76	1996–2008	Mean: 41 months (0–97 months)	23.4%	Freedom from late dissection 83%; rupture free survival 93%	

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² Boston Scientific Corp., Natick, MA

³ Thoracic Endoprosthesis, W.L. Gore & Associates, Flagstaff, AZ

⁴ Cook Inc., Bloomington, IN

⁵ Endomed Inc., London, United Kingdom

⁶ Vascor Medical Co. Ltd., Suzhou, China

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