Effectiveness of intensive medical therapy in type B aortic dissection: A single-center experience

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Objective: **Although the mainstay of managing acute descending thoracic aortic dissection (ADTAD) remains medical, certain patients will require emergency surgery for complications of rupture or ischemia. This study evaluates factors that affect outcome and determines which patients previously treated surgically would have been eligible for endovascular repair.**

Methods: **A single-institution retrospective study was conducted of patients who presented with clinical signs of ADTAD that was confirmed by magnetic resonance angiography (MRA) or computed tomography (CT). All patients were admitted to the intensive care unit (ICU) and medically managed to maintain systolic blood pressure <120 mm Hg and heart rate <70 beats/min. Two treatment groups were identified: group 1 received medical treatment only; group 2 received medical treatment plus emergency surgery. Patient demographic and clinical data were correlated with 30-day group mortality and morbidity and need for emergency surgery. The MRA and CT scan images of group 2 were retrospectively reviewed to determine if currently available endovascular treatment could have been done. The Fisher exact test was used to compare between the groups, and $P < .05$ was considered significant.**

Results: **Between 1991 and 2005, 83 patients (55 men) were treated for ADTAD. The mean age was 67 years (range, 38 to 85). Sixty-eight patients (82%) had hypertension, three (3.6%) had Marfan syndrome, and 51 (62%) were smokers. Twenty-five (32%) of the patients were receiving β-blocker therapy before the onset of their symptoms. Back pain was the most common initial symptom (72.2%). Emergency surgery was required in 19 patients (23%): 12 for rupture or impending rupture, four for mesenteric ischemia, and three for lower extremity ischemia. The need for emergency surgery was significantly higher in smokers ($P = .03$), in patients >70 years old ($P = .035$), and in patients who were not receiving β-blocker therapy before the onset of symptoms ($P = .023$). The combined overall morbidity rate was 33%, and the mortality rate was 9.6%. Morbidity in group 2 was 64% and significantly higher than the 23% in group 1 ($P = .00227$). The mortality rate was also higher in group 2 at 31.5% compared with group 1 at 1.6% ($P = .0004$). Factors affecting the overall mortality included age >70 years ($P = .057$), previous abdominal aortic aneurysm repair ($P = .018$), tobacco use ($P = .039$), and the presence of leg pain at initial presentation ($P = .013$). As determined from the review of radiologic data, 11 of 13 patients with scans available for review in group 2 could have been treated with currently available endovascular grafts.**

Conclusions: **Intensive medical therapies are effective in preventing early mortality associated with ADTAD. Predictably, the need for emergency surgery carries a high morbidity and mortality rate. Most patients in this series requiring emergency surgery could have been candidates for endovascular therapy had it been available. (J Vasc Surg 2007;45: 1114-9.)**

Aortic dissection is one of the major vascular catastrophes. Since the first description in 1796 by King George II's personal physician,[1] aortic dissection has been well studied. Early diagnosis remains a challenge, however, with a delay in diagnosis occurring in up to 30% to 40% of cases.[2] In the United States, the incidence of aortic dissection is about 9000 new cases per year.[3] Up to one third of these cases involve the descending thoracic aorta.[3,4]

The current standard therapy for descending thoracic aortic dissection (ADTAD) consists of intensive medical management with the use of β-blockade, followed by vasodilators such as nitroglycerin or nitroprusside to reduce heart rate and the systematic blood pressure and analgesics to control pain. In current clinical practice, surgery is reserved for patients who fail aggressive medical therapy or present with complications of ADTAD such as organ or limb ischemia, rupture, impending rupture, or progression to aneurysmal dilatation of the aorta.

A review of the literature on early mortality in uncomplicated acute aortic dissection clearly favors medical management, whereas historically, emergency surgical management of ADTAD carries a very high morbidity and mortality rate.[5] In recent years, alternative less invasive endovascular therapy has been introduced with caution and has been associated with encouraging results.[6-9] The objectives of this study are:

1. to evaluate factors that affect clinical outcomes as they pertain to morbidity and mortality in the first 30 days after admission for acute symptoms, including the need for emergency surgery in the first 2 weeks from the onset of symptoms, and

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2. to determine which patients previously treated with emergency surgery for complications or failure of medical therapy could have been eligible for endovascular repair with the currently available endovascular therapy.

MATERIALS AND METHODS

This was a single-center retrospective study of patients treated for ADTAD at the University of Wisconsin Hospital and Clinics. The study was reviewed and approved by the Institutional Review Board. All patients presenting to the emergency department with ADTAD were admitted to the Vascular Surgery Service. ADTAD was defined as a dissection starting distal to the origin of the left subclavian artery without involvement of the aortic arch or its branches. Patients were identified from medical records using diagnosis codes including 441.00, 401.01, 441.02, 441.03, and 441.04. The diagnosis of ADTAD was confirmed from magnetic resonance angiography (MRA) or computed tomographic (CT) imaging studies. The acuity of the diagnosis was based on onset of symptoms <2 weeks before admission. Patients with radiographic evidence of ascending aortic dissection were excluded from the study.

All patients were initially admitted to the intensive care unit (ICU) for standardized medical management with β-blocker and vasodilator medications unless there was clinical suspicion of rupture or ischemic complications that required immediate surgical intervention. The standard medical management targets were to keep systolic blood pressure <120 mm Hg and heart rate <70 beats/min and to control pain. Patients were treated in the ICU with intravenous administration of medications, followed by gradual transition to oral medications.

Follow-up was scheduled at 1, 3, and 6 months, 1 year, and every 2 years thereafter if no aneurysmal growth was seen. The survival data were supplemented by searching the Social Security Death Index.

Two treatment groups were identified: patients who were successfully managed with intensive medical therapy (group 1) and patients who required emergency surgery for ischemic complications, rupture, or impending rupture of the dissected aorta (group 2). Our thoracoabdominal aortic aneurysm repair protocol has been reported previously.[10]

Patient demographics, clinical presentation, comorbid conditions, use of β-blockers before the onset of symptoms, and history of smoking were recorded. Renal dysfunction was defined as serum creatinine level of ≥1.5 mg/dL. These data were correlated with the 30-day morbidity and mortality in each group, and factors that contributed to the need for emergency surgery were determined. The mean length of stay in the ICU and hospital were also compared between treatment groups.

A retrospective review of CT or MRA images in the group 2 patients whose images were still available was also undertaken to determine if currently available endovascular grafts could have been used to treat these patients. This was based on the determination of an adequate proximal aortic neck length of ’1.5 cm and an aortic neck diameter of ≤37 mm proximal to the primary dissection point, the goal for endovascular therapy being to seal the proximal dissection entry and preserve true lumen patency.

Table I. Patient characteristics

Characteristic	*Patients (n = 83), n (%)*
Mean age, years (range)	67 (38-85)
M:F	55:28
Hypertension	68 (81.9)
Marfan syndrome	3 (3.6)
Diabetes mellitus	7 (8.4)
Renal dysfunction	4 (4.8)
CABG	7 (8.4)
CAD	21 (25.3)
CVA	8 (9.6)

CABG, coronary artery bypass graft; *CAD,* coronary artery disease; *CVA,* cerebrovascular accident.

Univariate analysis was performed to assess factors that affected the overall mortality and need for emergency surgery. The Fisher exact test was used for comparisons between the groups, and $P < .05$ was considered significant. Kaplan-Meier survival curves were plotted to compare survival between the groups.

RESULTS

The database search identified 148 patients, and 83 patients (55 men) fulfilled the criteria for this study. The mean age was 67 years (range, 38 to 85). Forty-two (50%) patients presented to the emergency department within hours of onset of symptom. The most common symptoms included back pain (72.2%), chest pain (44.5%), abdominal pain (30%), and leg pain (4.5%). Predictably 82% of the patients were known to have hypertension, and 25 (32%) were receiving β-blocker therapy as part of their antihypertensive regimen before onset of their symptoms. At the time of discharge, 25 patients (40%) in group 1 were taking four or more antihypertensive medications. Other comorbid conditions included coronary artery disease (CAD) in 21, diabetes mellitus (DM) in seven, and a history of previous stroke (CVA) in eight. Only three patients (3.6%) had a diagnosis of Marfan syndrome. Fifty-one (62%) of patients were smokers, and 20 (24%) had history of previous abdominal aortic aneurysm repair (Table I).

Intensive medical therapy alone was successfully used to treat 64 patients (77%; group 1), and 19 (23%) required emergency surgery for rupture/impending rupture (n = 12) mesenteric ischemia (n = 4) and lower extremity ischemia (n = 3). Of the 19 patients who required surgery, 14 (6 rupture, 3 symptomatic >6 cm aneurysms, 3 mesenteric ischemia, and 2 extremity ischemia) were operated the day of admission, and five patients requiring emergency surgery were initially treated with intensive medical therapy. Three of these five patients had continued chest pain and enlargement of the aorta, one developed leg ischemia, and one patient developed mesenteric ischemia. No intervention was performed solely for renal dysfunction.

Patients aged >70 years ($P = .035$) and smokers ($P = .03$) were more likely to require emergency surgery. In

Table II. Factors affecting mortality and the need for emergency surgery

	All patients (n = 83)		*Group 2 (n = 19)*	
	Factors affecting mortality		*Factors affecting emergency surgery*	
Factors	*Mortality rate (%)*	*P*	*Mortality rate (%)*	*P*
Age				
<70	2.4	.05	12	.035
>70	16.6		34	
Gender				
M	9	1	22	.7
F	10		25	
Leg pain				
Yes	33	.013	45	.1
No	5.6		19.7	
Previous AAA repair				
Yes	25	.018	26	.7
No	4.7		22	
Tobacco use				
Yes	14	.039	30.6	.03
No	0		9.6	
β-blocker				
Yes	8	1	8.3	.025
No	6		32.6	

AAA, Abdominal aortic aneurysm.

contrast, patients who were taking a β-blocker as part of their antihypertensive regimen before onset of symptoms seemed to be protected from emergency surgery (P = .023).

The overall 30-day mortality was 9.6% (8 deaths). Age >70 years (P = .057), clinical presentation with leg pain (P = .013), previous abdominal aortic aneurysm repair (P = .018), and a history of tobacco use (P = .039) increased significantly the possibility of death. Gender, presentation with chest or abdominal pain, history of hypertension, coronary artery disease, and history of diabetes mellitus were not associated with an increase in the over all mortality (Table II).

The mortality rate was significantly higher in group 2 (P = .00046) than in group 1. Two patients (1.6%) in group 1 died from sudden cardiopulmonary arrest (no autopsy was performed), and six patients died in group 2. Four of these deaths occurred in patients who required emergency surgery for rupture or impending rupture. Their deaths were due to multisystem organ failure (MSOF). One of the remaining two patients who died presented with limb ischemia and died of MSOF, and another who presented with mesenteric ischemia died from hemorrhage (Table III).

The overall morbidity rate was 33% (n = 27), consisting of 15 in group 1 and 12 in group 2. Morbidity in group 2 was significantly higher compared with group 1 (P = .002). Two patients in group 1 presented with paraplegia as part of their initial symptoms. Two patients in group 2 developed paraplegia after surgery. Other morbidities included renal failure, bacteremia, mental status changes, renal dysfunction, myocardial infarction, and arrhythmias.

Table III. Complications

Type of complication	*Group 1 (n = 15)*	*Group 2 (n = 12)*
Paraplegia	2	2
Mental status changes	7	3*
Respiratory failure	1	2
Myocardial Infarction	4	2
Arrhythmia	2	
Sepsis	1	2*
Phrenic nerve injury		1
Bleeding		2†
Cerebrovascular accident		1†
Lower extremity ischemia		1

*Each star represents death. Of the 3 patients with mental status changes, 2 died, both patients with sepsis died as well, and 1 patient with bleeding and 1 with cerebrovascular accident died. There were a total of six deaths.
†In-hospital mortality.

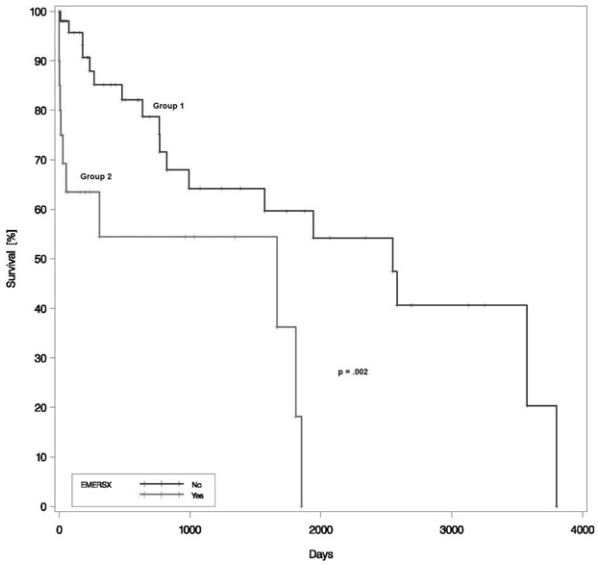

Fig. Kaplan-Meier curves for long-term survival for group 1 (blue line) and group 2 (red line). *EMERSX,* Emergency surgery.

The overall mean length of hospital stay was 14 days and length of ICU care was 8 days. There was no significant difference in the mean length of hospital stay (P = .1) and ICU stay (P = .12) between the two groups.

The mean follow-up time for the cohort of patients who survived the initial admission was 38.2 months. During follow-up, 18 patients (28%) required surgery for continued aneurysmal growth of the dissected aorta. Of these elective surgery cases, 15 were performed ≤1 year from onset of symptoms. The overall survival for the entire cohort was 75%, 60%, and 49% at 1, 3, and 5 years, respectively. For group 1 vs group 2 patients, survival was 85% vs 54% at 1 year, 64% vs 54% at 3 years, and 59% vs 18% at 5 years (P = .002; Fig).

A review of available MRA or CT scans, or both, of the 13 patients in group 2 revealed that 11 could have been

treated with the currently available Excluder thoracic endovascular graft system (W. L. Gore & Assoc, Flagstaff, Ariz). Mean aortic diameter proximal to the proximal aortic tear was 34 mm (range, 26 to 42 mm), and coverage of the left subclavian artery origin would have been required in five cases to achieve an adequate proximal aortic length for seal.

DISCUSSION

ADTAD in the United States continues to be a challenging medical condition, with no significant improvement in the treatment outcomes during the past several decades. As reported by several authors, hypertension seems to be the most common predisposing factor, whereas dissection related to collagen vascular disease is relatively rare.[3,4] Hypertension was present in 82% of our patients, and only 3.6% were known to have Marfan syndrome. The clinical presentation in this study was also similar to other reports and consisted of severe chest, back, or abdominal pain in the presence of uncontrolled hypertension.

The gold standard for treating newly diagnosed uncomplicated ADTAD remains medical therapy, whereas surgery is reserved for complicated presentations of ADTAD such as visceral or limb ischemia or symptoms of rupture or impending rupture.[3] This is because of the high morbidity and mortality rates associated with emergency surgery in patients with ADTAD.[5] Our current intensive medical therapy protocol consists of the intravenous administration of a β-blocker, followed by a vasodilator (nitroglycerine) and other additional oral or parenteral antihypertensive medications as needed.

Most recent reports indicate the overall mortality rate for ADTAD to be about 10%.[11] This compares favorably with our result, where the overall in-hospital mortality rate was 9.6%. Predictably, the mortality rate was significantly higher if patients required emergency surgery compared with those uncomplicated patients treated with medical therapy alone. Similar to other reports, age >70 also appeared to adversely affect overall survival and increased the need for emergency surgery.[12] Contrary to the data reported by Niehuber et al,[13] our study did not identify any gender-related increase in morbidity and mortality. However, other factors that did adversely affected outcomes by increasing overall mortality included previous infrarenal aortic aneurysm repair, initial clinical presentation with leg pain, and tobacco use. We have no explanation why leg pain was associated with an increased risk of mortality, although this may be related to a more extensive dissection.

In our series, the early mortality rate for patients who were successfully treated medically was remarkably low (1.6%). From these data it is difficult to justify endovascular therapy in patients who do not develop complications at their initial presentation. This statement seems to be supported by a report by Kato et al,[14] where stent grafting of acute dissection was associated with early and late complication rates of 33% and 36%. In contrast, early and late complication rates of 4% and 0% were reported for similar treatment of chronic dissections. These authors thus recommended delay in stent graft treatment of uncomplicated dissections but also suggested that endovascular therapy may decrease the significant morbidity and mortality associated with emergency surgery.[14]

Emergency surgery was required in 23% of our patients. As indicated by several authors, mortality rates after emergency surgery are very high.[3,11,12,15] The high mortality rate seen in patients requiring emergency surgery for ADTAD demonstrated the need for an alternative, safer approach to this complex problem.

In recent years, several reports have documented results of the use of various endovascular therapies as a less invasive alternative treatment for ADTAD. These include covered stent grafting, percutaneous aortic fenestration, and aortic branch vessel stenting.[6,7,16-19] The goals of endovascular therapy using stent grafts in ADTAD include coverage and stabilization of the proximal dissection entry site, thrombosis of the false lumen, preservation of the true lumen and branch artery flow, and ideally, a decrease in the diameter of the aorta.

Although there is no level I evidence on the use of endovascular therapy for ADTAD, several reports of small series are available. Technical success with this approach is up to 100% in most series, and mortality rates are 6% to 10%.[6,7,17] Xu et al[17] recently reported successful proximal dissection entry site sealing in up to 95.2% of patients and a false lumen thrombosis rate of 98.4% at the 1-year follow-up.[17] In addition, Gaxotte et al[16] as well as Xu reported that when complete thrombosis of the false lumen occurred, it resulted in a decrease in the aortic diameter, whereas partial thrombosis was associated with continued aneurysm enlargement.[16,17] Review of CT scans and MRA of 13 patents in group 2 in our study whose images were available revealed that 11 patients could have been treated with currently the available Excluder thoracic endograft system. In four patients, however, coverage of the origin of the left subclavian artery would have been needed to achieve a sufficient proximal aortic length for endograft sealing.

Patients in group 1 in our study were monitored closely, and 28% required subsequent surgical repair. Most of these procedures, 15 (83%) of 18, were performed ≤1 year from the initial presentation, suggesting the need for very close follow up in the earlier months following ADTAD. According to a report by Gallo et al,[15] most medically treated patients eventually require surgery for aneurysmal growth. On the other hand, Estrera et al[11] reports that only 4.3% of their patients required surgical intervention after discharge. Regardless, as a result of our findings we currently recommend follow-up imaging with a CT scan or MRA at discharge, 3 and 6 months, and yearly thereafter.

CONCLUSION

Most patients with ADTAD can initially be successfully managed medically, and when successful, such management is associated with a very low mortality and morbidity rates. Careful follow-up is required, however, because up to one third of these patients require elective intervention in the future. In contrast, outcomes are significantly worse if

patients with ADTAD initially require emergency surgery. From retrospective review of imaging studies of patients who required emergency surgery after ADTAD in this series, most patients could have been treated with endovascular grafts, which might have improved outcomes. Further clinical studies are needed to carefully assess the value of endovascular stent grafting in the treatment of patients requiring emergency intervention for ADTAD and the need for endovascular stent grafting in patients with uncomplicated ADTAD.

AUTHOR CONTRIBUTIONS

Conception and design: GT, CA
Analysis and interpretation: GT
Data collection: GT
Writing the article: GT, CA, JH
Critical revision of the article: GT, CA, JH, MM, WT
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Overall responsibility: GT

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DISCUSSION

Dr Walter McCarthy (Chicago, Ill). Tell us now what you have done as far as using endovascular techniques. What is your protocol at this point? You have gone back over your data. You have convinced yourself that you might be able to use it. What have you done?

Dr Girma Tefera. Thank you for the question. Since we reviewed this data we have a protocol in place to treat an endovascular patient who may present with complications requiring surgery. In fact, we recently had a case of acute type B dissection with occlusion of the aorta at the celiac artery. This patient presented with paraplegia, anuria, and abdominal pain. We were able to open the true lumen with balloon mounted palmaz stents deployed across the occluded segment. Although this procedure temporarily was successful, it reoccluded 48 hours later. The patient was then taken to surgery for aortic replacement. Intravascular ultrasound was very helpful in identifying the true lumen. If I am faced with a similar situation, I will probably try to address the proximal tear also with covered stent graft.

Dr Michael Dalsing (Indianapolis, Ind). Very nice paper, Dr Tefera. Why do you think that having a prior aortic aneurysm repair makes things worse and that you have to operate on them more quickly than if they do not?

Dr Tefera. The short answer to your question is, I do not know. It is, however, interesting that this same phenomenon was observed during thoracic endograft trials. This may have something to do with the absence of lumbar arteries in the infrarenal aorta.

Dr John Matsumura (Chicago, Ill). I don't know about why open repair might make it risky, but I have noticed in the patients who have an endovascular infrarenal repair who develop a thoracic dissection that it is a very lethal combination because it tries to stent open the true lumen, and because both legs feed off the true lumen, oftentimes the legs create a huge outflow and increase the chance for true lumen collapse. Roy, have you seen patients after infrarenal repair who have type B dissection? I have seen two cases where it is a very bad combination.

Unidentified speaker. We wrote up one case after an endograft with an aortic dissection where the fixation and the seal in the iliacs was so much stronger so that the false lumen kind of terminates in the aneurysm sac and crushed the endograft itself.

That was probably a year or two after an endo AAA repair, but I have not seen a lot of dissections in those endo repair patients, infrarenal endo repairs, or open repair patients. I mean, I have always viewed open repair as a little bit protective of dissection because it always prevents the dissection from going down to the iliacs but I do not know. Your data are very interesting.

Unidentified speaker. We have seen two patients with aortic occlusion who had infrarenal aortic aneurysms and later dissection. One was an endograft and one was a standard repair. I think it can occur with either patient. Both were successfully treated for their aortic occlusion with fenestration and revision. The endograft we had to explant which was kind of a chore in the fact of an acute dissection but it was able to be done.

Dr Jacobs–Don v Chad. I would question your conclusion. One of them was that we should not be treating _____ currently _____ with an endograft. I am not arguing that point, but I am not sure that your data would say that because we do not know what they die of. With your survival curve there looking pretty steep on these patients, I think you would have to know what those complications potentially of the dissection are down the line that you may be able to prevent. That may be the mortality that you are seeing in your survival group.

Dr Tefera. I think the point I indirectly wanted to make was when you are successful treating these patients medically the mortality rate of 1.6 is very hard to argue with because when you see stent graft placement in dissection settings, high technical success is reported but the mortality rate is not that low yet. That is why I question and basically want to bring this point up, but I think when we followed our patients, about a third them do require elective surgery for further aneurysm expansion. We did not go specifically to see of those who died during follow-up why they died, but you also saw that from the curve point of view the 1-year survival was 85% when they were successfully medically treated.

Dr Roy Greenberg (Cleveland, Ohio). I think that your results are right on in terms of the low mortality rate associated with medical therapy for uncomplicated dissections, but something that we have struggled with is how to define optimal medical therapy because it varies so much. So I was wondering if you had a system for how you looked at patients. When was medical therapy optimal and what was the drug regimen? Were they always beta-blockers? Did you combine beta-blockers with ACE inhibitors? How did you manage those patients?

Dr Tefera. At our institution all patients who come in with type B dissection come to the Vascular Surgery Service, and we basically admit all of them. Unless there is a life-threatening, limb-threatening situation that needs to go to the operating room, these patients will all go to the intensive care unit, have an arterial line placed, and we have preprinted orders for esmolol drips and nitroglycerin to be started right away. On top of that, we start of a course oral medications to supplement. We take actually several days of ICU stabilization prior to these patients being transferred to the floor. In fact, if you look at the hospital length of stay for successful medical therapy patients, it is almost the same as for those who needed surgery. We take really our time and we have liberal usage of the maximum dosage of esmolol and nitroglycerin, and occasionally nitroprusside, and we transition them as quickly as possible into an oral regimen.

Of course they all go home with a lot more oral medication than what they had before, and we communicate with the primary care physicians prior to their being discharged. We really take total control, and of course medical therapy might be a failure sometimes, but it does not really occur in terms of not being able to control the blood pressure, particularly, if you have a pseudocoarctation kind of picture is rare certainly and by having these patients on your service I think helps.