Aortic remodeling after endografting of thoracoabdominal aortic dissection

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Purpose: **This study assessed the clinical outcome, morphologic changes, and behavior of acute and chronic type B aortic dissections after endovascular repair and evaluated the extent of dissection and diameter changes in the true (TL), false (FL), and whole lumen (WL) during follow-up.**

Methods: **From May 2000 to September 2006, preprocedural and follow-up computed tomography scans were evaluated in 106 patients. Indices of the TL (TLi) and FL (FLi) were calculated at the proximal (p), middle (m), and distal (d) third of the descending thoracic aorta by dividing the TL or FL diameter by the WL. Analyses were by paired *t* test and χ^2.**

Results: **Stent grafts were used to treat 106 patients (mean age, 55 years, 70% men) with acute 59 (55.7%) and chronic 47 (44.3%) lesions. The entry site was successfully covered in 100 patients. The incidences of paraplegia and paresis were 2.8% and 1.0%. Mortality was 7.5% (8 patients), including two intraoperative deaths of contained ruptures. Seven (6.6%) early endoleaks occurred. At a mean follow-up of 15.6 months, TLi improved from 0.45 to 0.88 in the proximal third (p/3), from 0.42 to 0.81 in the middle third (m/3), and from 0.44 to 0.74 in the distal third (d/3), demonstrating expansion of the TL. Two patients had decrease in TL due to endoleak needing reintervention. The FLi decreased from 0.41 to 0.06 in p/3, from 0.44 to 0.10 in the m/3, and from 0.42 to 0.21 in the d/3, indicating FL shrinkage. Changes in the TLi and FLi were statistically significant. The decrease in the WL after repair was statistically significant in the proximal and middle aorta. Fourteen patients (13.2%) had increase in WL; seven required a second intervention. FL thrombosis occurred in 69 (65.1%). During follow-up, 36 (36.9%) patients had no retrograde flow, with complete shrinkage of the FL. The FL completely shrank in 28 patients (26.4%) despite retrograde flow. The FL increased in eight patients (7.5%); five needed reintervention. Thrombosis of FL was statistically significant with acute dissections and when dissection remained above the diaphragm (type IIIA; *P* = .001 and *P* = .0133).**

Conclusion: **Remodeling changes were seen when the entry tear was covered. The fate of the FL was determined by persistent antegrade flow and the level of the retrograde flow. Endografting for thoracic type B dissection was successful and promoted positive aortic remodeling changes. (J Vasc Surg 2008;47:1188-94.)**

The optimal treatment of type B aortic dissections is becoming controversial. Medical treatment is the first line of management, and surgical repair is reserved for those who have organ or extremity malperfusion, impending rupture, blood pressure refractory to medication, and persistent pain. Medical treatment is favored rather than open repair in noncomplicated acute dissection because of associated surgical morbidity and mortality.[1-4] The effectiveness of endovascular treatment has been demonstrated in various aortic pathologies with encouraging results[5-13] and appears very attractive in treating type B aortic dissections because of the procedure's relative safety and avoidance of thoracotomy and aortic cross-clamping.

In patients managed with medical therapy, the false lumen is subject to aneurysmal dilatation in 20% to 40%, occurring usually between 2 and 5 years after the acute phase.[14-20] Although aggressive treatment with stent graft placement in the descending thoracic aorta to exclude antegrade flow to the false lumen and avoid dilatation or rupture seems logical, the long-term durability of this therapy requires confirmation. Desired results with this modality are exclusion of false lumen, expansion of the true lumen, reduction in whole lumen in aneurysmal disease, and remodeling of the aorta.

This retrospective study assessed the clinical outcome, morphologic changes, and behavior of acute and chronic type B aortic dissection after endovascular repair. The extent of the dissection and the diameter changes in the true, false, and whole lumen were also evaluated.

MATERIALS AND METHODS

Confirmation of dissection was based on contrast-enhanced computed tomography (CT) with scanning from the supra-aortic vessels to the common femoral arteries. The CT examinations were routinely performed preoperatively and within the immediate postoperative period, and at 3, 6, and 12 months and yearly thereafter. With each CT, the extension of the dissection and diameter changes in true and false lumen were measured. The extent of thrombosis of the false lumen and evidence of false lumen antegrade/retrograde flow were assessed. Diameter changes were evaluated between the preoperative and postoperative CTs measurements. Acute dissection was defined as ≤14 days from the onset of symptoms and chronic dissection was defined as >14 days from the onset of symptoms.

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Competition of interest: none.

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CME article

0741-5214/$34.00

doi:10.1016/j.jvs.2008.01.022

The true lumen index (TLi) was calculated by dividing the true lumen diameter by the whole lumen diameter. The false lumen index (FLi) was calculated in the same manner. The desirable TLi value approaches or equals 1 (suggesting increase in true lumen to normal), whereas the sought-after FLi value approaches or equals 0 (suggesting shrinkage in false lumen). The presence of false lumen thrombus may prevent complete expansion of the true lumen, thus the TLi may not reach 1. Results for FLi were analyzed using the paired *t* test. False lumen thrombosis and retrograde flow were assessed with χ^2. Measurements of three selected aortic segments were made below the left subclavian artery, at the carina level, and at the celiac artery level. These measurements were obtained at the same level in each CT scan and were made parallel to the line of the intimal flap using a combination of anatomic parameters and digital software.

The definition of technical success was (1) coverage of initial tear/flap in the proximal descending thoracic aorta, completely eliminating the antegrade flow in the false lumen; and (2) no proximal endoleak. Retrograde flow from distal thoracic aorta, abdominal aorta, or iliac arteries was not considered a failure unless expansion of the false lumen was noted.

All patients were treated with commercially available stent grafts or under investigative protocols. All acute type B dissections received treatment except those that had responded to medical therapy, had thrombosis of the false lumen, or had < 50% collapse of the true lumen, or a combination of these. All patients presenting with chronic type B aortic dissection had treatment for aneurysmal lesions with a diameter >60 mm.

An average of three CT scans per patient were evaluated. The mean CT scan follow-up was 15.6 months (range, 1-46 months), and 25% of the entire group had at least 3 CTs and more than 24 months follow-up.

RESULTS

Stent grafts were used to treat 106 patients (mean age, 55 years, 70% men) with acute (55.7%) and chronic (44.3%) lesions; of these, 64 (60.4%) were classified as asymptomatic and 42 (39.6%) as symptomatic. Symptomatic patients were defined as those with organ or limb malperfusion, intractable pain, and hemodynamic instability. All patients included in this study had significant comorbidities (Table I).

Table I. Patient comorbidities

Comorbidities	*No.*	*%*
Hypertension	106	100
Coronary artery disease	38	36
Peripheral artery disease	40	38
Diabetes mellitus	8	7
COPD	48	45
Cerebrovascular disease	10	9
Renal insufficiency[a]	21	20
End-stage renal disease	2	2
Obesity	9	8
Tobacco use[b]	62	58

COPD, Chronic obstructive pulmonary disease.
[a]Defined as >1.5 mg/dL.
[b]Any use during lifetime.

The indications for endovascular repair were acute symptoms in 39 patients (36.7%) and chronic dilatation in 47 (44.3%). Treatment was offered to 20 acute asymptomatic patients (18.8%) because of significant collapse of the true lumen in hopes of preventing future sequelae of aneurysmal dilatation. Also treated were 39 symptomatic patients (36.7%) with acute emergent type B aortic dissection, including 10 (9.4%) with rupture, 8 (7.5%) with malperfusion (3 renal and 7 lower extremity ischemia; 2 patients having compromise of both), and 21 (19.8%) with intractable pain. The remaining three symptomatic patients had chronic dissections with pain due to aneurysmal dilatation. Four Marfanoid patients were treated, and two required later conversion to open repair.

Dissections extended to the thoracic aorta in 35 patients (33.0%), to the visceral arteries in 22 (20.7%), to the abdominal aorta in 19 (17.9%), and into one or both iliac arteries in 30 (28.3%). The left renal artery flow came from the false lumen in 14 patients (13.2%). The primary intimal tear was just distal to the left subclavian artery in 102 patients (96.2%), whereas in the remaining four (3.7%), the entry tear was in the middle third of the descending aorta.

Deployment of the stent grafts was successful in all patients. Treatment was with one stent graft in 57 patients (54%) and with two stent grafts in 40 (37.7%). The remaining nine patients received three or four grafts. Gore TAG stent grafts (W.L. Gore & Associates, Flagstaff, Ariz) were used in 84 patients (79.2%), Endofit stent grafts (Endomed Inc, Phoenix, Ariz) were deployed in 16 (15.0%), and Talent endoprostheses (Medtronic Vascular, Santa Rosa, Calif) were placed in six (5.6%). The diameter of the stent graft was calculated from the largest diameter of the proximal landing zone and oversized by 10% to 20%.

The preoperative mean (range) sizes of the whole lumen proximally was 49 (29-95), middle aorta was 42 (13-62), and distally was 34 (14-51) mm. After repair, the means (ranges) were 44 (26-82), 40 (13-64), and 33 (16-50) mm, respectively. The decrease in the proximal and middle aorta whole lumen was statistically significant ($P <$ .0001 and $P = .0148$). The whole lumen increased in 14 of the 106 patients (13.2%), occurring in nine chronic and five acute cases. Seven patients had a minimal increase in the whole lumen, with no identifiable endoleaks, remaining near the size at initial presentation and < 5 cm. These were managed conservatively. Six of the remaining patients had endovascular repair, one had open repair, and one had late open repair 5 years postoperatively after an initially successful endovascular repair.

The preoperative mean (range) sizes of the true lumen were 29 (10-48), 26 (2-55), and 21(5-47) mm, and postoperatively were 35 (18-44), 32 (13-43), and 24 (10-50) mm. The increase in the true lumen in all three regions was statistically significant after repair ($P <$ .0001, $P <$ .0001,

Table II. Actual lumen measurements before and after repair

	Before repair			*After repair*			P		
	Prox	*Mid*	*Dis*	*Prox*	*Mid*	*Dis*	*Prox*	*Mid*	*Dis*
True lumen							<.0001[a]	<.0001[a]	.0006[a]
Minimum	10	2	5	18	13	10			
Maximum	48	55	47	44	43	50			
Mean	29	26	21	35	32	24			
False lumen							<.0001[a]	<.0001[a]	.0019[a]
Minimum	0	0	0	0	0	0			
Maximum	63	49	36	46	47	37			
Mean	16	15	12	7	8	9			
Whole lumen							<.0001[a]	.0148	.2890
Minimum	29	13	14	26	13	16			
Maximum	95	62	51	82	64	50			
Mean	49	42	34	44	4033				

Dis, Distal third; *Mid*, middle third; *Prox*, proximal third.
[a]Significant *P* value.

Table III. Comparison of overall lumen indices preoperatively and postoperatively, and acute versus chronic dissection

	True lumen index[a]			*False lumen index*[a]		
Thoracic level	*Pre-op*[a]	*Post-op*	P	*Pre-op*	*Post-op*	P
Overall						
P/3	0.45	0.88	<.0001	0.41	0.06	<.0001
M/3	0.42	0.81	<.0001	0.44	0.10	<.0001
D/3	0.42	0.74	<.0001	0.42	0.21	<.0001
Dissection type						
Acute						
P/3	0.46	0.92	<.0001	0.41	0.06	
M/3	0.47	0.89	<.0001	0.43	0.06	<.0001
D/3	0.46	0.78		0.49	0.18	
Chronic						
P/3	0.34	0.80		0.80	0.09	
M/3	0.32	0.66		0.62	0.22	
D/3	0.30	0.67		0.61	0.28	

[a]Values in acute, chronic, and overall group. Desirable true lumen index, 1.0; and false lumen index, 0.

and $P = .0006$, respectively). There was no change in true lumen size in five patients (4.7%) and a decrease in two (1.9%). Of the latter, the dissections were chronic in one patient and acute in the other, and both required endovascular reintervention to repair the compromise of the true lumen. The mean (range) sizes of preoperative false lumen were 16 (0-63), 15 (0-49), and 12 (0-36) mm, and these decreased postoperatively to 7 (0-46), 8 (0-47), and 9 (0-37) mm. The decrease in false lumen size in all three regions was statistically significant ($P < .0001$, $P < .0001$, $P = .0019$; Table II). There was an increased false lumen size in 8 patients (7.5%), half of which were chronic cases. Five required repair because of expansion—four endovascular at 3, 3, 12, and 60 months, respectively, and one open repair at 72 months.

The overall average preoperative TLi was 0.45 (0.46 in acute, 0.34 in chronic) in the proximal third of the thoracic aorta, 0.42 (0.47 in acute, 0.32 in chronic) in the middle third, and 0.42 (0.46 in acute, 0.30 in chronic) in the distal third. Postoperatively, the overall average TLi increased to 0.88 (0.92 in acute, 0.80 in chronic) in the proximal third, to 0.81 (0.89 in acute, 0.66 in chronic) in the middle third, and to 0.74 (0.78 in acute, 0.67 in chronic) in the distal third (Table III). The increase in TLi was statistically significant at all three levels ($P < .0001$). After repair, the increase in the proximal and middle TLi was significant in acute cases (Table III).

The overall average preoperative FLi was 0.41 (0.41 in acute, 0.47 in chronic) in the proximal third, 0.44 (0.43 in acute, 0.47 in chronic) in the middle third, and 0.42 (0.49 in acute, 0.31 in chronic) in the distal third. The overall average postoperative FLi decreased to 0.06 (0.06 in acute, 0.09 in chronic) in proximal third, 0.10 (0.06 in acute, 0.22 in chronic) in middle third, and 0.21 (0.18 in acute, 0.28 in chronic) in the distal third (Table III). The decrease in FLi was statistically significant at all three levels ($P < .0001$, $P < .0001$, and $P = .0008$); Table III).

Complete false lumen thrombosis of the descending thoracic aorta without evidence of antegrade or retrograde flow occurred in 69 (65.1%) patients (Table IV). According to the DeBakey classification of IIIA (above the diaphragm) and IIIB (below the diaphragm), there were 25

Table IV. Comparison of dissection extension (DeBakey classification) with false lumen and retrograde flow outcome

		Graft coverage, No. (%)			*False lumen thrombosis, No. (%)*		
DeBakey Extent[a]	*No (%)*	*Prox*	*Mid*	*Dist*	*Prox*	*Mid*	*Dist*
Acute							
IIIA	25 (23.5)	4	6	15	0	2	23
IIIB	34 (32.0)	1	10	23	3	9	22
Total	59 (55.6)						
Chronic							
IIIA	14 (13.2)	0	3	11	0	4	9
IIIB	33 (31.1)	1	15	17	5	13	15
Total	47 (44.3)						
Total overall	106 (100)	6 (5.6)	34 (32)	66 (62.2)	8 (7.5)	28 (26.4)	69 (65.1)

Abd-Ao, Abdominal aorta; *Dist*, sistal third; *Mid*, middle third; *Prox*, proximal third.
[a]Extent IIIA, above diaphragm; extent IIIB, below diaphragm.

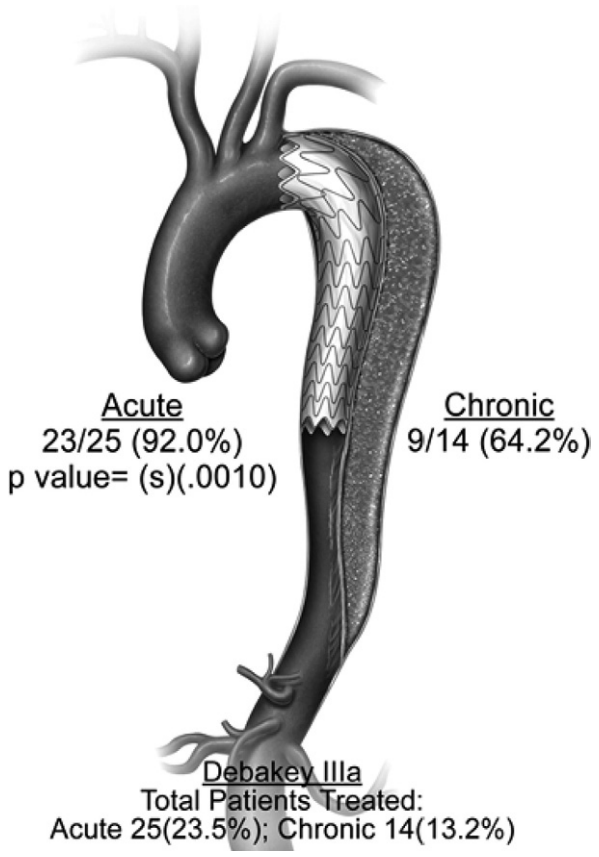

Fig 1. Complete false lumen thrombosis of thoracic aorta (DeBakey IIIA).

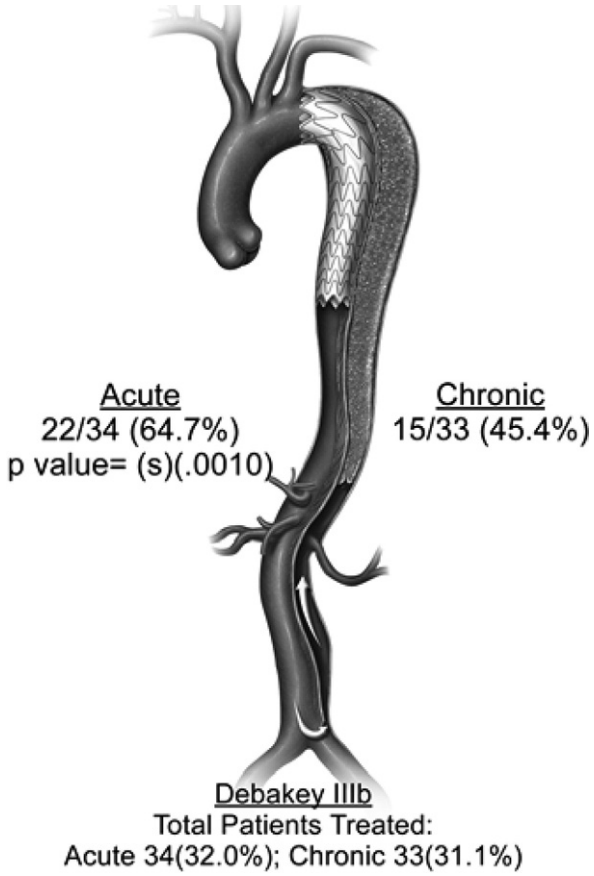

Fig 2. Complete false lumen thrombosis of thoracic aorta (DeBakey IIIB).

acute and 14 chronic type IIIA, and 34 acute and 33 chronic type IIIB cases (Table IV). False lumen thrombosis occurred in 23 (92.0%) of acute type IIIA, 22 (64.7%) of acute type IIIB, 9 of chronic type IIIA, and 15 of chronic type IIIB cases (Figs 1 and 2).

Thrombosis of the false lumen was significant, or more common, for acute ($P = .0010$) and type IIIA cases ($P = .0133$). False lumen thrombosis of acute dissections occurred between 1 day and 13 months (median, 2 days). False lumen thrombosis of chronic dissections occurred between 1 day and 12 months (median, 5 days). In 28 patients (26.4%), thrombosis of the false lumen occurred from the proximal to the middle third of thoracic aorta without evidence of antegrade flow. Eight (7.5%) of those had proximal thrombosis only, and an endoleak had developed in one. No retrograde flow in the thoracic aorta was

Table IV. Continued.

P	*Retrograde flow, No.(%)*					P
	Prox	*Mid*	*Dist*	*Abd-Ao*	*None*	
<.001	2	0	3	0	20	<.001
<.001	2	4	12	9	7	<.001
<.001	1	1	4	0	7	<.001
<.001	4	6	14	6	2	<.001
	9 (8.4)	11 (10.3)	33 (31.1)	15 (14.1)	36 (36.9)	

Table V. Extent of dissections and complications

DeBakey classification[a]	*No. (%)*	*Subclavian coverage, No. (%)*	*CVA, No. (%)*	*Paraplegia, No. (%)*	*Endoleak, No. (%)*	*Mortality, No. (%)*
Acute						
IIIA	25 (23.5)	1	1	1	1	0
IIIB	34 (32.0)	3	2	2	4	1
Total	59 (55.6)					
Chronic						
IIIA	14 (13.2)	0	0	0	1	0
IIIB	33 (31.1)	8	2	1	1	7
Total	47 (44.3)					
Total overall	106 (100)	12 (11.3)	5 (4.7)	4 (3.7)	7 (6.6)	8 (7.5)

CVA, cerebrovascular accident.
[a]DeBakey IIIA, above diaphragm; IIIB, below diaphragm.

seen in 36 of 106 cases (36.9%). Absence of retrograde flow was statistically significant for acute ($P = .0124$) and type IIIA ($P = .0124$; Table IV).

Seven patients (6.6%) had postoperative endoleaks, six had proximal type I endoleaks, and one had a type III endoleak. Five of these six type I endoleaks were identified perioperatively, with an attempt to treat intraoperatively. The sixth type I endoleak was caused by graft collapse at 72 hours. Of the six type I endoleaks, an additional endoluminal graft was placed in two patients that successfully sealed the endoleak. Attempted endoluminal treatment failed in the four other patients, two of whom died of multisystem organ failure and one of arrhythmia before reintervention, and the other required open repair. Three of the type I endoleaks occurred in patients with acute dissections, and the remaining three in chronic cases. Late endoleak (>30 days) occurred in three patients, of whom two were type I, and one patient had both type I and type II endoleaks. All were treated with stent grafts, and coil embolization of the subclavian artery was added in the patient with type II endoleak.

Cerebrovascular accident (CVA) occurred in five patients, (4.7%), including a patient who sustained intracranial bleeding (Table V). Spinal cord ischemia occurred in four patients (3.8%), of which one (1.0%) had paresis and three (2.8%) had paraplegia. Two patients received spinal drains, and one regained sensation, the other resolved the deficit in one limb and improved in the other. The remaining two patients were critically ill; one had intracranial bleeding, the other had bowel ischemia, and neither received a drain. Three of the four incidences of spinal cord ischemia occurred after acute emergency cases. One of the patients with paraplegia had a contained rupture and the left subclavian artery was covered. The other patient resented with visceral and lower extremity malperfusion. The left subclavian artery was intentionally covered in 12 patients (11.3%) without revascularization. An additional six patients had left carotid-subclavian bypass before endoluminal stent grafting (Table V).

Eight patients (7.5%) died. Five deaths occurred with emergency cases, and included two (1.8%) intraoperative deaths caused by contained ruptures that became free ruptures. Three deaths followed elective cases, consisting of two fatal arrhythmias and one intracranial hemorrhage.

DISCUSSION

Conservative management in the form of analgesia and aggressive blood pressure control is the mainstay of therapy for acute type B aortic dissections, with intervention reserved for complicated cases.[21] This approach is based on the observation that mortality rates <10% can be achieved with pharmacotherapy alone, in contrast to the 50% to 60%

mortality rate seen with early surgical intervention for acute type B aortic dissection.[21] The prognosis, however, is poor for chronic type B aortic dissection managed conservatively. The false lumen is subject to aneurysmal development in 20% to 40% of the chronic dissection cases during the next 2 to 5 years after the acute phase.[14-21]

In response to the high morbidity and mortality associated with surgical aortic repair, especially in the presence of ischemia, endovascular management of acute and chronic aortic dissection was pioneered in the past decade, with reported survival rates as high as 100%.[10,11,22] The approach to endovascular repair of type B aortic dissection involves placement of the stent graft across the primary entry tear with the objective of depressurizing the false lumen and inducing proximal false lumen thrombosis. The false lumen flow often persists in the lower thoracic and abdominal aorta due to re-entry tears or intimal fenestrations and is retrograde in orientation. Arresting the antegrade flow is the primary function of the stent graft.

By covering the proximal tear in acute dissections, we obtained significant shrinkage of the false lumen within an average of 6 months; in chronic dissections, shrinkage occurred at average of 12 months. The delay in shrinkage in the latter cases may be due to the stiffness of the septum that separates both lumens. The reduction in the false lumen and FLi was statistically significant after stent grafting, suggesting shrinkage of the false lumen. In addition, the whole lumen decreased as well, suggesting a reduction in aneurysmal dilatation. We believe covering the proximal tear is the most important aspect of successful stent graft treatment of the type B dissection and that it is key to resolving antegrade flow and establishing thrombosis of the false lumen. In addition, acute dissection and a limited extent of dissection (type IIIA), correlated with increased likelihood false lumen thrombosis, whereas chronic dissections and type IIIB dissections correlated with persistent retrograde flow (Table IV).

Schoder et al[23] recently reported that 90% of patients were treated successfully for acute type B aortic dissection with thrombosis of the false lumen in the stented segment; however, false lumen perfusion distal to the stent graft resulted in a diameter increase in some patients. In contrast, we found persistent false lumen flow in the distal thoracic and abdominal aorta was not associated with an increase in the diameter of the false lumen at 15.6 months.

As noted, false lumen thrombosis occurred significantly more often with acute and type IIIA dissections; this, along with the likelihood of no retrograde flow in the thoracic aorta, is encouraging and may be an incentive to treat acute type IIIA dissections. Perhaps a randomized study may be necessary comparing endoluminal grafting and optimal medical management vs medical management alone for this group. We did not observe aorta diameter expansion in segments distal to the stented region; however, longer follow-up is also necessary to determine if the false lumen diameter increases. Expansion of the aorta was seen in those who developed type I endoleaks. We cannot speculate whether the endoleak led to aneurysmal expansion or the expansion led to endoleak. At 1year, Nienaber et al[24] showed some type of stent-induced aortic false lumen thrombosis in nearly all of the stent graft patients treated, but these are unpublished data.

The incidence of spinal cord injury in our series compared favorably with the current outcomes of open repair in acute and chronic cases.[14] The paraplegia rate of 2.8% for treatment with dissections is, however, somewhat higher than the overall paraplegia rate of 1.5% of our entire thoracic series.[25] To prevent neurologic symptoms, we medically induce hypertension to maintain mean arterial pressure >90 mm Hg or above the patient's baseline systolic pressure, only treating a systolic blood pressure >180 mm Hg. If neurologic symptoms develop, hypertension is maintained, steroids are given, and if the patient fails to show improvement ≤1 to 2 hours, a spinal drain is placed.

Debate exists regarding the need for extensive coverage of the descending thoracic aorta to gain immediate expansion of the true lumen and to eliminate retrograde flow or malperfusion, or both. In this series, in which endovascular grafting was limited to coverage of the proximal entry tear, the increase in true lumen and TLi was statistically significant, particularly at the proximal and middle regions, and supports this approach. Although the paraplegia rate is still fairly low, the "extensive coverage" approach may increase the incidence of spinal cord injury.

We prefer to use a single graft; however, if the entry tear remains patent, the true lumen collapses, or a second entry site is identified, it may be necessary to place additional grafts. The placement of a bare metal stent in the distal thoracic aorta is an option when significant collapse of the true lumen persists despite coverage of the proximal tear with an endograft. This technique can be performed during the initial procedure or as a staged procedure as described by Nienaber to enhance expansion of the true lumen.[26] With this approach, intercostal artery perfusion is maintained, while expansion of true lumen is observed.

We also covered the left subclavian artery in 18 patients (17.0%) to effectively close the proximal tear that communicated with the false lumen. Six patients received a left carotid–subclavian bypass before endoluminal grafting. Spinal cord injury occurred in one of these patients, possibly because collateral flow was sacrificed; however, this is difficult to assess, because the patient also had a ruptured thoracic aorta and episodic hypotension.

Some investigators have focused endovascular management on acute thoracic dissection because remodeling changes seem more likely to occur in early stages of this condition.[24,27] Our results, however, demonstrate that both acute and chronic dissection can be treated successfully with stent grafts. The indications to treat acute dissection (36.7%) were malperfusion, rupture, an emergency scenario, or a severely collapsed true lumen, whereas chronic dissections (30%) were treated to manage aneurysmal dilatation. When chronic dissections were effectively treated, and hence, there was resolution of false lumen antegrade flow, the true lumen of the thoracic aorta re-

mained stable and the false lumen and whole lumen decreased.

CONCLUSION

The endovascular approach is feasible for the management of acute and chronic type B dissections. This approach permits gradual aortic remodeling, and in some cases, results in complete resolution of the false lumen. Endoluminal grafting has the potential to become an adjunctive therapy to antihypertensive agents in the management of thoracic type B dissections.

AUTHOR CONTRIBUTIONS

Conception and design: JR
Analysis and interpretation: DO, JR, LL, GW, VR, ED
Data collection: DO, LL
Writing the article: JR, LL, DO
Critical revision of the article: JR, DO, ED
Final approval of the article: JR, DO, LL, GW, VR, ED
Statistical analysis: DO, JR
Obtained funding: Not applicable
Overall responsibility: JR

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Submitted May 30, 2007; accepted Jan 7, 2008.