TEVAR for Symptomatic Stanford B Dissection: A Systematic Review of 30-Day Mortality and Morbidity

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Abstract

Background The aim of this study was to determine morbidity and 30-day mortality rates of thoracic endovascular aortic repair (TEVAR) for Stanford B dissection over a 16-year period and determine if these rates have improved with better stent-graft technology and surgical technique.

Methods Electronic databases were searched in all languages and a systematic review conducted. A comparison of the early (1998–2007 = 787 patients) and later (2007–2013 = 787 patients) halves of the patient population was done. Studies were chosen based on availability of details regarding morbidity and mortality. Ambiguous studies were excluded.

Results A total of 69 suitable studies published between 1998 and 2013 (1,574 patients) were examined including 1 randomized control trial, 55 retrospective studies, 3 prospective, 1 mixed, and 9 case reports. Overall mortality and morbidity rates for TEVAR was 8.07% ($n = 127$) and 30.8% ($n = 485$), respectively. The stent-graft-related death rate was 6.20% (97 cases excluding medically related deaths). The endoleak rate was 5.9% of which most were type I. Major complications include stroke (2.7%), paraplegia (1.9%), partial thrombosis of false lumen (2.5%), retrograde type A dissection (3.1%), visceral malperfusion (2.0%), conversion to open intervention (1.9%), and secondary intervention (4.1%). The stent-graft-related mortality rate increased in the 2007 to 2013 group compared with the 1998 to 2007 group (56.2 vs. 24% of patients who died; $p < 0.05$). There was also an increase in the overall morbidity rates from 25% (1998–2007) to 36.6% (2007–2013) but did not reach statistical significance $p > 0.05$.

Conclusion Mortality and morbidity rates for TEVAR seemed to have increased over the past 16 years despite improved technology and surgical technique. This may be explained by the increasing liberal use of TEVAR intervention and quite possibly better reporting. The current data are highly heterogenous making it difficult for solid conclusions to be drawn. The only way forward is through better data registries and well-designed clinical trials.

Keywords

- ▶ endovascular repair
- ▶ Stanford type B dissection
- ▶ acute and chronic aortic dissection

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Introduction

The first endoluminal procedure was conducted by Dotter and Judkins in 1964¹ and progressed slowly up to 1991 when Parodi et al² made significant advancements in the treatment of abdominal aortic aneurysms. Techniques and technology evolved over the years and spread into the various branches of vascular surgery. Up to 2013, major studies on type B dissection included meta-analyses by Eggebrecht et al³ in 2006, Walsh et al⁴ in 2008, Xiong et al in 2009,⁵ and Zhang et al in 2012,⁶ as well as the INSTEAD^{7,8} and IRAD⁹ trials by Nienaber et al and Fattori et al, respectively. This article is intended to be a systematic review of available data for 16 years from 1998 to 2013 with regard to morbidity and mortality with a comparison of the first and later halves of the patient population to determine if rates have improved with the advent of improved technology and surgical techniques.

Methodology

A systematic review was conducted using separate search strategies in the PubMed, MeSH thesaurus, and Cochrane databases. The following registries were included: EUROSTAR Registry, United Kingdom Thoracic Endograft Registry, and International Registry of Acute Aortic Dissection. The PRISMA guidelines were followed as recommended for systematic review reporting.

PICOS Statement

The study was conducted focusing on the 30-day mortality and morbidity of symptomatic type B acute and/or chronic aortic dissections (S-B-AD). Suitable studies for 16 years from 1998 to 2013 were selected for analysis. The objectives of the study were to determine overall rates for the 16-year period and to perform a comparison of the first (787 patients from 1998 to 2007) and later (787 patients from 2007 to 2013) halves of the patient population to determine if rates have improved with the advent of improved technology and surgical techniques.

Protocol and Registration

No review protocol was created online.

Eligibility Criteria

Suitable studies were chosen based on the accuracy of documentation of 30-day mortality and morbidity specifically for the S-B-AD. Once the information was clearly documented, the study was included.

Information Sources

A systematic review was conducted using separate search strategies in the following databases: PubMed, MeSH thesaurus, and Cochrane databases. The Embase, Ingenta, Zetoc, and Ovid databases were also used as well as the following registries were included: EUROSTAR Registry, United Kingdom Thoracic Endograft Registry, and International Registry of Acute Aortic Dissection.

Search

A standard PubMed search using the “Advanced Search Option” for matching terms was first used to obtain the meta-analyses, review articles, and case series on the treatment of S-B-AD, whether acute or chronic as well as involving consequent aneurysm formation or not.

The search topics included the following:

1. “Type B dissection and descending thoracic dissection”
2. “Medical treatment”
3. “Open surgery”
4. “Endovascular/stent repair”

Combinations were done using the “AND” option. This obtained the following results:

- “Type B dissection and descending thoracic dissection” yielded 340 articles.
- The 340 articles were then combined using the “AND” option in the search box yielding 108 for medical treatment, 69 for open surgery, and 44 for endovascular/stent repair. There were 51 suitable articles selected for analysis.

Another search strategy was employed by searching through a MeSH search in PubMed combining “Aortic Aneurysm, Thoracic [MeSH]” AND “Stents [MeSH]” AND “Aneurysm, dissecting [MeSH].” This yielded 441 articles from which the search was confined to clinical trials (three selected), review articles (two selected), randomized controlled trials (one selected), and meta-analyses (six selected).

The databases from the Cochrane library, Embase, Ingenta, Zetoc, and Ovid did not yield any new relevant information; however, hand-searching relevant references obtained via a standard PubMed search for the topic yielded a further 14 relevant articles.

Cumulatively, 69 suitable studies were selected. The six meta-analyses and two reviews were excluded to avoid duplicating data (►Fig. 1). A total of 1,574 patients were treated for S-B-AD. There were no ethical considerations. Results were entered into SPSS 20 (IBM SPSS 20 STATISTIC, United States) and analyzed using Pearson chi-square testing.

Results

A total of 69 articles with 1,574 patients were selected for the 16-year period, 1998 to 2013. Most included a mix of various types of descending thoracic aortic pathology including acute rupture, chronic aneurysmal disease, Stanford type A and B dissections, and traumatic disruption. Selected articles contained a detailed breakdown of the results, patient demographics, and details of complications associated with S-B-AD (►Table 1).

Thoracic endovascular aortic repairs (TEVARs) were done in the following countries: 11 (16%) studies from China, Japan, and Korea in the Far East; 16 (23%) from the United States; and 38 (55%) from European region including Germany, Austria, France, United Kingdom, Switzerland, Norway, Sweden, Italy, Belgium, and Denmark and Turkey. There were two from Brazil and one from New Zealand and Canada each (6%).

Fig. 1 PRISMA flow diagram showing search strategy.

The largest series were from Kische et al,¹⁰ Leurs et al,¹¹ and Nienaber et al⁷ with 171, 106, and 72 cases, respectively. The other studies contained small and intermediate sized populations and nine case reports were also included. Tests for heterogeneity in patient demographics in terms of age, ASA (American Society of Anaesthesiologists Physical Status Classification) grade, and comorbidities were not conducted because there was great variation in the way in which data were recorded.

The mean age range was 62 years with a range of 54 to 75, a median of 60, and mode of 59 with the age stated in only 36 articles (52%). Studies selected included the following according to year: 1998 = 1, 1999 = 2, 2000 = 4, 2001 = 3, 2002 = 7, 2003 = 7, 2004 = 7, 2005 = 2, 2006 = 7, 2007 = 6, 2008 = 10, 2009 = 6, 2010 = 0, 2011 = 2, 2012 = 2, and 2013 = 3.

In terms of device, the most commonly used devices were the Medtronic Talent (Medtronic, Minneapolis, Minnesota, United States) and Gore TAG Thoracic Endoprosthesis (Gore Medical, Flagstaff, Arizona, United States) devices in 30 studies (43%) followed by the Zenith Excluder (Cook Medical, Bloomington, Indiana, United States) and the Valiant (Medtronic, Minneapolis, Minnesota, United States) types. The device used was not stated in 36 studies.

Of 1,574 patients treated for Stanford B dissections, the overall 30-day mortality was 8.07% ($n = 127$) (►Table 1).

These were related to a variety of causes including procedure-related false aneurysm rupture, retrograde type A dissection, ruptured type I endoleak, persistent visceral and lower limb malperfusion, aorto-esophageal fistula, further type B dissection, conversion to open surgery, as well as ruptured access vessels. There were 24 (1.52%) medical-related death within 30 days. The total number of deaths directly related to stenting was 97 (6.20%) (excluding medical-related deaths) within 30 days of TEVAR (►Table 2).

Procedure-related morbidities included retrograde type A dissection (49), visceral malperfusion (32), lower limb ischemia (5), aorto-esophageal fistula (4), stroke (41), paraplegia/paraparesis (30), type I endoleak (82), type II endoleak (11), partial thrombosis of the false lumen (39), conversion to open surgery failed procedure (30), secondary interventions (65), renal failure (31), device migration/modular disconnection/junctional endoleaks (7), emboli (4), overstenting of the celiac artery (1), postimplantation syndrome (3), and failure (2). The overall morbidity rate for the patient population was 30.8% (►Table 3).

With regard to the comparison of the 1998 to 2007 period (787 patients) with the 2007 to 2013 period (787 patients), the findings were quite significant. The stent-graft-related mortality rates (which includes the subset of patients who died; $n = 127$) doubled in the 2007 to 2013 period versus the 1998 to 2007 period; 56.2 versus 24.0%, respectively

Table 1 Summary of selected articles on TEVAR in S-B-AD

Author	Year published/ country	Duration/type	Clinical inclusion criteria	Indications	Device	Age	N	30-d mortality	Cause for mortality	Morbidity
Hanna et al ¹⁸	2014/United States	2005–2012/retrospective, single institution	Acute complicated type B aortic dissection	Rupture (10) Malperfusion (24) Refractory pain (17) (one patient had malperfusion and rupture)	Gore TAG Zenith TX2 Talent Valiant	59 (53.3–70.0)	50	0	–	Adjunctive procedures for malperfusion ×10 Stroke ×1 Paraplegia ×1 Renal failure ×4 Type I endoleak ×13 with 17 procedures Type II endoleak ×2 Persistent FL ×4 Retrograde type A dissection ×1
Wilkinson et al ¹⁹	2013/United States	1995–2012/retrospective, single institution	Acute type B dissection	Rupture (12) Rapid expansion (21) Uncontrolled pain (10) Aortic size > 5 cm (18) Refractory hypertension (1) Malperfusion (7) Multiple indications (18)	Gore TAG Talent Valiant Cook TX2	70.1 ± 12.3	49	5	Rupture ×2 Impending rupture with emergent open conversion and stroke ×1 Acute on chronic aneurysmal dissection ×1 Renal, visceral, and lower limb malperfusion ×1	Stroke ×3 Renal failure ×2 Paraplegia ×2
Minami et al ²⁰	2013/Japan	2000–2012/retrospective, single institution	Acute type B dissection	Acute dissection	NS	71 ± 15	9	0	–	Stroke ×1
Lombardi et al ²¹	2012/United States, Europe, and Australia	2007–2009/prospective, nonrandomized, multicenter	Acute (24) (< 14 d) and subacute (6) (15–30 d) dissection	Malperfusion, periaortic effusion/hematoma, impending rupture, resistant hypertension, persistent pain, aortic growth	Zenith TX	58 (35–83)	30	2	Visceral malperfusion and right common iliac artery (day 11) Renal artery malperfusion (day 20 at home)	Stroke intraoperatively ×1 Retrograde type A aortic dissection ×2 (31, 365 d) Secondary intervention ×7
Heijmen et al ²²	2012/The Netherlands	2009–2010/mixture of retrospective and prospective at 15 sites in Europe	Acute dissection	Pain, dissection, aortic wall hematoma	Valiant Captivia	58.4 ± 13.5 (32–83)	19	4	3 device-related multiorgan failure (day 20), retrograde dissection (day 22), rupture (day 14), rupture (day 1)	Paraplegia ×1 Stroke ×4 Retrograde type A dissection ×2 Type I endoleak ×2 Type II endoleak ×3 Respiratory ×2 Cardiac ×1 Bowel ischemia ×1 Vessel rupture ×1

Table 1 (Continued)

Author	Year published/ country	Duration/type	Clinical inclusion criteria	Indications	Device	Age	N	30-d mortality	Cause for mortality	Morbidity
Mitchell et al ²³	2011/United States	2007–2010/retrospective	Acute complicated type B dissection Ruptured descending thoracic aneurysm Traumatic aortic disruption	Visceral malperfusion	Gore TAG (Excluder)	Mean = 67 (50–77)	4	1	Postop pneumonia and respiratory failure after successful TEVAR	Stroke ×1 Paraplegia (spinal ischemia) ×1 Renal failure ×1
Fioranelli et al ²⁴	2011/Brazil	2004–2007/retrospective, case series	Complicated type B aortic dissections Acute phase = 10 (< 14 d) Chronic phase = 13(> 14 d)	Rupture (6) Persistent thoracic pain (1) Thoracic pain + dilation (1) Lower limb ischemia (2)	Talent Valiant (11) Gore TAG (7) Zenith (4) Endofit (1)	Acute phase = 56.3 ± 12.3 Chronic phase = 60.8 ± 10.8	23	3	Retrograde dissection ×1 Aorto-esophageal fistula ×1 Renal failure ×1 (all related to proximal endoleak. One immediate and two massive hematomas in pleural space, died on days 3 and 12)	Type I endoleak ×4 (2 acute/2 chronic) Paraplegia = 0 Note: 8 died after 30 d (3 aorto-esophageal fistulas 2, 3, and 23 mo; 1 renal failure; 2 ischemic strokes; 1 AAA rupture; 1 ruptured false lumen)
Cambria et al ²⁵	2009/United States	2005–2007/prospective, nonrandomized	Complicated type B = 19 Ruptured degenerative aneurysm = 20 Traumatic aortic tear = 20	Renal (4) Lower limb (5) Malperfusion (1)	Gore TAG	Mean 58.9 ± 14.7	19	3	Aortic rupture ×2 Strokes ×2	Paraplegia ×3 Reintervention ×2 Conversion to open ×1 Type A dissection ×1 Stroke ×2 (1 death) Type I endoleak ×2 Bleeding ×1 Further dissection < 30 d ×2 Adverse events ×14 (cardiac 3, pulmonary 5, renal 2, cerebrovascular 4, DVT/PE/coagulopathy 2, other 8)
Nienaber et al ⁷	2009/Germany, Italy, France	2003–2005/randomized controlled trial, multicenter	Chronic type B dissection	Chronic type B dissection	Talent	61.8 (61–73)	72	5	Postprocedural rupture of access vessel ×1, malperfusion ×1, type A dissection with tamponade ×1, ruptured thoracic aorta ×1, type I endoleak ×1	Retrograde type A dissection ×1 Ruptured iliac access vessel ×3 Paraplegia ×2 Major stroke ×1 Type I endoleak ×1
Patel et al ²⁶	2009/United States	1997–2008/retrospective, institutional review	Presentation within 14 d and type B dissection with malperfusion	Malperfusion on angiography	NS	65.8	69	12	Lower extremity ischemia ×4, renal/mesenteric ischemia ×8	Renal failure ×10 Stroke ×3 Paraplegia ×2

(Continued)

Table 1 (Continued)

Author	Year published/ country	Duration/type	Clinical inclusion criteria	Indications	Device	Age	N	30-d mortality	Cause for mortality	Morbidity
Kische et al ¹⁰	2009/ Germany, Bologna, Lille, Marseille, Neuweglein, Rostock, Toulouse, and Vienna	1996–2004/retrospective, multicenter	Type B dissection (171) Thoracoabdominal type A dissection (9)	Complicated type B aortic dissection	Talent	59.6 ± 13.0	171	9	Ruptured type B dissection ×1	Retrograde extension of type B dissection after TEVAR ×4 Conversion to open ×5 Type I endoleak ×6 Enlargement of false lumen ×2 Migration ×1 Modular disconnection ×1 Fatal rupture ×7 Procedural stroke ×7 Paraplegia ×5
Day and Buckenham ²⁷	2009/New Zealand	2001–2007/retrospective, multicenter	TEVAR for type B dissection, elective thoracic aneurysms, and traumatic injury	Type B dissection	NS	NS	39	7	Hemorrhage ×3 MI ×1 Cardiac arrest ×1 Stroke ×1 Renal failure ×1 Multiorgan failure ×2 Sepsis ×1 Aneurysm rupture ×1 Type A dissection ×1 Oesophageal necrosis ×1	NS
Porcu et al ²⁸	2009/France	2002/case report	TEVAR for acute type B dissection	Type B dissection	Talent	73	1	0	–	Aneurysmal dilatation secondary to giant-cell arteritis and reintervention ×2 Type I endoleak ×1
Chemelli-Steingruber et al ²⁹	2008/Austria	1996–2008/retrospective, institutional	Acute type B dissection	Type B dissection—symptomatic	NS	NS	38	6	Late aortic rupture ×6	Type I endoleak ×9 Retrograde type A dissection ×4 Conversion to open ×3 Secondary intervention ×2 Paraplegia ×0
Mastoroberto et al ³⁰	2008/Italy	2002–2007/retrospective, institutional	Acute type B dissection	Aortic ulceration (9) Left pleural effusion (2)	Talent	NS	11	0	–	Paraplegia ×1 (30 d) Endoleak ×0 Retrograde type A dissection ×0
Flecher et al ³¹	2008/France	2007–2008/retrospective, institutional	Acute type B dissection < 14 d (7) Chronic type B dissection > 14 d (28)	Persistent pain Rupture Visceral malperfusion Lower limb ischemia Abdominal pain	NS	NS	35	5	Retrograde type A dissection ×3 (died days 2, 22 other not stated) Ruptured aorta ×2 (died days 2 and 10)	Retrograde type A aortic dissection ×3 Reintervention ×14, partial thrombosis of FL ×15 Absence of thrombosis ×4 Ancillary procedures ×4
Apple et al ³²	2008/United States	2005–2007/retrospective, case series	Thoracic aortic aneurysmal disease	Contained rupture of a type B dissection	NS	NS	1	0	–	0

Table 1 (Continued)

Author	Year published/ country	Duration/type	Clinical inclusion criteria	Indications	Device	Age	N	30-d mortality	Cause for mortality	Morbidity
Khoynezhad et al ³³	2008/United States	1998–2005/retrospective, institutional	Symptomatic type B dissections, aneurysms of descending thoracic aorta, penetrating aortic ulcers, traumatic dissection (153 patients)	Acute complicated type B dissection (pain, uncontrolled hypertension, malperfusion, rupture, or enlargement > 5 mm/y) (25) Chronic type B dissection (42)	Talent (145), AneurRx (Medtronic, Minneapolis, Minnesota, United States) (37), Gore TAG (2)	NS	67	7	Rupture Retrograde Dissection Medical (not clearly stated)	Retrograde aortic dissection ×1 (had immediate arch replacement) Stroke ×3 Type I endoleak ×1
Dagenais et al ³⁴	2008/Canada	2002–2008/retrospective, case series	Acute type B aortic dissection	Intramural hematoma Intractable pain	Valiant (Freeflo)	59.5 (40–83)	4	0	–	0
Boufi et al ³⁵	2008/France	2004–2006/retrospective	Complicated type B dissection	Type B dissection	NS	75.4 (30–94)	4	1	–	Paraplegia ×1 type I endoleak 3
Misfeld et al ³⁶	2008/Germany	1993–2006/retrospective	Type B dissection	Type B dissection	NS	55 ± 16 (25–80)	24	3	MI	Paraplegia ×2
Hager et al ³⁷	2008/United States	2008/case report	Chronic type B dissection	Type B dissection	Gore TAG	56	1	0	–	0
Neuhauser et al ³⁸	2008/Austria	1997–2007/retrospective	Type B dissection	Type B dissection	NS	NS	28	1	Open conversion	Conversion to open (retrograde type A dissection) ×4
Duebener et al ³⁹	2007/Germany	2000–2006/retrospective	Complicated acute type B dissection	Pain, rupture, rapid increase in aortic diameter, visceral malperfusion	Talent Valiant	59.3 ± 6.3 (46–66)	13	2	Persistent visceral malperfusion	Retrograde type A dissection ×1 Conversion to open ×3 Acute renal failure ×1 Hemiplegia ×3 Stent dislocation ×1
Czerny et al ⁴⁰	2007/Austria	2005–2006/retrospective	Chronic type B dissection	Aneurysms	NS	63 (50–75)	6	0	–	Type I endoleak ×1
Schoder et al ⁴¹	2007/Austria	2006/retrospective, single institution	Acute type B dissection	Rupture, contained rupture, visceral malperfusion, persistent pain, rapid increase in aortic diameter	Talent, Gore Excluder	57 (33–83)	24	3	Aortic rupture, cardiac, retrograde type A dissection, and cardiac tamponade	Paraplegia ×2 Retrograde type A dissection ×1 Lower Limb amputation ×1 Renal failure ×2 Small bowel and right hemicolectomy ×1
Sharif et al ⁴²	2007/UK	2007/case report	Acute type B dissection	Pain, uncontrolled hypertension	Gore TAG	57	1	0	–	0
Bingol et al ⁴³	2007/Turkey	2002–2005/retrospective, institutional	Mixture of thoracic aneurysms	Type B dissection	Talent, Gore Excluder	NS	3	0	–	0

(Continued)

Table 1 (Continued)

Author	Year published/country	Duration/type	Clinical inclusion criteria	Indications	Device	Age	N	30-d mortality	Cause for mortality	Morbidity
Slovut et al ⁴⁴	2007/United States	2007/case report	Type B dissection	Pain and uncontrolled hypertension	Gore TAG	61	1	0	-	0
Song et al ⁴⁵	2006/United States	1999–2005/retrospective, institutional	Acute (25) and chronic (17) type B dissection	Chest pain (11), aneurysmal enlargement (11), uncontrolled hypertension, progression of dissection, rupture (5), visceral malperfusion, aortoenteric fistula	AneuRx (5) Talent (37)	64 ± 14 (35–88)	42	0	-	Type I endoleak × 6 Secondary interventions × 18 Junctional leaks × 3 Continued perfusion of false lumen from distal reentry sites × 3 Conversion to open for type A retrograde dissection × 4
Kaya et al ⁴⁶	2006/The Netherlands	2002–2006/retrospective, institutional	Acute aneurysms and dissection	Pain, visceral malperfusion	Talent, Gore Excluder	NS	12	2	Visceral malperfusion × 1 Rupture × 1	Type II endoleak × 2 Stroke × 1
Xu et al ⁴⁷	2006/Beijing	2001–2005/retrospective, institutional	Acute type B aortic dissection	Pain, uncontrolled hypertension	Talent, Vascor, Suzhou, China), Aegis (Microport, Inc., Shanghai, China), Grikim Advanced Materials, Inc., Beijing, China), Ankura (Life- tech Scientific Inc., Shenzhen, China)	NS	63 (59 treated with- in 2 wk)	2	Retrograde type A aortic dissection × 1 Renal failure × 1	Retrograde type A dissection × 3 Stroke × 1 Renal failure × 2 Transient left arm ischemia × 1 Type I endoleak × 3 Type II endoleak × 1
Augustine et al ⁴⁸	2006/Germany	2005/case report	Type B aortic dissection and aneurysm	Pain, penetrating ulcer, and hypertension	NS	59	1	0	-	Overstenting LCCA and LSCA × 1
Holst et al ⁴⁹	2006/Denmark	2005/retrospective	Type B dissection	Type B dissection	NS	NS	3	0	-	0
Duda et al ⁵⁰	2006/Germany	1997–2000/prospective controlled	Type B dissection, ruptures, aneurysms	Type B dissection	Talent, Gore TAG	NS	5	0	-	Retrograde type A dissection × 1
Wheatley et al ⁵¹	2006/United States	2000–2004/retrospective	Acute (22) and chronic (14) type B dissection	Type B dissection	Gore TAG	NS	36	3	-	Type I endoleak × 5

Table 1 (Continued)

Author	Year published/ country	Duration/type	Clinical inclusion criteria	Indications	Device	Age	N	30-d mortality	Cause for mortality	Morbidity
Wang and Li ⁵²	2005/China	2004/ retrospective	Type B dissection	Type B dissection	Not stated	NS	16	0	–	Type I endoleak ×1 Conversion to open ×1
Dialetto et al ⁵³	2005/Italy	1999–2004/ retrospective	Type B dissection	Type B dissection	Not stated	59.5 ± 11.5	28	3	NS	Medical problems ×3 Partial thrombosis ×7 Aneurysmal dilatation ×1
Sujimoto et al ⁵⁴	2004/Japan	2003/case report	Type B dissection	Type B dissection	Not stated	66	1	0	–	0
Joung et al ⁵⁵	2004/Korea	2003/case report	Type B dissection	Type B dissection	Not stated	54	1	0	–	Reintervention ×1
Bortone et al ⁵⁶	2004/Italy	1999–2003/ retrospective	Complicated type B dissec- tion (43), aneurysms, posttraumatic	Complicated type B dissection	Talent, Gore Excluder, Zenith (Cook), EndoFit Thoracic Stent Graft (Le Maitre, Germany)	NS	43	3	Retrograde type A dissection ×3	Type II endoleak ×2 Paraplegia ×0 Retrograde type A dissec- tion ×3
Leurs ¹¹	2004/UK	1997–2003/ret- rospective multi- center (EURO- STAR and UK Thoracic Endog- raft Registry)	Aneurysm or dissection	Type B dissection	Talent, Gore Excluder, Zenith, EndoFit	NS	106 (type B dissection)	11	Rupture from type I endoleak in ×8	Type II endoleak ×1 Reintervention ×1 Paraplegia ×1 Stroke ×2
Iannelli et al ⁵⁷	2004/Italy	2001–2002/ retrospective	Type B dissection	Type B dissection	NS	NS	8	0	–	Overstenting LSCA ×2 Overstenting celiac artery ×1 Postimplantation syndrome ×3
Hansen et al ⁵⁸	2004/United States	2001–2003/ retrospective	Type B dissec- tion (16 acute, 8 chronic)	Type B dissection	AneuRx, Talent, Gore Excluder	69 (43–86)	24	3	Retrograde type A dissection ×2 and ×1 ruptured endoleak	Retrograde type A dissec- tion ×3 Type I endoleaks ×4 Aneurysmal dilatation ×3 Recurrent dissection ×1 Medical (cardiac and pul- monary) ×11 Renal failure ×2 Stroke ×1 Paraplegia ×0
Duebener et al ⁵⁹	2004/ Germany	2001–2003/ retrospective	Acute type B dissection	Rupture, hemo- thorax, visceral malperfusion, and refractory pain	Talent	59.2 (46–65)	10	2	Intimal tear ×1 Hemorrhagic shock (malper- fusion) ×1	×3 open surgical (retro- grade type A dissection, acute stent dislocation by fractured wires, and sec- ondary leakage and late an- eurysm formation)
Grabenwoger et al ⁶⁰	2003/Austria	1997–2002/ retrospective	Complicated type B dissection	Pain Uncontrolled hypertension	Talent, Gore Excluder	NS	11	0	–	Paraparesis ×1 Overstented left subclavian artery ×1

(Continued)

Table 1 (Continued)

Author	Year published/ country	Duration/type	Clinical inclusion criteria	Indications	Device	Age	N	30-d mortality	Cause for mortality	Morbidity
Lonn et al ⁶¹	2003/Sweden	1999–2002/ retrospective	Type B dissection (14 acute, 4 chronic)	Type B dissection	NS	NS	18	3	Medical (cardiac ×3)	Paraplegia ×1 Stroke ×4
Lambrechts et al ⁶²	2003/ Belgium	2000–2002/ retrospective	Type B dissection	Type B dissection	Talent, Gore Excluder, AneuRx	NS	11	0	–	Partial thrombosis ×1
Bell et al ⁶³	2003/London	1997–2002/ retrospective	Thoracic endoluminal repairs	Type B dissection	NS	NS	5	0	–	Paraplegia ×1 Stroke ×1 Type I endoleak ×2
Orend et al ⁶⁴	2003/ Germany	1995–2001/ retrospective	Aneurysms, dissection, posttraumatic	Type B dissection	Talent, Gore Excluder	NS	14	1	Retrograde type A dissection. Four other deaths recorded after 30 d	Type I endoleak ×4 Conversion to open ×4 Retrograde type A dissection ×2 False lumen rupture ×1 Overstenting ×2 TIA ×1
Weber et al ⁶⁵	2003/ Germany	2002/case report	Chronic type B dissection	Peripheral malperfusion	NS	71	1	0	–	0
Matravers et al ⁶⁶	2003/London	1999–2002/ retrospective	Chronic type B dissection	Chronic type B dissection	NS	NS	9	1	–	Common iliac artery rupture ×1 Secondary intervention ×2 Stroke ×0
Shim et al ⁶⁷	2002/Korea	1998–2001/ retrospective	Type B dissection	Persistent back or chest pain, progressive enlargement of false lumen	NS	55.9 ± 13.7 (32–82)	15	1	Medical cause	Procedural failure ×1 Device migration ×1 Conversion to open ×2 Retrograde type A dissection ×1
Herold et al ⁶⁸	2002/ Germany	1999–2001/ retrospective	Acute (6) and chronic (12) type B dissection	Type B dissection	Talent	68.6 ± 7 (58–84)	18	1	MI (day 6)	Reintervention ×3
Kato et al ⁶⁹	2002/Japan	1997–2002/ retrospective	Acute (14) and chronic (14) type B dissection	Type B dissection	NS	NS	28	2	Visceral malperfusion ×1 Hemothorax ×1	Type I endoleak ×1 Aneurysmal dilatation ×1 Late rupture ×1 Respiratory failure ×1
Shimono et al ⁷⁰	2002/Japan	1999–2001/ retrospective	Acute type B dissection	Type B dissection	NS	NS	37	1	–	Failed ×1
Totaro et al ⁷¹	2002/Italy	1999–2001/ retrospective	Type B dissection	Type B dissection	NS	NS	25	0	–	Retrograde type A dissection ×1
Palma et al ⁷²	2002/Brazil	1998–2001/ retrospective	Type B dissection	Persistent pain, mediastinal hematoma, hemothorax	NS	NS	58	2	Medical ×2	Type I endoleak ×11 Conversion to open ×5

Table 1 (Continued)

Author	Year published/ country	Duration/type	Clinical inclusion criteria	Indications	Device	Age	N	30-d mortality	Cause for mortality	Morbidity
Criado et al ⁷³	2002/United States	1998–2002/ retrospective	Type B dissection	Type B dissection	NS	NS	16	1	Iliac rupture –1	Medical ×8 Partial thrombosis ×4 Type I endoleak ×1
Bortone et al ⁷⁴	2001/Italy	1999–2000/ retrospective	Type B dissection	Type B dissection	NS	NS	5	0	–	Redissection with visceral/leg ischemia ×1
Sailer et al ⁷⁵	2001/Austria	1997–2000/ retrospective	Type B dissection	Type B dissection	NS	NS	7	0	–	Reintervention ×1
Won et al ⁷⁶	2001/Korea	1994–1999/ retrospective	Type B dissection	Type B dissection	NS	NS	12	0	–	Failure ×2
Yamazaki et al ⁷⁷	2000/Japan	1999/case report	Type B dissection	Type B dissection	NS	56	1	0	–	0
Shimono et al ⁷⁸	2000/Japan	1998–2000/ retrospective	Acute (8), subacute (4), and chronic (6) type B dissection	Type B dissection	NS	NS	18	0	–	Emboli ×4
Czernak et al ⁷⁹	2000/Austria	1996–2000/ retrospective	Acute (5) and chronic (2) type B dissection	Type B dissection	Talent	67 (43–80)	7	0	–	Retrograde type A dissection ×1 LSCA overstented ×3 Ruptured CFA ×1 Dissection of CFA ×1
Beregi et al ⁸⁰	2000/France	1997–1999/ retrospective	Type B dissection	Type B dissection	NS	NS	12	4	–	Renal failure ×2
Nienaber et al ⁸¹	1999/Germany	1997–2000/ retrospective	Type B dissection	Type B dissection	NS	62 ± 11	12	0	–	Partial thrombosis of false lumen ×3
Dake and Kato ⁸²	1999/United States	1996–1998/ retrospective	Type B dissection	Type B dissection	NS	NS	15	2	Acute rupture of false lumen ×2	Partial thrombosis ×4
da Fonseca et al ⁸³	1998/United States	1997/ retrospective	Type B dissection	Type B dissection	NS	NS	4	0	–	0
Total							1,574		Overall mortality = 8.07%	

Abbreviations: ARF, acute renal failure; DVT, deep venous thrombosis; FL, false lumen; LCCA, left common carotid artery; LSCA, left subclavian artery; MI, myocardial infarction; NS, not stated; PE, pulmonary embolism; S-B-AD, symptomatic Stanford type B aortic dissection; TEVAR, thoracic endovascular aortic repair.

Table 2 Mortality associated with TEVAR in < 30 days^a (see ► **Table 1**)

Complication	44 studies (1998–2007) (n = 787 patients)	25 studies (2007–2013) (n = 787 patients)	Total (%) (n = 121/127)
Retrograde type A dissection	8	9	17 (14%)
Persistent visceral and lower limb malperfusion	3	17	20 (16.5%)
Rupture of false lumen or type I endoleak or aorto-esophageal fistula	17	30	47 (38.8%)
Aorto-esophageal fistula	0	2	2 (1.7%)
Further type B dissection	0	2	2
Ruptured iliac or access vessel	1	1	2
Open conversion	0	2	2
Device-related multiorgan failure	0	5	5 (4.1%)
Total stent-related mortality (medical causes excluded) (n = 121)	29/121 (24.0%)	68/121 (56.2%)	121 (95%) clearly documented out of 127 mortalities
Medical cause (cardiac or pulmonary, renal failure or stroke)	8	16	24 (12%)

Abbreviation: TEVAR, thoracic endovascular aortic repair.

^aRates calculated from available information of 121 out of 127 deaths.

($p < 0.05$ using Pearson chi-square test with a linear-by-linear association of $p = 0.020$). This was contributed mainly by three patient groups: (1) the persistent visceral malperfusion/lower limb malperfusion group, (2) the ruptured false lumen/type I endoleak/aorto-esophageal fistula group, and (3) the all-cause medical-related group (► **Table 2**).

The morbidity rates revealed a similar pattern showing 36.6 versus 25.0% in the 2007 to 2013 and 1998 to 2007 groups, respectively, but did not reach statistical significance ($p > 0.05$). The main contributors to this included retrograde type A dissections, visceral malperfusion, cerebrovascular accidents, partial thrombosis of the false lumen, secondary interventions, and renal failure (► **Table 3**).

There was no major statistical difference with regard to age or stent-graft used.

Discussion

Background

Acute aortic syndrome describes the acute presentation of patients with characteristic aortic pain caused by potentially life-threatening thoracic aortic pathology. These include aortic dissection, intramural hematoma, penetrating aortic ulcer, aneurysmal leakage or rupture, and traumatic transection.¹² Aortic dissection can lead to aneurysm formation and its associated complications. The etiology is multifactorial with causes related to connective tissue or inherited disorders, developmental disorders associated with pregnancy, syphilis, crack cocaine usage, and cardiac catheterization.

Traditional management consisted of hypotensive medical management with open surgical intervention reserved for complicated cases with intractable pain, uncontrolled hyper-

tension, progression of dissection, rupture, dilatation, lower limb ischemia, or visceral hypoperfusion.¹³ Stent-graft technology has caused a paradigm shift in the approach for symptomatic cases. This is not without major risk which includes stroke, transient ischemic attacks, paraplegia, paraparesis, distal embolization, migration, endoleak, progressive dilatation, and inadvertent covering of arch or visceral vessels.¹⁴

Literature Review

De Baek et al¹³ described their experience Between 1953 and 1993 with a total of 659 patients undergoing descending thoracic aneurysm resection. The most common cause was atherosclerosis and pain was the main presenting symptom. Perioperative mortality fell from 24.2% between 1953 and 1964 to 14.3% between 1970 and 1993 using conventional open techniques. Paraplegia occurred in 4.1%, and paraparesis occurred in 5.9%. The major source of perioperative morbidity and mortality was cardiac causes (48%) followed by perioperative hemorrhage (14.4%), pulmonary complications (14.4%), and rupture of another aneurysmal segment (12.0%).¹³

TEVAR has now become the standard of care for symptomatic disease in the past 10 years. The largest articles included that of Eggebrecht et al from Germany in 2005 in the *European Heart Journal*³ with an analysis of 609 cases collected from 39 studies from 1999 to 2004. There has also been another article published by Walsh et al⁴ from Cambridge, United Kingdom, in the *Journal of Vascular Surgery*, 2008, with 17 eligible series totaling 1,109 patients between 1991 and 1997. This particular article did not specifically address Stanford B dissection and included data with respect to aneurysm formation and traumatic rupture.

Table 3 Morbidity associated with TEVAR

Complication	44 studies (1998–2007) (n = 787 patients)	25 studies (2007–2013) (n = 787 patients)	n = 1,574 (%)
Retrograde type A dissection	21	28	49 (3.1%)
Visceral malperfusion	5 (including 1 lower limb)	27 (4 lower limbs with 1 amputation)	32 (2.0)
Aortoesophageal fistula	0	4 (1 esophageal necrosis)	4 (< 0.1)
Cerebrovascular accident	11 (including 1 TIA)	30	41 (2.7)
Type I endoleak	39	43	82 (5.2)
Type II endoleak	6	5	11 (< 0.1)
Paraplegia/paraparesis	6	24 (3 hemiplegia)	30 (1.9)
Partial thrombosis of false lumen	16	23	39 (2.5)
Conversion to open	16	14	30 (1.9)
Secondary interventions	25	40	65 (4.1)
Device migration/modular disconnection/junctional leaks	5	2	7 (< 0.1)
Emboli	4	0	4 (< 0.1)
Medical complications (cardiac, pulmonary, multiorgan failure)	29	25	54 (3.4)
Renal failure	8	23	31 (2.0)
Overstenting celiac artery	1	0	1 (< 0.1)
Postimplantation syndrome	3	0	3 (< 0.1)
Failure	2	0	2 (< 0.1)
Total complication rate	197 (25.0%)	288 (36.6%)	485 (30.8%)

Abbreviation: TEVAR, thoracic endovascular aortic repair.

Recently, the first randomized control trial was published by Nienaber et al⁷ in December 2009 and most of the studies have been retrospective observational studies. Nienaber et al randomized 72 patients to elective stent-graft repair with medical management and 68 patients to medical therapy alone with surveillance with the primary end point being all-cause death at 2 years in uncomplicated type B aortic dissection. TEVAR failed to improve the 2-year survival and adverse event rates despite favorable aortic remodeling.^{7,8}

The NICE (National Institute for Clinical Excellence) guideline was based on a systematic review of published evidence commissioned by the institute with a total of 29 studies (27 case series and 2 comparative observational studies). In one comparative study, the technical success was 100% (67 patients) and overall technical success rate was 93% across 18 studies (16 case series and 2 comparative studies). The conversion to open rate varied from 0% (26 cases) to 7% (1 of 14 cases) and the number who developed aneurysmal dilatation varied from 0% (18 cases) to 7% (2 of 29 cases). The largest study of 84 patients showed an aneurysmal dilatation rate of 5% (4 of 84). The number of patients who experienced a

decrease in aneurysm size varied from 100% in one study to 17% in another. Of relevance to this present article, the 30-day mortality by NICE varied from 0% in several studies with a combined population of 94 patients to 14% (2 of 14 patients). The overall mortality ranged from 3 to 24% across 17 studies with a mean follow-up of 14 months. The most common complication was endoleak with a mean incidence of 13% over 12 months (752 patients). In five studies, there were no endoleaks reported in 83 patients. In terms of major complications, injuries to access artery occurred in nine case series and included iliac artery dissection in one, perforation of the iliac artery in one, and rupture of the femoral artery in two cases. There were six cases of stent-graft migration in 15 case series. Other complications included wound problems in 25%, stroke in 19%, renal failure requiring dialysis in 11%, and paraplegia in 7%.¹⁴

The article of Eggebrecht et al showed procedural success in 98% and major complications in 11% including a stroke rate of 0.8% and paraplegia of 1.9%. Overall complications were higher in acute dissections compared with chronic. Overall survival rates were 90% at 6 months and 88% at 2 years.³

A recent study by Thrumurthy et al¹⁵ in 2010 (at St. George's Vascular Institute, London) showed the role of TEVAR in chronic type B aortic dissection with reference to its midterm success. Analysis on 17 studies of 567 patients was conducted showing technical success in 89.9% and a midterm mortality of 9.2% (46/499) with a median follow-up of 24 months. Morbidity included an 8.1% endoleak rate (type I) and reintervention rates ranging from 0 to 60% with a median follow-up of 31 months. Of 332 patients, 26 (7.8%) developed aneurysms of the distal aorta or continued false lumen perfusion with aneurysmal dilatation. Rare complications included delayed retrograde type A dissection (0.67%), aortooesophageal fistula (0.22%), and neurological complications (paraplegia 2/447, 0.45%; stroke 7/475, 1.5%). It was concluded that the absolute benefit of TEVAR over alternative treatments for chronic type B aortic dissection remains uncertain with a lack of natural history data for medically treated cases; significant heterogeneity in case selection and the absence of consensus reporting standards for interventions are significant obstructions.¹⁵

In 2010, Nienaber et al critiqued the INSTEAD trial and concluded that there was no difference in all-cause mortality at 2 years, with cumulative survival of 95% with optimal medical therapy versus 88% with TEVAR ($p = 0.15$). Aorta-related mortality was also not different ($p = 0.44$) within the first 2 years of follow-up, and risk for the combined endpoints of aorta-related death (rupture) and progression (including conversion or additional endovascular or open surgery) was similar ($p = 0.65$). They concluded that the data in the INSTEAD trial show that in the setting of clinically stable, so-called uncomplicated type B aortic dissection, elective stent-graft placement on top of optimized medical management fails to improve survival and adverse events within an observation period of 2 years, despite favorable aortic remodeling.¹⁶

Fattori et al¹⁷ in the team's consensus document examined 63 studies published from 2006 to 2012. There were 1,548 patients treated medically, 1,706 with open surgery, and 3,457 underwent TEVAR. The pooled early mortality rates (first 2 weeks) were 6.4% with medical treatment, 10.2% with TEVAR, and 17.5% with open surgery, mostly for complicated cases. Limited data for treatment of subacute (2–6 weeks after onset) type B aortic dissection showed an early mortality rate of 2.8% with TEVAR. In chronic (after 6 weeks) type B aortic dissection, 5-year survival of 60 to 80% was expected with medical therapy. If interventional treatment was applied, the pooled early mortality rate was 6.6% with TEVAR and 8.0% with open surgery.¹⁷

It seems that overall mortality and morbidity rates have been increased for TEVAR intervention for S-B-AD. This was demonstrated in the study herein presented, whereby the overall mortality and morbidity rates for TEVAR were 8.07% ($n = 127$) and 30.8% ($n = 485$), respectively. Additionally, the stent-graft-related mortality rate (which includes the subset of patients who died; $n = 127$) increased in the 2007 to 2013 group (787 patients) compared with the 1998 to 2007 group (787 patients) (56.2 vs. 24%; $p < 0.05$) as well as the overall morbidity rates from 25% (1998–2007) to 36.6% (2007–2013).

With respect to type I endoleak rates, this study showed a marginal increase in the leak rate from 39 in the 1998 to 2007 group to 43 in the 2007 to 2013 group with overall type I endoleak rates averaging 5.2%. Type II endoleaks also showed few numbers with an overall leak rate of $< 0.1\%$.

Retrograde aortic dissection accounted for 3.1% (49) of the study population with only a marginal increase from 21 to 28 in the 1998 to 2007 period and 2007 to 2013 periods, respectively. This is in keeping with most of the other studies with rates in a similar range.

Visceral malperfusion was, however, on a significant increase as seen from 5 in the 1998 to 2007 period compared with 27 cases in the 2007 to 2013 period.

This may be explained by a more aggressive clinical approach to patient selection for S-B-AD and quite possibly better reporting leading to bias from previous inadequate data collection in older studies. This has occurred in spite of overall improvement in stent-graft technology and techniques. In conclusion, the data available are still too heterogeneous to make solid conclusions and the only way forward is through better data registries and well-designed clinical trials.

Conflict of Interest

No conflicts of interest declared.

Declaration

This article is based on a thesis for the Masters of Science in Surgical Practice done in 2009 at the University of Kent at Canterbury, England.

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