Systematic Review

Thoracic endovascular repair of chronic type B aortic dissection: a systematic review

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Background: At present, the optimal management strategy for chronic type B aortic dissection (CTBAD) remains unknown, as equipoise remains regarding medical management versus endovascular treatment versus open surgery. However, the results over recent years of thoracic endovascular aortic repair (TEVAR) in CTBAD appear promising. The aim of this systematic review was to provide a comprehensive analysis of the available data reporting outcomes and survival rates for TEVAR in CTBAD.

Methods: Electronic searches of six databases were performed from inception to April 2021. All studies reporting outcomes, specifically 30-day mortality rates, for endovascular repair of CTBAD were identified. Relevant data were extracted, and a random-effects meta-analysis of proportions or means was performed to aggregate the data. Survival data were pooled using data derived from original Kaplan-Meier curves, which allows reconstruction of individual patient data.

Results: Forty-eight studies with 2,641 patients were identified. Early (<30 days) all-cause and aortic-related mortality rates were low at 1.6% and 0.5%, respectively. Incidence of retrograde type A dissection in the post-operative period was only 1.4%. There were also low rates of cerebrovascular accidents and spinal cord injury (1.1% and 0.9%, respectively). Late follow-up all-cause mortality was 8.0%, however, late aortic-related mortality was only 2.4%. Reintervention rates were 10.1% for endovascular and 6.7% for surgical reintervention. Pooled rates of overall survival at 1-, 3-, 5- and 10-year were 91.5%, 84.7%, 77.7% and 56.3%, respectively.

Conclusions: The significant heterogeneity in the available evidence and absence of consensus reporting standards are important considerations and concern when interpreting the data. Evaluation of the evidence suggests that TEVAR for CTBAD is a safe procedure with low rates of complications. However, the optimal treatment strategy for CTBAD remains debatable and requires further research. Evidence from high-quality registries and clinical trials are required to address these challenges.

Keywords: Chronic type B aortic dissection (CTBAD); thoracic endovascular aortic repair (TEVAR); descending thoracic aorta

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Introduction

Aortic disease, and more specifically aortic dissection, comprises a significant disease burden, occurring twice as often in males compared with females and frequently occurring in patients aged between 50–70 years (1,2). With an incidence estimated at 5–30 per million per year, 20% of patients with aortic dissection die before reaching hospital, with a further 30% of those who do reach tertiary centres dying during their hospital admission (1,3,4). In light of this, there has been increased interest in the management of aortic dissections, particularly with the shift towards endovascular management as endovascular graft technology continues to evolve.

Disease processes, such as atherosclerosis, chronic hypertension, or the presence of genetic conditions (such as vascular Ehlers-Danlos or Marfan syndromes) that alter the integrity of the elastic or muscular components of the aortic wall, predispose patients to the development of aortic dissection (4). Whilst this pathology can be classified using multiple systems, one of the most common classifications is the Stanford classification, which describes dissections as either Type A, with the entry tear proximal to the ostium of the left subclavian artery, or Type B, with the entry tear distal to the ostium of the left subclavian artery (4). Whilst most Type A aortic dissections are surgical emergencies and require open replacement of the ascending aorta with or without aortic root or arch replacement (5), uncomplicated Type B aortic dissections have classically been managed medically with strict blood pressure and heart rate control to reduce pulse pressure, statins and lifestyle modification (6,7). Surgical management has classically been reserved for complicated Type B dissections (those associated with rupture or malperfusion syndromes), via open surgical repair or thoracic endovascular aortic repair (TEVAR) (8-10). However, it is estimated that 25–50% of patients with acute Type B aortic dissections who are managed medically will undergo aneurysmal degeneration of the dissected segment and require surgical repair, either via open or endovascular methods, during the chronic phase of their disease process (3,10).

There is ongoing debate within the literature surrounding the management of chronic type B aortic dissection (CTBAD) and whether optimal medical therapy or the use of TEVAR is most effective in the management of this pathology (9-11). Historically, the definition of chronicity in type B aortic dissection has been after 14 days have elapsed from symptom onset. This classification is based on the high rate of mortality (up to 70%) in aortic dissection within the first 2 weeks of onset (12). In recent years, a third "sub-acute" category (between 2 weeks and 3 months) has been proposed in the classification system, as the risk of death remains high in the first 3 months (13). CTBADs are associated with a risk of rupture, with recent guidelines suggesting that this risk increases with aneurysmal dilatation (2,8,10). Rates of aortic rupture have been quoted as high as 28.6% in aortic diameters of up to 6.4 cm (8). As such, aneurysmal dilatation and rapid growth of aneurysms are one of the most frequent indications for the treatment of CTBAD (2,3). Open surgical repair has several significant disadvantages compared to endografting, including the necessity for a posterolateral thoracotomy or thoracoabdominal incision depending on the extent of the required repair, single lung ventilation, full or partial cardiopulmonary bypass, and possible circulatory arrest and hypothermia (4). Subsequently, the use of TEVAR in CTBAD has had an increased focus over the past decade.

This systematic review sought to provide a comprehensive analysis of the currently available literature to determine early outcomes, reintervention rates and mid- or long-term survival rates for endovascular repair of CTBAD.

Methods

Literature search strategy

Six electronic databases were used to perform the literature search including Ovid MEDLINE, EMBASE, Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews (CDSR), SCOPUS, and Database of Abstracts of Review of Effectiveness (DARE). These databases were searched from date of inception to 26th April 2021. The search strategy included a combination of keywords and Medical Subject Headings (MeSH) including "Aorta" AND "Dissection" AND "Chronic" AND "Endovascular Procedures" OR "Endovascular repair" OR "TEVAR". Predefined selection criteria were used to assess all relevant articles that were identified. Our methods adhered to the guidelines set forth in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement (14). Two reviewers (MLW and MDB) independently screened the title and abstract of all identified records in the search. Where the title/abstract provided insufficient detail to determine study relevance, a full-text copy of the article was retrieved for review. The reference lists of selected studies were reviewed manually

to identify any extra relevant studies not identified in the electronic search.

Selection criteria

Studies were eligible for inclusion in this systematic review if they included a patient population who underwent TEVAR for Chronic Stanford type B/DeBakey type III aortic dissection. Chronicity of dissection for this systematic review was defined as greater than 2 weeks following symptomatic presentation or documentation of an intimal entry tear. Studies that described mixed populations of acute and chronic dissections without separate patient or outcome data were deemed ineligible for inclusion. Studies had to report the primary outcome of interest (see below) and have a minimum CTBAD cohort greater than ten patients to be eligible for inclusion. Residual CTBAD post previous Type A and open ascending aorta surgical repair were included due to a number of studies reporting mixed populations with no separate data/outcomes for these cohorts. Cases that involved the abdominal aorta only were excluded. Hybrid (open and endovascular) procedures or branched and/or fenestrated endovascular repair for CTBAD were excluded. When trials/registries/institutions published duplicate studies with extended length of follow-up or larger study populations, the most updated and complete study was included. Included studies were limited to those in English and only involving human subjects. Abstracts, case reports, conference presentations, editorials and reviews were excluded.

Outcomes of interest

The primary outcome of interest was in-hospital/30-day mortality. Secondary outcomes of interest included mid- to long-term survival rates and other post-operative outcomes, including aortic related mortality, rupture, retrograde type A dissection, cerebrovascular accident (CVA) and spinal cord injury (SCI).

Data extraction and critical appraisal

Two independent reviewers (BH and BW) extracted data directly from publication texts, tables and figures. A third reviewer (MLW) independently reviewed and confirmed all extracted data. Differences of opinions between the two main reviewers (BH and BW) were resolved through means of discussion and consensus, including primary investigator (MLW) where necessary. Attempts were made to clarify insufficient or indistinct data from corresponding authors of included studies where required. Outcome data was extracted in a manner in which each study was effectively treated as a case series regardless of actual study design, and therefore a critical appraisal of the quality of each individual included study was not performed.

Statistical analysis

A meta-analysis of proportions or means were performed for categorical and continuous variables, as appropriate. A random effects model was used to account for differing center/surgeon experience, different endoprostheses used, and different operative and management protocols across the included studies. To facilitate this statistical pooling, means and standard deviations were calculated from the median (with range or interquartile range), where reported, using the methods described by Wan and colleagues (15). Pooled data are presented as n (%) with 95% confidence intervals (CI). For outcome data, heterogeneity amongst studies was assessed using the I^2 statistic. Thresholds for I^2 values were considered as low, moderate and high heterogeneity as 0–49%, 50–74% and ≥75%, respectively (16). Meta-analysis of proportions or means were performed using Stata (version 17.0, StataCorp, Texas, USA).

Survival data was calculated from aggregation of Kaplan-Meier survival data from included studies, where reported, using the methods described by Guyot and colleagues (17). Aggregation of this data was performed by reconstructing individual patient data from digitized Kaplan-Meier survival curves and patient number-at-risk data. The reconstructed individual patient data were then pooled and used to generate an aggregated survival curve. Digitization of source Kaplan Meier curves was performed using DigitizeIt (version 2.5.9, Braunschweig, Germany) and survival analysis was performed using R (version 3.5.2, R Foundation for Statistical Computing, Vienna, Austria).

Results

The literature search identified a total 915 studies (*Figure 1*). An additional nine articles were identified on manual searches of reference lists. After exclusion of duplicates or irrelevant studies, 97 articles were deemed appropriate to undergo full-text review. Forty-eight studies with a total

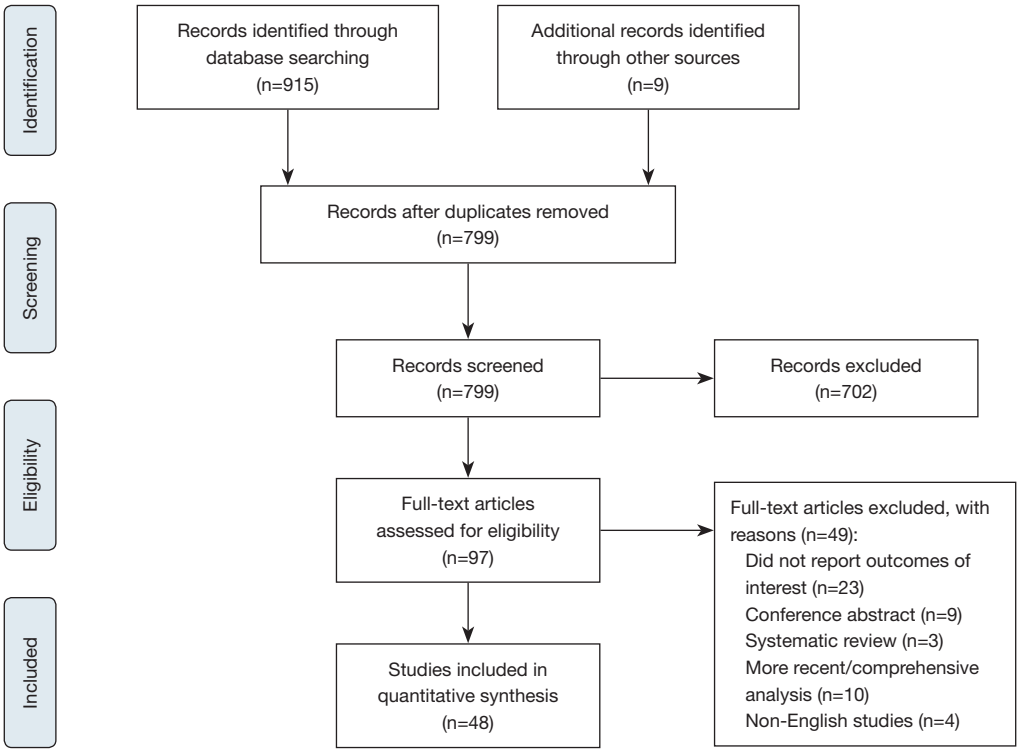

Figure 1 PRISMA flow-chart summarizing the search strategy for relevant publications.

of 2,641 patients undergoing TEVAR for CTBAD were deemed suitable to be included for quantitative analysis. The remaining 49 articles were deemed unsuitable, predominantly for lacking adequate reporting of outcomes of interest. Two articles were excluded for reporting outcomes for branched/fenestrated endovascular repair for CTBAD.

Of the 48 included studies, nine were prospective and the remaining 39 were retrospective cohort studies (*Table 1*). Included studies had varying cohort sizes from 10 to 208 patients. Definition of chronicity varied between the included studies, however, the majority using greater than 2 weeks after the onset of symptoms or diagnosis to define a CTBAD. The majority of included studies reported outcomes for CTBAD DeBakey type III (type IIIa, IIIb or both), while 13 studies reported outcomes for mixed cohorts of residual type I and III CTBAD patients. The indication for intervention for CTBAD varied amongst the included studies (Table S1). The majority of the included studies included mixed cohorts of both complicated and uncomplicated CTBAD. The weighted mean follow-up period of all included studies was 33.8 months.

Baseline characteristics

Overall, the weighted pooled age of all patients was 60.5 years. The entire patient population was comprised of 76.7% males. The majority of patients had a history of hypertension (89.4%). Only a fraction of the included patients had a history of diabetes (10.4%), prior cerebrovascular accident (CVA) (5.6%) or renal insufficiency (11.3%). Pooled interval between diagnosis and endovascular repair of CTBAD was 20.6 months (95% CI, 14.5–25.8). Approximately one third of the patient population had undergone prior cardiac or open aortic surgery (29.2%). Other patient baseline characteristics are seen in Table S2. History of peripheral vascular disease, congestive heart failure and other comorbidities were poorly reported across the included studies.

A number of different endoprostheses were used across the included studies (Table S3). These included the Zenith TX1 and TX2 (Cook Medical Incorporated, Bloomington, Indiana, USA), Talent and Valiant Captiva (Medtronic Incorporated, Santa Rosa, California, USA), TAG and cTAG (W.L Gore & Associates, Newark, Delaware, USA) and Relay endoprostheses (Terumo Aortic, Tokyo, Japan).

Table 1 Study characteristics

Primary author	Year	Institution(s)	Study period	Type of study	Definition of chronic	n	Mean follow up time (months)	Extent of aortic pathology (DeBakey)	Complicated/ uncomplicated dissection
Kato (18)	2002	Mie University Hospital, Mie, Japan	1997–2002	RC	>1 month after symptoms	14	27.0±12.0	Residual type I and type III	Both
Greenberg (19)	2005	The Cleveland Clinic Foundation, Ohio, USA	2001–2004	PC	NR	15	14.0	NR	NR
Baumgart (20)	2006	West German Heart Center, University Duisburg-Essen, Essen, Germany	1999–2004	PC	>2 weeks after symptoms	35	21.0±18.0	NR	NR
Böckler (21)	2006	German Cancer Research Center, Heidelberg, Germany	1997–2004	PC	>2 weeks after symptoms	15	24.0±14*	Type III	Both
Song (22)	2006	Harbor-UCLA Medical Center, Harbor, California, USA	1999–2005	RC	>2 weeks after symptoms	17	11.0±16.4*	NR	Complicated
Thompson (23)	2007	7 European Centers	2005–2006	RC	NR	52	5.3±3.5	Type III	Both
Jing (24)	2008	Shenyang Northern Hospital, Liaoning, China	2002–2007	RC	>2 weeks after symptoms	35	17±14	Type III	Both
Marcheix (25)	2008	7 European Centers	1996–2004	RC	NR	15	25.2±16.8	Residual type I and type III	Both
Sayer (26)	2008	St George's Vascular Institute, London, UK	2000–2007	RC	>2 weeks after symptoms	40	30.0	NR	NR
Alves (27)	2009	Federal University of Sao Paulo and Hospital do Coracao Da Associacao, Sao Paulo, Brazil	1997–2004	RC	>2 weeks after symptoms	61	35.9±28.5	Type III	Both
Guangqi (28)	2009	The First Affiliated Hospital of Sun Yat-sen University, Guangzhou, China	2001–2006	RC	>2 weeks after symptoms	49	22.1±20.8	Type III	Both
Kim (29)	2009	Yonsei University College of Medicine, Seoul, Republic of Korea	1994–2007	RC	>2 weeks after symptoms	72	64.4±38.8	Type III	Uncomplicated
Manning (30)	2009	Malmo University Hospital, Malmo, Sweden	2000–2006	RC	>2 weeks after symptoms	10	56.0	Type IIIb	Uncomplicated
Czerny (31)	2010	3 European Centers (Switzerland and Austria)	2004–2009	RC	NR	14	34.5±14.5*	Residual type I and type III	Uncomplicated
Xu (32)	2010	Capital Medical University, Beijing Anzhen Hospital, Beijing, China	2001–2007	RC	>1 month after symptoms	84	33.2±19.2	Type III	Uncomplicated
Kang (33)	2011	Cleveland Clinic Foundation, Cleveland, Ohio, USA	2000–2007	RC	>2 weeks after symptoms	76	33.5±29.4	Type III	Complicated

Table 1 (*continued*)

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Primary author	Year	Institution(s)	Study period	Type of study	Definition of chronic	n	Mean follow up time (months)	Extent of aortic pathology (DeBakey)	Complicated/ uncomplicated dissection
Oberhuber (34)	2011	University of Ulm, Ulm, Germany	1990–2011	RC	>2 weeks after symptoms	19	37.8±92.9*	Type III	Uncomplicated
Parsa (35)	2011	Duke University Medical Center, Durham, North Carolina, USA	2005–2009	RC	>2 weeks after symptoms	51	27.0±16.5	Residual type I and type III	Uncomplicated
Andacheh (36)	2012	Harbor-UCLA Medical Center, Torrance, California, USA	2002–2010	PC	>2 weeks after symptoms	73	18.0	Type III	Complicated
Mani (37)	2012	Guy's and St Thomas NHS Foundation Trust, London, UK	2000–2010	RC	>2 weeks after symptoms	58	38.0±28.0	Residual type I and type III	Both
Nathan (38)	2012	Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania, USA	2005–2010	RC	>6 weeks after symptoms	27	27.3±22.1	Residual type I and type III	Both
Qing (39)	2012	The University of Hong Kong, Queen Mary Hospital, Hong Kong	NR	PC	>4 weeks after dissection	32	31.7±17	Type III	Uncomplicated
Yang (40)	2012	Taipei City Hospital, Taipei, Taiwan	2006–2011	RC	>2 weeks after symptoms	28	24.1±15.6	Type III	Both
Chen (41)	2013	Nanjing First Hospital, Nanjing, China	2000–2011	PC	>2 weeks after symptoms	56	37.6±28.4	Type III	Complicated
Jia (42)	2013	Chinese PLA General Hospital, Beijing, China	2007–2010	PC	>2 weeks after symptoms	208	16.3±9.5*	Type III	Uncomplicated
Lee (43)	2013	Severance Hospital, Yonsei University College of Medicine, Seoul, Republic of Korea	1997–2010	RC	>2 weeks after symptoms	71	53.2±52.9	Type III	Complicated
Leshnower (44)	2013	Emory University School of Medicine, Atlanta, Georgia, USA	2005–2011	RC	>8 weeks after acute event	31	21.0±20.0	Residual type I and type III	Uncomplicated
Nozdrzykowski (45)	2013	Heart Center Leipzig, University of Leipzig, Leipzig, Germany	2000–2010	RC	>2 weeks after symptoms	32	52.2±31.2	Residual type I and type III	Both
Patterson (46)	2013	MOTHER Database (Combined data from 5 prospective trials)	2004–2012	PC	>2 weeks after symptoms	195	28.8	NR	NR
Scali (47)	2013	University of Florida College of Medicine, Gainesville	2004–2011	RC	NR	80	31.8±12.2*	Residual type I and type III	Uncomplicated
Andersen (48)	2014	Duke University Medical Center, Durham, North Carolina, USA	2005–2013	RC	>2 weeks after symptoms	44	44.3±14.3*	Residual type I and type III	NR
Kitamura (49)	2014	Kitasato University School of Medicine, Kanagawa, Japan	1998–2012	RC	>2 weeks after symptoms	45	90.0±36.8	Type III	Uncomplicated

Table 1 (*continued*)

Table 1 (*continued*)

Primary author	Year	Institution(s)	Study period	Type of study	Definition of chronic	n	Mean follow up time (months)	Extent of aortic pathology (DeBakey)	Complicated/ uncomplicated dissection
Lombardi (50)	2014	STABLE Trial Investigators (multicenter)	2007–2012	PC	>2 weeks after symptoms	31	24.0	Type IIIb	Complicated
Song (51)	2014	Gangnam Severance Hospital, Yonsei University College of Medicine, Seoul, Republic of Korea	2012–2013	RC	>3 months after diagnosis	20	10.3±4.9	Type IIIb	Complicated
Nathan (52)	2015	University of Washington, Seattle, Washington, USA	2006–2013	RC	>2 weeks after symptoms	47	35.1±20.9	Residual type I and type III	Both
van Bogerijen (53)	2015	University of Michigan Hospitals, Ann Arbor, Michigan, USA	1993–2013	RC	>8 weeks after diagnosis	32	41.5±54.4*	Type IIIb	Uncomplicated
Zhang (54)	2017	General Hospital of People's Liberation Army, Beijing, China	2011–2013	RC	>2 weeks after symptoms	25	26.4±15.6	Type III	Complicated
Chou (55)	2018	National Taiwan University Hospital, Taipei, Taiwan	2008–2014	RC	>2 weeks after symptoms	23	27.5±19.1	Type III	Complicated
Huang (56)	2018	Taipei Veterans General Hospital, Taipei, Taiwan	2006–2013	RC	>2 weeks after symptoms	65	36.0	Type IIIb	Uncomplicated
Tjaden (57)	2018	Gore Global Registry (worldwide multicenter registry)	2010–2016	RC	>3 months after symptoms	94	26.0	Residual type I and type III	Both
Kim (58)	2019	Yonsei University College of Medicine, Seoul, Republic of Korea	2012–2017	RC	>3 months after symptoms	75	37.2±17.7*	Type IIIb	NR
Wang (59)	2019	VQI TEVAR Registry	2013–2016	RC	>30 days after symptoms	193	30.0	NR	Both
Zha (60)	2019	The First Affiliated Hospital of Anhui Medical University, Anhui, Chine	2012–2016	RC	>2 weeks after symptoms	23	39.5±10.5*	Type III	Both
Conway (61)	2020	Lenox Hill Hospital, Northwell Health, New York, USA	2010–2016	RC	>30 days after symptoms	208	49.2	Type III	Both
Li (62)	2020	First Affiliated Hospital, Zhejiang University, Zhejiang, China	2009–2013	RC	>3 months after symptoms	34	68.1±22.9	Type III	Both
Oishi (63)	2020	Kyushu University Hospital, Higashi, Fukuoka, Japan	2009–2019	RC	>3 months after symptoms	40	39.2±27.1	Residual type I and type III	Uncomplicated
Puech-Leao (64)	2020	Hospital das Clinicas, University of Sao Paulo, Faculty of Medicine, Sao Paulo, Brazil	2004–2017	RC	>2 weeks after symptoms	42	57.2	NR	Uncomplicated
Ueki (65)	2021	Shizuoka General Hospital, Kita-Ando Aoi-ku, Shizuoka, Japan	2014–2019	RC	>3 months after diagnosis	35	27.3±14.2*	Type IIIb	Uncomplicated

*, calculated from median and range/IQR using methods of Wan *et al.* NR, not reported; RC, retrospective cohort study; PC, prospective cohort study.

Operative details were poorly reported across the included studies, which limited the statistical analysis. Left subclavian artery coverage was performed in 38.6% of patients (95% CI, 28.9–48.7). Revascularization of the left subclavian artery in these instances of left subclavian coverage was poorly reported. The pooled weighted technical success was high at 99.0% (95% CI, 97.7–99.8) and the need for open surgical conversion was low at 0.4% (95% CI, 0.01–1.3). The usage of spinal drainage peri- or intra-operatively was poorly reported. Other operative details including number of stents used per patient and stent dimensions are summarized in Table S3.

Early post-operative outcomes

All 48 included papers reported early (<30 days) mortality rates. The weighted pooled estimate of early all-cause mortality was 1.6% (95% CI, 0.8–2.6; I^2=44%) (*Table 2*). Pooled rates for early aortic-related mortality were low (0.5%; 95% CI, 0.1–1.3; I^2=35%). Twenty-five studies reported rates for retrograde type A dissection and the pooled estimate for this outcome was 1.4% (95% CI, 0.6–2.5; I^2=15%). The weighted pooled rates for CVA and SCI were 1.1% (95% CI, 0.5–1.7; I^2=2%) and 0.9% (95% CI, 0.3–1.6; I^2=16%), respectively. The rate of renal insufficiency was 3.2% (95% CI, 1.3–5.7; I^2=68%). Other early post-operative outcomes were inconsistently reported, such as length of intensive care unit stay/length of hospital stay, pneumonia, infection and access site complications. Rates of endoleaks were inconsistently reported and therefore a meta-analysis for this outcome was not performed. Data regarding reported rates of endoleaks are summarized in Table S4. Other early post-operative outcomes are summarized in *Table 2*.

Late outcomes

Late follow-up all-cause mortality was reported in almost all of the included studies (46/48). Weighted pooled estimate for late all-cause and aortic-related mortality were 8.0% (95% CI, 5.8–10.5; I^2=72%) and 2.4% (95% CI, 1.3–3.6; I^2=29%), respectively (*Table 3*). Reintervention mortality rate was low at 0.1% (95% CI, 0.0–0.6; I^2=0%). Rates of retrograde type A aortic dissection and aortic rupture were also low at 0.8% (95% CI, 0.2–1.6.; I^2=0%) and 1.2% (95% CI, 0.3–2.4; I^2=31%), respectively. Rates for CVA and SCI were inconsistently reported for late follow-up, however, when reported these rates were low (between 0 and 3.2%). Rates of endovascular reintervention were 10.1% (95% CI, 6.8–13.9; I^2=71%) and open surgical reintervention surgical reintervention were 6.7% (95% CI, 4.0–10.0; I^2=74%). Pooled estimate of complete false lumen thrombosis for 823 patients across 23 studies was 54.0% (95% CI, 42.0–65.7; I^2=92%). The reported rates of complete false lumen thrombosis varied significantly across studies shown by the high heterogeneity result. Complete false-lumen thrombosis was also inconsistently defined in the included studies as either complete thrombosis along the length of the stent graft or the entire length of the thoracic/thoracoabdominal aortic dissection. Again, reported rates of endoleaks were inconsistently reported for late follow-up (Table S4). Other late outcomes of interest, such as rates of stent graft induced new entry tears, were inconsistently reported among the included studies.

Survival

Aggregation of overall survival was performed from 23 of the included studies (20,26-28,32,33,35,37,38,40,42,45-49,52-54,56,62-64). Overall survival rates at 1-, 2-, 3-, 4- and 5-year were 91.5%, 88.5%, 84.7%, 82.2% and 77.7%, respectively (*Figure 2*). At 10-year post TEVAR for CTBAD the overall survival rate was 56.3%. Aorta-related survival was aggregated from six included studies (29,35,42,46,54,62). Aorta-related survival rates were 97.2%, 95.8%, 94.9%, 94.4%, 94.4% and 90.9% at 1-, 2-, 3-, 4-, 5- and 10-year, respectively (*Figure 3*).

Freedom from re-intervention

Kaplan-Meier curves reporting rates of freedom from re-intervention were available in 10 of the included studies for aggregation (29,33-35,46-49,63,64). Rates of freedom from re-intervention at 1-, 2-, 3-, 4- and 5-year were 85.2%, 80.6%, 79.2%, 77.6%, and 73.3%, respectively (*Figure 4*). At 10-year post TEVAR for CTBAD freedom from re-intervention was 55.1%.

Discussion

CTBAD remains a challenging pathology with regards to the optimal management strategy. TEVAR has undergone considerable evolution and expansion over the past two decades and provides a less invasive procedure compared to open surgery with promising results. However, the role of TEVAR in CTBAD remains controversial with most

Table 2 Early post-operative outcomes (<30 days)

Parameter	Events/total	Number of studies	Weighted pooled estimate (%) (95% CI)	Heterogeneity I^2 (%)
All-cause mortality	75/2,641	48	1.6 (0.8–2.6)	44
Aortic-related mortality	32/2,000	39	0.5 (0.1–1.3)	35
Retrograde TAAD	32/1,299	25	1.4 (0.6–2.5)	15
Cerebrovascular accident	43/2,051	42	1.1 (0.5–1.7)	2
Spinal cord injury	42/2,017	40	0.9 (0.3–1.6)	16
Rupture	11/880	20	0.5 (0.1–1.3)	0
Renal insufficiency	42/1,125	23	3.2 (1.3–5.7)	68

CI, confidence interval; TAAD, type A aortic dissection.

Table 3 Late outcomes (>30 days)

Parameter	Events/total	Number of studies	Weighted pooled estimate (%) (95% CI)	Heterogeneity I^2 (%)
All-cause mortality	219/2,322	46	8.0 (5.8–10.5)	72
Aortic-related mortality	51/1,494	32	2.4 (1.3–3.6)	29
Reintervention-related mortality	8/949	20	0.1 (0.0–0.6)	0
Retrograde TAAD	18/1,141	21	0.8 (0.2–1.6)	0
Rupture	26/1,179	21	1.2 (0.3–2.4)	31
Endovascular reintervention	136/1,280	29	10.1 (6.8–13.9)	71
Surgical reintervention	132/1,433	29	6.7 (4.0–10.0)	74
Complete false lumen thrombosis	478/873	23	54.0 (42.0–65.7)	92

CI, confidence interval; TAAD, type A aortic dissection.

institutions favoring optimal medical therapy as the primary management strategy for uncomplicated cases. The aim of this systematic review was to provide an updated and comprehensive review of the available evidence regarding the safety and efficacy of TEVAR in patients with CTBAD.

The pooled in-hospital all-cause and aortic-related mortality rates in this present study were low at 1.6% and 0.5%, respectively. On the contrary, some of the included studies reported higher mortality rates, such as the study by Puech-Leao *et al.*, which reported an in-hospital mortality rate of 12.0% (64). However, this was a small cohort of patients (37), and the majority of the deaths were related to retrograde type A dissections. The mortality rates in this present study also highlight the difference between outcomes for acute and chronic aortic dissections undergoing TEVAR, especially in the early post-operative period. A recent meta-analysis assessing TEVAR in type B aortic dissections reported in-hospital all-cause and aortic-related mortality rates of 9.4% and 5.6%, respectively, during the acute phase of the disease (66). Interestingly, in that same study during late follow-up the all-cause mortality rate for TEVAR in acute phase dissection were similar to this present study, reported at 10.0%.

Important causes of early mortality after TEVAR are aortic rupture and retrograde type A dissection. It has been hypothesized that proximal bare springs or barbs for fixation of the stent in some models of endoprosthesis can increase the risk of retrograde dissection, along with guidewire manipulation and device delivery sheaths (67). The incidence of both rupture and retrograde dissection were low in the present review, at 0.5% and 1.4%, respectively. This rate of 1.4% for retrograde type A dissection compares

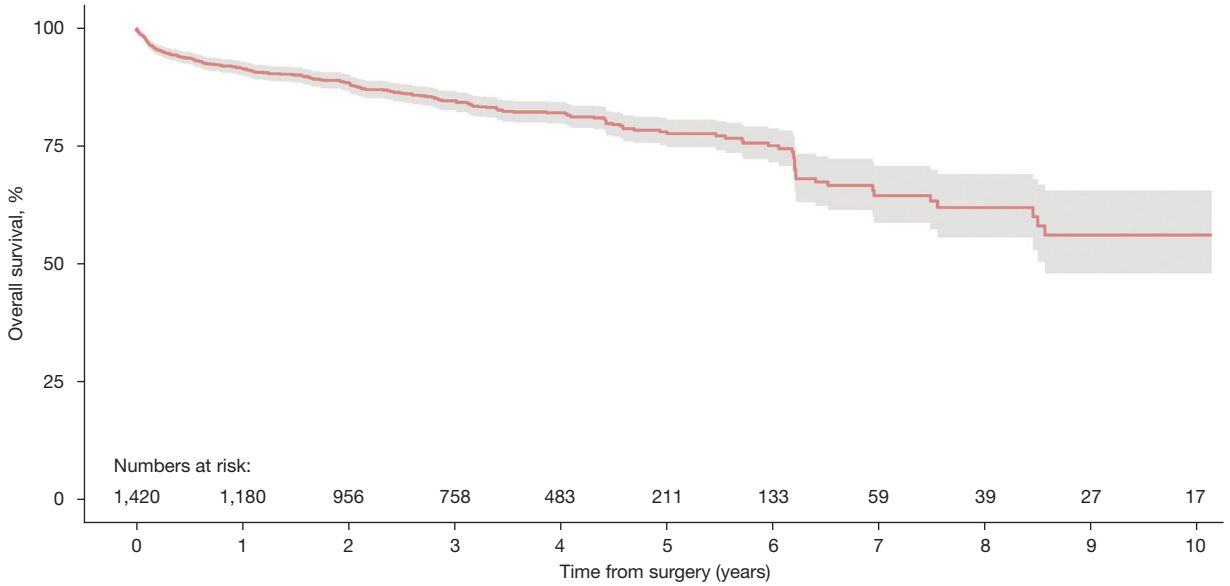

Figure 2 Aggregated overall survival after TEVAR in CTBAD (shaded region represents 95% CI). TEVAR, thoracic endovascular aortic repair; CTBAD, chronic type B aortic dissection.

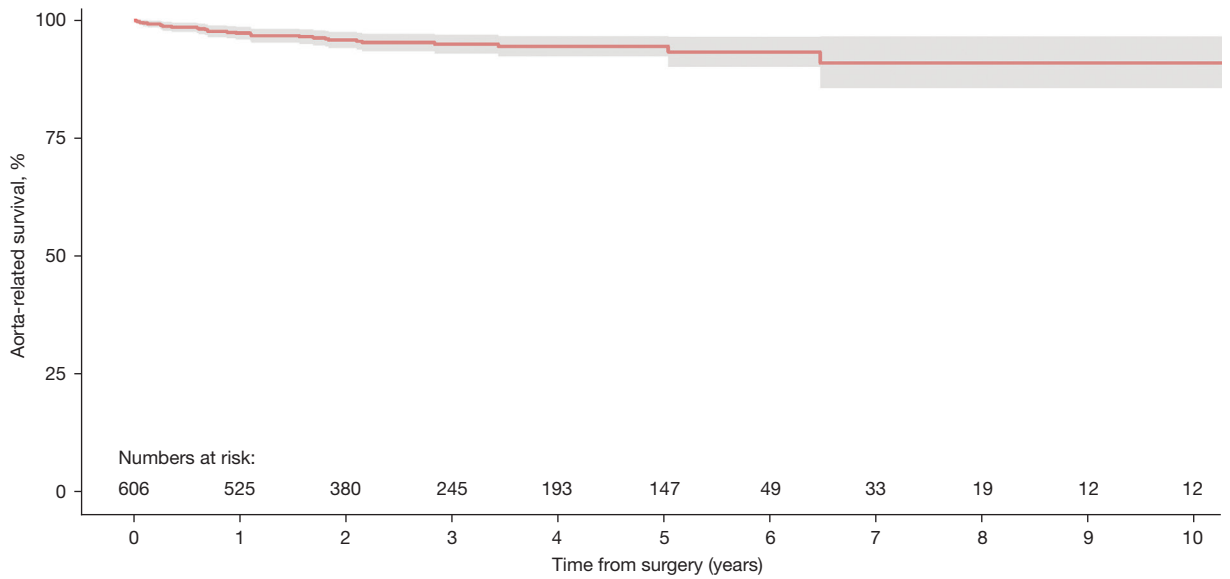

Figure 3 Aggregated aorta-related survival after TEVAR in CTBAD (shaded region represents 95% CI). TEVAR, thoracic endovascular aortic repair; CTBAD, chronic type B aortic dissection.

similarly to the 1.33% in the European Registry on Endovascular Aortic Repair complications and favorable to the 3.17% reported in a recent single center study of over 850 patients (68). The rates of CVA and SCI in the present study were 1.1% and 0.9% respectively. This is lower than the rates reported by Boufi *et al.*, who when comparing TEVAR to open surgery for CTBAD reported CVA and SCI rates of 2.7% and 2.2%, respectively, for TEVAR and 4.5% and 5.0%, respectively, for open surgery (69).

One main concern regarding TEVAR for CTBAD is its durability and the requirement for subsequent reintervention. In the present study, the rate of secondary

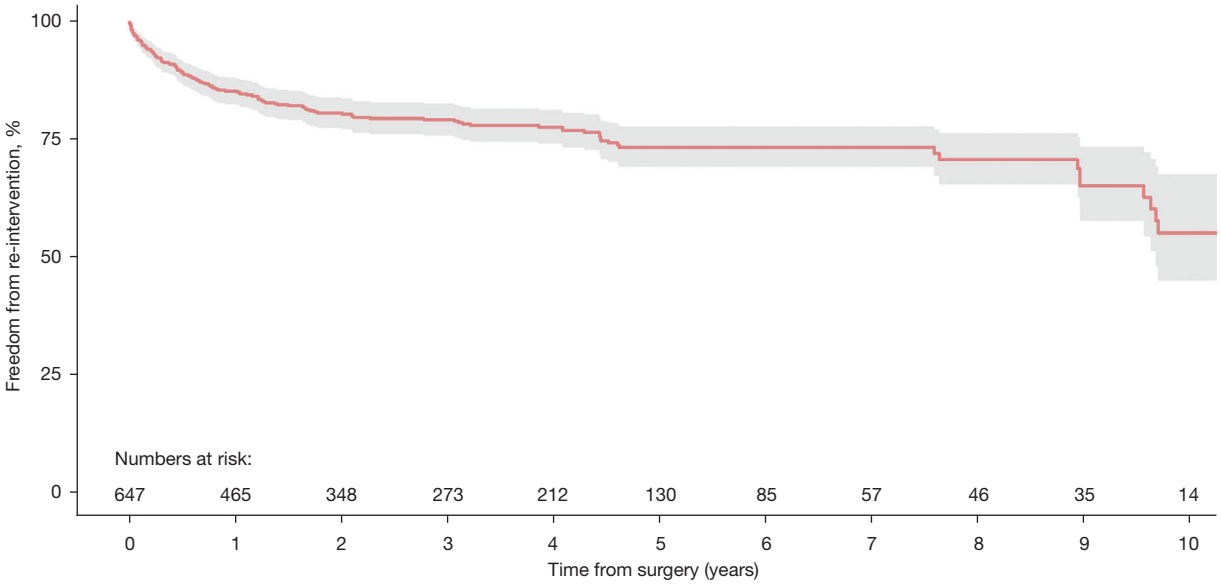

Figure 4 Aggregated freedom from re-intervention after TEVAR in CTBAD (shaded region represents 95% CI). TEVAR, thoracic endovascular aortic repair; CTBAD, chronic type B aortic dissection.

endovascular intervention was 10.1% and 6.7% for open surgical reintervention. These are comparable to a recent review of over 5,000 patients (both acute and chronic cases) that reported rates of 12.5% and 6.1% for endovascular and surgical reintervention, respectively (66). However, another systematic review reported varying rates of endovascular reintervention from 4.3 to 47.4% after TEVAR for CTBAD (70).

It has previously been reported in the literature that TEVAR has higher rates of 1-year survival when compared to open surgery (90–93% *vs.* 79–81%, respectively), however, at 3-year follow-up this survival benefit was lost (TEVAR 67% *vs.* open surgery 71%) (71). However, a recent meta-analysis reported that there was no benefit of one technique over the other regarding 1- and 3-year survival (69). In comparison, the results of the current study report a similar 1-year survival rate for TEVAR at 91.5%, however, the 3-year survival rate is considerably higher at 84.7%. When compared to medical therapy alone, the INSTEAD trial reported improved 5-year aorta-specific survival and delayed disease progression for TEVAR with optimal medical treatment for patients with uncomplicated CTBAD (72). This is the only randomized control trial to date comparing TEVAR to optimal medical therapy for uncomplicated CTBAD [the TEVAR cohort of which is included in this present study in the larger MOTHER database (46)].

It is known that aortic remodeling and complete thrombosis of the false lumen are important factors for positive long-term results for TEVAR in type B dissection (26,37,43). In CTBAD, there is thickening of the dissection septum which progresses over time, and consequently, there is usually less aortic remodeling seen in chronic compared to acute dissections. Recently, the VIRTUE study confirmed this, reporting that when compared to acute or sub-acute patients, patients who underwent TEVAR with CTBAD had lesser degrees of aortic remodeling (73). Unfortunately, in the present study, aortic remodeling was either poorly or inconsistently reported across studies, limiting its analysis. Rates of complete false lumen thrombosis were the most consistent outcome reported across approximately half of the included studies. Even though the pooled estimate for this outcome was 54.0%, the specific anatomic location where the false lumen thrombosis was complete (i.e., length of the stent graft versus entire length of dissected aorta) was poorly reported. This is lower than the 71.7% rate of complete false lumen thrombosis reported by Boufi and colleagues for TEVAR patients in CTBAD (69). To improve the future analysis of this important outcome of aortic remodeling and its link to long-term patient outcomes, guidelines and consensus reporting standards should be implemented to improve the degree of heterogeneity in reporting of this outcome in the literature.

Another important consideration when reviewing the

available literature is the recent change in definition of chronicity for type B aortic dissections. Some studies divide type B dissection patients into three distinct different groups, acute (<2 weeks), sub-acute (2 weeks to 3 months) and chronic (>3 months), including a separate "sub-acute phase" (74). A large number of the more recent studies included in this review defined CTBAD to be greater than 3 months from diagnosis or symptom onset (51,57,58,62,63,65), whereas the overall majority of studies, especially those published before 2014, used a definition of greater than 2 weeks to define chronicity. It is unclear exactly how this change in classification will affect long-term results or ideal timing for intervention, however, some studies have reported that patients treated in the sub-acute phase exhibit better early, mid- and long-term outcomes when compared to acute or chronic patients who do not need emergent intervention (62). This hypothesis however requires prospective randomized controlled trials to confirm this reported evidence in observational studies.

There are a number of important limitations to consider when interpreting the results described in this present study. As mentioned above, there were different definitions of chronicity and indications for TEVAR in CTBAD used across the included studies. There was also significant heterogeneity for some of the reported outcomes, including late all-cause mortality, endovascular and surgical reintervention rates and false lumen thrombosis rates. This could represent a number of different factors, such as different patient population or selection, differing centers with varying operator experience or the different endoprostheses used across the included studies. Studies also inconsistently reported loss to follow-up and some studies reported outcomes with high complication rates in small patient populations. The observational nature of all included studies also presents an inherent source of bias in the present study. There were also varying definitions of technical success across the included studies.

Conclusions

In summary, TEVAR provides a safe and effective treatment modality for patients with CTBAD. It can be performed with low complication rates in high volume, experienced centers. Due to the limited evidence, based mainly on retrospective cohort studies, and the heterogeneity of the reported outcomes, the optimal treatment strategy for CTBAD remains debatable. Further high quality prospective multicenter registry data and randomized control trials are required to evaluate the different treatment strategies. Consensus reporting standards with a focus on aortic remodeling are required to improve our understanding of the long-term outcomes of TEVAR in CTBAD.

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Footnote

Conflicts of Interest: Dr. OP provides consultation for and participates in clinical trials with Medtronic and W.L. Gore & Associates. The other authors have no conflicts of interest to declare.

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Supplementary

Table S1 Indications for CTBAD endovascular repair

Primary author	Indication(s)
Kato, 2002	(I) visceral or leg ischemia, aortic rupture, refractory hypertension, and/or refractory pain; (II) descending thoracic aorta 50 mm or larger in diameter
Greenberg, 2005	(I) Chronic aortic dissection with aneurysm 5 cm or rapid growth; (II) life expectancy >2 years; (III) high-risk for open surgical repair; (IV) absence of an uncorrectable coagulopathy; (V) absence of allergy to stainless steel or polyester; (VI) absence of a serious groin infection; (VII) absence of systemic sepsis
Baumgart, 2006	(I) TAA and PAU recurrent pain; (II) enlargement of aortic diameter to more than 5 cm
Bocker, 2006	(I) Chronic expansive aortic dissection (CEAD) greater than 55 mm of maximum diameter
Song, 2006	(I) Chronic dissection with aneurysmal dilatation of the proximal descending aorta >5 cm, or; (II) chronic dissections with acute symptoms
Thompson, 2007	NR
Jing, 2008	(I) Aortic rupture; (II) continued chest pain despite rigorous medical management; (III) refractory hypertension or branch vessel ischemia; (IV) a maximum diameter of >50 mm (chronic AD) of the descending thoracic aorta or; (V) documented aortic enlargement of >1.0 mm/year
Marcheix, 2008	Aneurysmal dilatation defined as diameter over 40mm and rapid aortic enlargement of more than 0.5 cm in 6-month period
Sayer, 2008	Presence of complications (rupture, acute dissection, end organ ischemia or pain), maximum short axis thoracic aortic diameter exceeding 5.5 cm, or rapid growth of the thoracic aorta (1 cm in 6 months or local protocol).
Alves, 2009	(I) Persistent symptoms or; (II) total aortic diameter greater than 55 mm
Guangqi, 2009	(I) Pseudo-aneurysm with diameter 5 cm or larger or rapid enlargement >0.5 cm per year and; (II) acute symptoms
Kim, 2009	(I) Persistent or recurrent pain that was unresponsive to medical treatment; (II) dynamic obstruction; (III) aortic diameter ≥6 cm; (IV) progressive enlargement of the false lumen (>5 mm), or; (V) continuous false lumen leakage.
Manning, 2009	Aneurysmal expansion of the false lumen
Czerny, 2010	(I) Median diameter DTA > or equal to 6cm
Xu, 2010	(I) Type B aortic dissection confirmed by magnetic resonance angiography or computed tomographic angiography (CTA) with patent false lumen and no intramural hematoma; (II) time between onset of dissection and admission longer than 1 month; (III) arch diameter (landing zone) less than 38 mm; (IV) adequate access route, and; (V) no aberrant right subclavian artery; (V) distance between entry tear and opening of left subclavian artery (LSCA) more than 10 mm; (VI) no Marfan syndrome or any suspected connective tissue diseases.
Kang, 2011	(I) Maximum aortic diameter of at least 55 mm; (II) rapid aortic growth (10 mm/y); (III) clinical or radiographic evidence of rupture; (IV) intractable chest pain despite maximal medical therapy, and; (V) visceral, renal, or lower extremity malperfusion.
Orberhuber, 2011	(I) Maximum diameter of thoracic aorta >6 cm, and; (II) rapid expansion >1 cm/year
Parsa, 2011	(I) Rapid enlargement >5 mm in 6 months; (II) aneurysm >5.5 cm; (III) saccular aneurysm >2 cm protrusion beyond aortic wall
Andacheh, 2012	(I) Aneurysmal enlargement; (II) failure of medical management; (III) perforation
Mani, 2012	(I) Aneurysmal dilatation >5.5 cm; (II) rapid expansion of >1cm in one year, or; (III) symptomatic disease (i.e., leaking aneurysm, back or chest pain with dilatation)
Nathan, 2012	(I) Aneurysm >5.5 cm; (II) rapid aneurysmal enlargement >5 mm over 6 months; (III) saccular aneurysm >2 cm and; (IV) rupture

Table S1 (*continued*)

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Table S1 (*continued*)

Primary author	Indication(s)
Qing, 2012	(I) Patients were categorized as having chronic type B aortic dissections (group A) if their maximum aortic diameters were <50 mm and as having; (II) chronic dissections with aneurysms (group B) if their maximum aortic diameters were ≥50 mm
Yang, 2012	(I) Refractory hypertension; (II) persistent or recurrent pain; (III) aneurysmal rupture; (IV) visceral or lower limb ischemia; (V) aneurysmal dilatation with aortic diameter 6.0 mm
Chen, 2013	Descending thoracic aorta ≥40 mm in diameter at onset of aortic dissection with complications requiring surgical intervention, including visceral or leg ischemia, aortic rupture, refractory hypertension, refractory pain, or growth of ulcer-like projections.
Jia, 2013	(I) Patients who were diagnosed as having uncomplicated type B aorta dissection; (II) patients who were at least 20 years old but were younger than 80 years, and; (III) patients who were able to cooperate with the study procedure and provided written informed consent
Lee, 2013	(I) Rapid enlargement of the aorta or aortic diameter > 55 mm; (II) persistent pain; (III) clinical or radiological malperfusion; (IV) rupture
Leshnower, 2013	(I) A maximum aortic diameter 5.5 cm or greater; (II) rapid aortic growth of 5 mm or greater over a 6-month period.
Nozdrzykowski, 2013	(I) Enlarged aortic diameter; (II) impending rupture; (III) end-organ malperfusion, or; (IV) recurrent pain
Patterson, 2013	Varied across the included trials in the MOTHER Database
Scali, 2013	Maximal thoracic aneurysm diameter ≥6.0 cm or documented growth rate ≥1.0 cm on serial centreline computed tomography (CTA) measurements over 12 months
Andersen, 2014	For elective: (I) aneurysmal degeneration with an absolute aortic diameter of ≥5.5 cm; (II) rapid aneurysm enlargement (>5 mm in 6 months); (III) saccular aneurysm protruding ≥2 cm beyond the aortic wall; For non-elective: (I) symptomatic aneurysm with impending rupture; (II) aorto-esophageal fistula; (III) ruptured aneurysm; (IV) dynamic iliofemoral malperfusion
Kitamura, 2014	NR
Lombardi, 2014	(I) Branch vessel obstruction/compromise; (II) impending rupture; (III) resistant hypertension; (IV) persistent pain/symptoms; (V) or aortic growth >5 mm in 3 months (or transaortic diameter >40 mm
Song, 2014	Newly developed, continuing back pain, and aneurysmal degeneration (maximal thoracic aneurysm diameter 5.5 cm or a documented growth rate of 0.5 cm within 6 months seen on serial computed tomography angiograms
Nathan, 2015	(I) Aneurysm size ≥5.5 cm; (II) Aneurysm expansion ≥ 0.5 cm over 6 months; (III) refractory chest pain, or; (IV) rupture
van Bogerijen, 2015	(I) Aortic enlargement (defined as maximum aortic diameter 55 mm or rapid aortic enlargement (5 mm/year); (II) clinical or radiologic evidence of rupture; (III) acute on chronic dissection; and; (IV) distal extension of the initial dissection
Zhang, 2017	(I) Maximum aortic diameter >55 mm; (II) an aortic increase of >5 mm within 3 months; (III) detection of organ ischemia; (IV) recurrence of other symptoms (pleural effusion, refractory pain, and resistant hypertension). Refractory pain was defined as ongoing symptoms of back and/or chest pain requiring narcotic medications in case of excellent blood pressure control.
Chou, 2018	(I) Aneurysmal degeneration with an aortic diameter >6 cm, or; (II) rapid aneurysmal growth (>0.5 cm within 0.5 year)
Huang, 2018	(I) Progressive aneurysmal enlargement to a maximum thoracic aortic diameter of greater than 6 cm or an annual increase in diameter of greater than 0.5 cm with maximal size greater than 5 cm on surveillance imaging
Tjaden, 2018	NR

Table S1 (*continued*)

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Table S1 (*continued*)

Primary author	Indication(s)
Kim, 2019	(I) Newly developed aneurysms; (II) intractable back pain; (III) aneurysmal degeneration (maximal thoracic aneurysm diameter>55 mm or; (IV) a documented growth rate of 5 mm in 6 months, as observed in serial computed tomography (CT) angiograms).
Wang, 2019	NR
Zha, 2019	(I) Impending rupture; (II) organ malperfusion; (III) resistant hypertension unresponsive to medical therapy; (IV) refractory pain (ongoing symptoms in the back and/or chest pain requiring narcotic medications); (IV) aortic growth (aortic diameter increase >5 mm within 3 months); (V) the patient's will or surgeon's decision.
Conway, 2020	(I) Pain; (II) Refractory hypertension; (III) aneurysm; (IV) rupture
Li, 2020	(I) Refractory hypertension; (II) intractable pain; (III) rupture or impending rupture; (IV) visceral malperfusion; (V) lower extremity ischemia; (VI) true lumen collapse <25% aortic diameter; (VII) rapid enlargement >4mm/year; (VIII) aneurysmal dilatation >55 mm
Oishi, 2020	(I) Acute enlargement of aneurysm diameter >5 mm over 6 months; (II) enlargement of the aneurysmal diameter >55 mm, and/or; (III) rupture or impending rupture
Puech-Leao, 2020	The maximum diameter greater than 55 mm, measured at the axial projection
Ueki, 2021	Rupture, impending rupture, aneurysmal degeneration (maximum aortic diameter of >55 mm), and rapid growth of the aortic diameter (>5 mm per 6 months).

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Table S2 Patient's characteristics

Primary author	n	Age (years)	Male (%)	Interval between diagnosis and repair (months)**	HTN (%)	DM (%)	CVA (%)	CAD (%)	COPD (%)	Smoking (%)	Renal in-sufficiency (%)	Previous cardiac/aortic surgery (%)	CTD (%)	Rupture (%)	Mal-perfusion syndrome (%)
Kato, 2002	14	61.0±14.0	86.0	35.0±94.0	64.0	NR	0.0	7.1	NR	NR	0.0	21.4	NR	NR	NR
Greenberg, 2005	15	54.0	73.0	NR	NR	NR	NR	NR	NR	NR	NR	26.7	NR	NR	NR
Baumgart, 2006	35	64.0±14.0	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Bockler, 2006	15	59.5±10.8*	80.0	NR	NR	NR	NR	NR	NR	NR	NR	6.7	NR	0.0	6.7
Song, 2006	17	64.0±14.0	58.8	NR	94.1	11.8	0.0	11.8	29.4	NR	29.4	11.8	0.0	6.3	6.3
Thompson, 2007	52	62.0±14.0	NR	NR	81.0	NR	NR	NR	NR	NR	21.4	59.5	9.6	NR	NR
Jing, 2008	35	69.0±12.7	85.7	NR	80.0	25.7	2.9	11.4	11.4	65.7	5.7	NR	NR	5.7	NR
Marcheix, 2018	15	38.7±12.8	66.7	NR	46.7	6.7	NR	NR	40.0	NR	33.3	73.3	100.0	NR	NR
Sayer, 2008	40	66.6±11.9	65.0	NR	67.5	10.0	NR	NR	NR	52.5	37.5	12.5	17.5	NR	NR
Alves, 2009	61	56.4±10.8	77.0	10.5±18.0	37.0	4.9	NR	NR	NR	NR	14.8	NR	NR	NR	NR
Guangqi, 2009	49	57.1±10.0	93.9	NR	95.9	NR	NR	NR	NR	NR	6.1	NR	2.0	4.1	0.0
Kim, 2009	72	55±12.0	65.3	NR	93.1	13.9	4.2	16.7	NR	47.2	5.5	5.6	2.8	NR	NR
Manning, 2009	10	63.0±8.5*	80.0	26.3±17.3*	NR	NR	NR	NR	NR	NR	NR	10.0	NR	NR	NR
Czerny, 2010	14	63.0	79.0	31.5±20.0*	100.0	NR	NR	NR	68.0	NR	NR	21.4	NR	NR	NR
Xu, 2010	84	53.3±11.6	82.1	13.9±22.0	79.8	10.7	NR	11.9	NR	31.0	NR	NR	1.2	3.6	NR
Kang, 2011	76	61.5±12.5	64.0	25.0±31.0	99.0	10.5	11.8	38.2	22.4	42.1	18.4	22.4	2.6	1.3	1.3
Orberhuber, 2011	19	57.0±11.5*	89.5	33.3±14.8*	94.7	NR	NR	21.1	21.1	47.4	31.6	NR	5.3	NR	NR
Parsa, 2011	51	57.0±12.0	72.5	46.2±53.7	94.1	7.8	5.9	NR	17.6	52.9	23.5	27.5	NR	NR	NR
Andacheh, 2012	73	58.0	71.0	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Mani, 2012	58	66.0±11.0	82.8	29.0±31.0	NR	NR	NR	NR	NR	NR	NR	13.8	NR	5.2	NR
Nathan, 2012	27	67.5±9.6	66.7	47.0±44.2	100.0	11.1	NR	14.8	25.9	NR	11.1	33.3	NR	3.7	0.0
Qing, 2012	32	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Yang, 2012	28	62.7±12.6	85.7	49.8±55.8	92.9	14.3	10.7	14.3	7.1	42.9	7.1	NR	NR	NR	NR
Chen, 2013	56	53.9±10.7	78.6	0.8±0.2	87.5	10.7	7.1	32.1	3.6	67.9	5.4	NR	NR	NR	NR
Jia, 2013	208	52.1±21.8	74.0	1.0±0.2*	89.9	NR	9.6	12.0	10.8	NR	7.7	NR	NR	NR	NR
Lee, 2013	71	55.1±12.1	70.4	26.0±42.9*	81.7	5.6	2.8	2.8	NR	39.4	4.2	23.9	7.0	2.8	9.9
Leshnower, 2013	31	62.0±10.0	58.0	47.0±44.0	77.0	9.7	9.7	NR	41.9	NR	6.5	32.3	NR	0.0	NR
Nozdrzykowski, 2013	32	61.2±10.0*	71.9	4.4±6.4*	100.0	28.1	6.2	18.8	21.9	NR	40.6	18.8	0.0	18.7	18.7
Patterson, 2013	195	63.1±11.0*	83.0	NR	83.0	11.3	3.1	NR	10.3	61.0	12.0	NR	NR	NR	NR
Scali, 2013	80	60.0±13.0	88.0	26.8±12.2*	95.0	NR	6.7	23.8	15.0	51.3	30.0	32.5	8.8	NR	NR
Andersen, 2014	44	58.0 ±11.1*	64.0	31.0±50.4*	98.0	11.4	0.0	11.4	15.9	59.1	15.9	27.3	0.0	NR	NR
Kitamura, 2014	45	55.5±13.1	94.3	16.8±33.6	NR	NR	NR	NR	NR	NR	7.5	NR	0 .0	NR	NR

Table S2 (*continued*)

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Table S2 (*continued*)

Primary author	n	Age (years)	Male (%)	Interval between diagnosis and repair (months)**	HTN (%)	DM (%)	CVA (%)	CAD (%)	COPD (%)	Smoking (%)	Renal insufficiency (%)	Previous cardiac/aortic surgery (%)	CTD (%)	Rupture (%)	Mal-perfusion syndrome (%)
Lombardi, 2014	31	59.8±13.3	77.4	1.4±0.8	100.0	12.9	9.7	16.1	6.5	48.1	6.5	NR	NR	NR	58.1
Song, 2014	20	50.2	85.0	18.1±11.9	90.0	5.0	0.0	5.0	NR	70.0	NR	50.0	5.0	NR	NR
Nathan, 2015	47	58.3±11.7	74.5	53.8±50.1	87.2	NR	NR	NR	NR	NR	NR	27.7	14.9	4.3	NR
van Bogerijen, 2015	32	69.2±10.7	46.9	20.3±27.6	93.8	9.4	3.1	25.0	25.0	71.0	NR	34.4	3.1	NR	NR
Zhang, 2017	25	65.5±10	64.0	1.9±2.2*	88.0	16.0	NR	4.0	0.0	40.0	8.0	NR	0.0	NR	NR
Chou, 2018	23	63.9±7.9	87.0	19.9±11.1	78.2	8.7	8.7	17.4	13.0	26.0	30.4	NR	0.0	0.0	0.0
Huang, 2018	65	56.3±3.8*	81.5	18.2±9.4*	95.3	7.7	7.7	20.0	NR	38.4	4.6	NR	4.6	NR	NR
Tjaden, 2018	94	61.0±2.8*	85.1	NR	93.6	6.4	7.4	20.2	10.6	50.0	24.5	22.3	1.1	NR	NR
Kim, 2019	75	58.2±12.1	78.7	15.8±20.2*	82.7	8.0	NR	6.7	4.0	52.0	2.7	52.0	2.7	NR	NR
Wang, 2019	193	62.2±12.4	71.5	NR	96.4	13.0	6.2	13.5	14.5	60.6	4.7	21.2	1.6	3.1	7.3
Zha, 2019	23	55.9±12.1	78.3	NR	87.0	17.4	NR	4.3	8.7	69.6	NR	NR	NR	NR	8.7
Conway, 2020	208	65.0±12.6*	72.1	NR	88.9	12.0	11.5	13.0	19.2	68.3	3.4	34.6	4.3	4.8	NR
Li, 2020	34	56.1±14.2	82.4	23.0±14.4*	82.4	2.9	2.9	5.8	8.8	47.1	0.0	NR	NR	0.0	5.9
Oishi, 2020	40	66.5±11.6	65.0	NR	97.5	12.5	NR	NR	NR	57.5	7.5	47.5	5.0	NR	0.0
Puech-Leao, 2020	42	59.1	76.2	NR	NR	NR	NR	NR	NR	NR	NR	NR	0.0	NR	NR
Ueki, 2021	35	63.4±2.9*	74.3	38.8±52.9*	94.3	14.3	5.7	5.7	NR	71.4	2.9	48.6	NR	11.4	NR
Pooled estimate % (95% CI)	2,641	60.5 (58.1–62.8)	76.7 (73.7–79.2)	20.6 (14.5–25.8)	89.4 (85.8–92.7)	10.4 (8.9–11.9)	5.6 (4.2–7.2)	13.8 (10.8–16.9)	15.0 (11.4–18.9)	52.7 (48.1–57.4)	11.3 (8.1–14.9)	29.2 (23.8–34.8)	4.3 (1.8–7.4)	–	–

*, calculated from median and range/IQR using methods of Wan et al.; **, when reported in days this was converted to months by dividing the number by 30. HTN, hypertension; DM, diabetes mellitus; CVA, cerebrovascular accident; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; CTD, connective tissue disorder; NR, not reported.

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Table S3 Procedural details

Primary author	Endoprostheses	Number of stent-grafts per patient	Stent length; diameter (mm)	LSA coverage (%)	Technical success (%)	Primary conversion (%)	Operative time (minutes)	CSF drain (%)	Over–sizing (%)
Kato, 2002	Custom graft	NR	NR	NR	100.0	0.0	NR	NR	NR
Greenberg, 2005	Zenith TX1, Zenith TX2	NR	NR	NR	NR	0.0	102.0	82.0	NR
Baumgart, 2006	Talent, GoreTAG	1.3±0.5	L: 123±23, D: 37±6	NR	100.0	0.0	NR	NR	NR
Bocker, 2006	Excluder, Talent, Endofit	1.1 (1–2)*	L: 162, D: 35.2	40.0	100.0	0.0	NR	NR	NR
Song, 2006	AneuRx and Talent	NR	L: 188±61	23.5	NR	NR	149.9±88.5	NR	NR
Thompson, 2007	Valiant	1.6	NR	NR	NR	0.0	NR	NR	NR
Jing, 2008	Talent, Zenith, Aegis	NR	L: 102.4±15.3, D: 37.2±4.2	5.7	100.0	NR	139±25	NR	NR
Marcheix, 2008	Talent	1.5±0.7	NR	13.3	66.7	0.0	NR	NR	5–10%
Sayer, 2008	Valiant, Talent, Excluder	2.1	L: 204±20 mm proximal: 32.9±1.3 mm, distal: 26.9±2.0	NR	100.0	NR	133.5	NR	NR
Alves, 2009	Braile stent	1.7±0.8	NR	NR	98.4	3.3	NR	NR	NR
Guangqi, 2009	Talent, Zenith, Ankura, Aegis	1.1±0.3	NR	22.4	77.6	0.0	NR	NR	NR
Kim, 2009	Custom made, separate-type modular stent	NR	L: 83±27, D: 33.8±6.8 prox, 33.2±6.3 distal	NR	97.2	NR	NR	NR	NR
Manning, 2009	Zenith, Excluder	NR	NR	30.0	100.0	0.0	NR	NR	10%
Czerny, 2010	Talent, Valiant, Relay	NR	L: 190 (100–250)*	NR	85.7	0.0	NR	NR	15–20%
Xu, 2010	Talent, EndoFit, Hercules, Vasoflow, Grikin	1.11	NR	4.8	NR	NR	150±18	NR	NR
Kang, 2011	Core TAG, Cook Zenith, Medtronic Talent, Homemade	1.9	NR	38.2	96.1	NR	NR	63.0	NR
Orberhuber, 2011	TAG, CTAG, Captivia, Valiant, Zenith	1.1	NR	47.4	94.7	NR	68 (55–83)*	NR	NR
Parsa, 2011	TAG, Zenith TX2, Talent	2.0±0.7	NR	68.6	100.0	NR	NR	17.6	NR
Andacheh, 2012	Talent, Valiant Captivia	NR	NR	NR	98.6	0.0	NR	0	NR
Mani, 2012	TAG, Zenith TX2, Combined, Endofit, Talent, Relay	2 (1)**	L: 220 mm (100)**	46.6	NR	3.4	98 (41)**	NR	5–15%
Nathan, 2012	TAG, Zenith TX2, Talent	NR	L: 221±43	51.9	NR	NR	NR	NR	NR
Qing, 2012	Zenith TX2	NR	NR	NR	100.0	NR	NR	NR	15.9±7.9%
Yang, 2012	NR	1.5	NR	71.4	100.0	NR	286.4±185.8	14.3	NR
Chen, 2013	Talent, Willis	1.1±0.2	L: 130.8±40.6, D: 36.5±4.3	5.4	100.0	0.0	NR	NR	NR
Jia, 2013	Valiant, Zenith TX2, Hercules	NR	NR	36.5	100.0	4.3	89 (35–180)*	NR	NR
Lee, 2013	SEAL, Taewoong, Valiant, Zenith TX2	NR	NR	NR	97.2	1.4	NR	NR	NR
Leshnower, 2013	TAG, Talent, Zenith TX2	2	L: 220±40	54.8	NR	NR	NR	45.2	NR
Nozdrzykowski, 2013	NR	NR	NR	NR	100.0	NR	NR	NR	NR
Patterson, 2013	Talent, Valiant, Captivia, Xcelerant	NR	NR	NR	NR	NR	NR	NR	NR

Table S3 (*continued*)

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Table S3 (*continued*)

Primary author	Endoprostheses	Number of stent-grafts per patient	Stent length; diameter (mm)	LSA coverage (%)	Technical success (%)	Primary conversion (%)	Operative time (minutes)	CSF drain (%)	Over–sizing (%)
Scali, 2013	Gore TAG, Cook TX2	NR	NR	75.0	98.8	NR	NR	77.5	NR
Andersen, 2014	NR	2 (1–2)*	L: 200 (160–290)**	NR	100.0	11.4	NR	NR	NR
Kitamura, 2014	Matsui–Kitamura or handmade	NR	NR	NR	100.0	NR	NR	NR	NR
Lombardi, 2014	Zenith TX2, bare metal stent	NR	NR	NR	100.0	NR	NR	NR	NR
Song, 2014	Zenith TX2 Proform, Valiant Captivia	1.5	NR	55.0	100.0	0.0	NR	65.0	10%–15%
Nathan, 2015	Zenith TX2, TAG, Talent	NR	NR	48.9	97.9	0.0	NR	74.5	10–15%
van Bogerijen, 2015	TAG, TX2, Talent	NR	NR	NR	100.0	0.0	NR	96.4	10%
Zhang, 2017	NR	1.04	NR	NR	100.0	0.0	183.7±98.4	NR	NR
Chou, 2018	TAG, Zenith, Talent, Variant, Relay	NR	D: prox 36.7±3.9, distal 33.5±4.3	8.7	100.0	0.0	286.4±185.5	4.3	10–15%
Huang, 2018	Zenith TX2, Valiant Captivia, Gore TAG	2 (1–2)*	NR	46.2	NR	NR	260 (160–330)*	NR	NR
Tjaden, 2018	TAG and CTAG	2 (1–2)*	L: 280 (150 to 400)*	41.5	NR	0.0	NR	NR	NR
Kim, 2019	NR	NR	NR	NR	NR	4.0	NR	NR	NR
Wang, 2019	Valiant, Gore CTAG, Cook TX2/Alpha	NR	NR	NR	98.9	NR	NR	65.3	NR
Zha, 2019	Captivia, Zenith, Ankura, Grink	NR	L: 193.35±13.6	56.5	100.0	NR	NR	NR	NR
Conway, 2020	NR	2 (1–2)*	NR	80.9	100.0	0.0	149.5 (103–219.5)**	68.7	NR
Li, 2020	Valiant, Zenith TX2, TAG, Hercules, Ankura	NR	L: 165.0±25.1, D: 36.0±3.3	32.4	91.2	NR	NR	NR	10%
Oishi, 2020	TAG, CTAG, Valiant, Zenith TX2, Relay Plus	NR	L: 10 (201.2±57.9) + 30 (187.7±61.6)	NR	100.0	NR	152.7±93.3(10pts) + 162.6±138.4 (30 pts)	NR	<10%
Puech-Leao, 2020	NR	1.3	NR	28.5	80.9	NR	NR	NR	NR
Ueki, 2021	Gore TAG, Valiant Captivia	2 (1–3)*	NR	37.1	100.0	NR	81.0 (50.0–214.0)*	NR	NR
Pooled estimate % (95% CI)	–	–	–	38.6 (28.9–48.7)	99.0 (97.7–99.8)	0.4 (0.01–1.3)	–	–	–

Data represented as mean ± standard deviation. *, median and range; **, median and interquartile range. LSA, left subclavian artery; L, length; D, diameter; NR, not reported.

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Table S4 Rates of endoleaks for both early (<30 days) and late (>30 days) outcomes

Primary author	Early post-op outcomes (%)	Late outcomes (%)
Kato, 2002	0.0	0
Greenber, 2005	NR	NR
Baumgart, 2006	NR	NR
Bocker, 2006	Type I: 6.7	NR
Song, 2006	NR	NR
Thompson, 2007	Type I: 6.0, Type III: 1.9	Type I: 12.0, Type III: 0.0
Jing, 2008	2.9	0
Marcheix, 2008	Type I: 26.7, Type II: 6.7	Type I: 26.7, Type III: 6.7
Sayer, 2008	5.0	NR
Alves, 2009	NR	NR
Guangqi, 2009	Type I: 12.5	NR
Kim, 2009	Type I: 8.3	Type 1: 8.3
Manning, 2009	NR	Type Ia: 20%, Type Ib: 10
Czerny, 2010	Type Ia: 14.3	14.3
Xu, 2010	8.3	NR
Kang, 2011	Type I: 9.2, Type IV: 1.3	1.3
Orberhuber, 2011	Type II: 15.8	NR
Parsa, 2011	NR	Type I: 7.8, Type II: 3.9
Andacheh, 2012	NR	9.6
Mani, 2012	NR	NR
Nathan, 2012	NR	Type II: 7.4
Qing, 2012	NR	0.0
Yang, 2012	Type I: 0.0, Type II: 3.6, Type III: 7.1, Type IV: 3.6	NR
Chen, 2013	Type I: 1.8	0.0
Jia, 2013	NR	1.4
Lee, 2013	NR	Type I: 12.7
Leshnower, 2013	NR	6.4
Nozdrzykowski, 2013	NR	NR
Patterson, 2013	NR	6.1
Scali, 2013	Type I: 1.2	6.2
Andersen, 2014	Type Ia: 2.0, Type Ib: 2.0	4.0
Kitamura, 2014	NR	NR
Lombardi, 2014	NR	NR
Song, 2014	Type Ia: 0.0, Type II: 10.0	NR

Table S4 (*continued*)

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Table S4 (*continued*)

Primary author	Early post-op outcomes (%)	Late outcomes (%)
Nathan, 2015	Type Ia: 2.1	NR
van Bogerijen, 2015	NR	Type I: 6.3, Type II: 15.6, Type III: 18.8
Zhang, 2017	NR	Type I: 0.0, Type II: 4.0
Chou, 2018	Type I: 4.3, Type II: 8.6	Type II: 8.7
Huang, 2018	NR	10.8
Tjaden, 2018	NR	12.8
Kim, 2019	NR	10.7
Wang, 2019	NR	NR
Zha, 2019	8.7	NR
Conway, 2020	Type 1a: 3.8, Type 1b: 4.6, Type II: 2.2	NR
Li, 2020	Type 1a: 8.8	Type II: 6.1
Oishi, 2020	NR	Type II: 5.0
Puech-Leao, 2020	Type Ia: 7.1	NR
Ueki, 2021	Type Ia: 8.5	NR

NR, not reported.

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