Risk Profile Analysis of Uncomplicated Type B Aortic Dissection Patients Undergoing Thoracic Endovascular Aortic Repair: Laboratory & Radiographic Predictors

Alexander Geragotellis[1], Mohammed Al-Tawil[2], Matti Jubouri[3], Sven Zhen Cian Patrick Tan[4], Ian Williams[5], and Mohamad Bashir[6]

[1]University of Cape Town
[2]Al Quds University Faculty of Medicine
[3]Hull York Medical School
[4]Queen Mary University of London Barts and The London School of Medicine and Dentistry
[5]University Hospital of Wales
[6]NHS Wales Health Education and Improvement Wales

April 18, 2022

Abstract

Background: There is emerging evidence to support pre-emptive thoracic endovascular aortic repair (TEVAR) intervention for uncomplicated type B aortic dissection (unTBAD). Pre-emptive intervention would be particularly beneficial in patients that have a higher baseline risk of progressing to complicated TBAD (coTBAD). There remains debate on the optimal clinical, laboratory, morphological and radiological parameters which would identify the highest-risk patients that would benefit most from pre-emptive TEVAR. **Aim:** This review summarises evidence on the clinical, laboratory, and morphological parameters that increase the risk profiles of unTBAD patients. **Methods:** A comprehensive literature search was carried out on multiple electronic databases including PubMed, EMBASE, Ovid and Scopus in order to collate all research evidence on the the clinical, laboratory, and morphological parameters that increase the risk profiles of unTBAD patients **Results:** At present, there are no clear clinical guidelines using risk-stratification to inform the selection of unTBAD patients for TEVAR. However, there are noticeable literature trends that can assist with the identification of the most at-risk unTBAD patients. Patients are at particular risk when they have refractory pain and/or hypertension, elevated C-reactive protein (CRP), larger aortic diameter and larger entry tears. These risks should be considered alongside factors that increase the procedural risk of TEVAR to create a well-balanced approach. Advances in biomarkers and imaging are likely to identify more pertinent parameters in future to optimise the development of balanced, risk-stratified treatment protocols. **Conclusion:** There are a variety of risk profiling parameters that can be used to identify the high-risk unTBAD patient, with novel biomarkers and imaging parameter emerging. Longer-term evidence verifying these parameters would be ideal. Further randomized controlled trials and multicentre registry analyses are also warranted to guide risk-stratified selection protocols.

Risk Profile Analysis of Uncomplicated Type B Aortic Dissection Patients Undergoing Thoracic Endovascular Aortic Repair: Laboratory & Radiographic Predictors

Alexander Geragotellis[1], Mohammed Al-Tawil[2], Matti Jubouri[3], Sven ZCP Tan[4], Ian Williams MD FRCS[5], Mohamad Bashir MD PhD MRCS[6]

1. University of Cape Town Medical School, Cape Town, South Africa.
2. Faculty of Medicine, Al-Quds University, Jerusalem, Palestine

3. Hull York Medical School, University of York, York, UK
4. Barts and The London School of Medicine and Dentistry, Queen Mary University of London, UK
5. Department of Vascular Surgery, University Hospital of Wales, Heath Park, Cardiff, UK
6. Vascular & Endovascular Surgery, Velindre University NHS Trust, Health Education & Improvement Wales (HEIW), Cardiff, UK

Correspondence:

Mohamad Bashir MD PhD MRCS

Vascular & Endovascular Surgery

Velindre University NHS Trust

Health Education & Improvement Wales (HEIW)

Cardiff

UK

Email: drmobashir@outlook.com

COI: None

Funding: No funding received.

Ethical approval: No ethical approval required for this review.

Contributorship: AG, MT and MJ involved in literature review design and writing. SZCPT, IW and MB involved in manuscript revision.

Acknowledgements: None

Abstract

Background: There is emerging evidence to support pre-emptive thoracic endovascular aortic repair (TEVAR) intervention for uncomplicated type B aortic dissection (unTBAD). Pre-emptive intervention would be particularly beneficial in patients that have a higher baseline risk of progressing to complicated TBAD (coTBAD). There remains debate on the optimal clinical, laboratory, morphological and radiological parameters which would identify the highest-risk patients that would benefit most from pre-emptive TEVAR.

Aim: This review summarises evidence on the clinical, laboratory, and morphological parameters that increase the risk profiles of unTBAD patients.

Methods: A comprehensive literature search was carried out on multiple electronic databases including PubMed, EMBASE, Ovid and Scopus in order to collate all research evidence on the the clinical, laboratory, and morphological parameters that increase the risk profiles of unTBAD patients

Results: At present, there are no clear clinical guidelines using risk-stratification to inform the selection of unTBAD patients for TEVAR. However, there are noticeable literature trends that can assist with the identification of the most at-risk unTBAD patients. Patients are at particular risk when they have refractory pain and/or hypertension, elevated C-reactive protein (CRP), larger aortic diameter and larger entry tears. These risks should be considered alongside factors that increase the procedural risk of TEVAR to create a well-balanced approach. Advances in biomarkers and imaging are likely to identify more pertinent parameters in future to optimise the development of balanced, risk-stratified treatment protocols.

Conclusion: There are a variety of risk profiling parameters that can be used to identify the high-risk unTBAD patient, with novel biomarkers and imaging parameter emerging. Longer-term evidence verifying these parameters would be ideal. Further randomized controlled trials and multicentre registry analyses are also warranted to guide risk-stratified selection protocols.

Keywords: Type B Aortic Dissection, Aortic Dissection, Aneurysm, Aortic Surgery

Introduction

Uncomplicated type B aortic dissection (unTBAD) is estimated to constitute 75% of type B aortic dissection (TBAD) cases, with up to 50% of those progressing to become complicated. Despite this, and even though thoracic endovascular aortic repair (TEVAR) has proven its safety and effectiveness in the treatment on unTBAD, it is still being managed conservatively with optimal medical therapy (OMT). However, with the surge of emerging evidence in the literature supporting the use of pre-emptive TEVAR in unTBAD, the paradigm of clinical practice is shifting (1). Yet, there seems to be a strong debate on the optimal clinical, laboratory, morphological and radiological parameters defining the intervention threshold in clinical guidelines.

This review will discuss current literature pertaining to the risk factors in unTBAD patients undergoing TEVAR, with a focus on clinical, laboratory and morphological/radiographic features that increase the risk profiles of unTBAD patients. In the absence of clear clinical guidelines, a stratification scheme to identify and managed more vulnerable acute unTBAD patients with TEVAR is presented which could be executed using the risk factors identified in this review.

Clinical parameters

One of the most important clinical parameters to control in unTBAD is the heart rate, as when this exceeds 60 bpm in unTBAD patients it could lead to poorer long-term outcomes. Kodama et. al (2) observed lower rates of aortic events in OMT managed unTBAD patients with heart rate controlled at < 60 bpm (OR 0.25, $p = 0.0059$) (2). Surgical intervention was also lower at < 60 bpm versus > 60 bpm (0% versus 18.7%, $P = 0.005$) (2). The patient's perception of pain also warrants caution. It is imperative to be vigilant of rarer absences of chest pain on admission, as this has been significantly associated with increased in-hospital mortality (OR 3.49, $p = 0.01$) (3). Even after OMT management, recurrent and/or refractory pain or refractory hypertension must be carefully observed. These parameters appear to elevate the risk of in-hospital mortality (17.4% versus 4%, $p = 0.0003$), and multivariate analysis showed are significant predictors of in-hospital mortality (OR 3.31, 95% CI 1.04 - 10.45, $p = 0.041$) (3).

Laboratory parameters

Certain blood tests are likely to have predictive and non-predictive values for aortic enlargement, and these will only be confirmed in studies that feature longer-term follow-ups. C-Reactive Protein (CRP) elevation is common in patients with TBAD and usually peaks between days 3-6 after the index event. Peak CRP, which needs to be obtained more than for reliable risk assessment (4), has been strongly linked to long-term adverse events in patients with unTBAD and could therefore be used as a prognostic marker. In a comparative study, a mean CRP levels of 12.0 mg/dL (with a lower limit of 9.61 mg/dL) and 19.5 mg/dL (with a lower limit of 14.90 mg/dL) were shown to be strong predictors of adverse events (HR 3.25, $p = 0.01$) (HR 6.02, $p = 0.0001$). This has been corroborated by others who demonstrate that CRP levels > 15.0 mg/dL may independently predict aortic events (OR 4.199, 95% CI 0.76 - 23.19, $p = 0.1$) (5). Peak CRP has been identified as a significant predictor of lower PaO2/FiO2 ratio which usually necessitates mechanical ventilation and is associated with further complications (6). However, as CRP is a non-specific marker, its utility in predicting the extent of aortic dissection may be limited by confounding inflammatory processes.

Fibrin degradation products (FDPs) may also be of prognostic value and appear to share a relationship with the patency and thrombotic status of the false lumen (FL) (7). The status of the FL is a known mortality determinant (8). Tsai et. al (8) previously found ± 3-year mean (± SD) mortality rates to be greatest in patients with partial FL thrombosis (31.6 ± 12.4 %) than those with patent (13.7 ± 7.1 %) or completely thrombosed FL (22.6 ± 22.6 %, $p = 0.003$). Nagaoka et. al (7) then found that FDP levels were significantly higher in patients with partial FL thrombosis (35.8 ± 43.2, median 18.8 μg/mL) versus those with patent or completely thrombosed FL (14.0 ± 21.3, median 5.5 μg/mL, $p = 0.01$). FDP may predict 1-year aortic events when admission levels are [?] 20 μg/mL (OR 7.8, 95% CI 1.4 - 43.3), and can further

assist radiological findings in the recognition of FL partial thrombosis (5, 7). Aortic enlargement, however, was not associated with platelet count, D-dimer, and thrombin-antithrombin III complex (5). The follow-up interval in this study (5) was only 1 year and therefore long-term significance remains elusive. FDP is also not easily measured in emergency settings and may therefore be less accessible for some institutions.

Troponin could be also used as a laboratory parameter in unTBAD by approximating the extent of ischaemic myocardial injury. Demand-ischaemia can occur due to blood pressure fluctuations, aortic regurgitation or an intimal flap covering the coronary ostia. Elevated troponin levels have been shown to be significantly associated with a higher risk for short-term mortality (OR 2.57, 95% CI 1.66 - 3.96) (9).

Morphological and radiologic parameters

Anatomic risk-stratification is currently the mainstay of TBAD assessment. Many morphological features have been noted that may lead to further aortic degeneration and secondary emergencies, including aortic morphology and diameter (10-14), position and size of entry tear (15, 16), and FL diameter and patency (12-14, 17, 18).

Aortic diameter

Aortic diameter has been reported as the most sensitive independent risk factor (HR 8.6, $p < 0.01$) for mortality in unTBAD (19). Strikingly, intervention rates at 1-, 5-, and 10- years in patients with an aortic diameter > 44mm were 18.8%, 29.5% and 50.3% versus 4.8%, 13.3% and 13.3% in those < 44mm ($p < 0.01$). Overall survival is illustrated in **Figure 1** (19). Others have also described aortic diameter > 40 mm as predictor for late aortic events (HR 3.18, 95% CI 2.12 to 5.05, $p < 0.001$) (12) and > 40 mm is generally regarded as the cut-off for predicting late aortic intervention (15, 20).

The morphology of aortic diameter is also dictated by interplaying pressure relationships between the TL and FL. Clearly demarcated circular FL configurations are more highly pressurised due to their blood volumes. The fragility of the vessel wall in these "fusiform"- shaped aortas likely contributes to dilatation (12). Some have suggested that a circular FL may cause an elliptic TL configuration that has been associated with adverse aortic growth based on results from a conservatively managed cohort ($n = 62$) (21). Similarly, Song et. al (22) found increased FL: total aortic diameter ratios in the proximal and descending thoracic aortas of patients showing aortic growth during their follow-up. Radiology metrics have been proposed that could guide quantification. Marui et. al (12) suggested that a fusiform index of 0.64 could be an independent predictor of adverse aortic events. Briefly, the radiologic formula is defined as A / (B+C) where A is the maximum diameter of the proximal descending aorta, B is the diameter of the distal aortic arch, and C is the diameter of the descending aorta at the anatomical level of the main pulmonary artery.

It is likely that advancing MRI techniques will improve understanding of aortic diameter changes. Sailer et. al (23) devised the "FL circumferential extent" (FL-CE) metric which, unlike the conventional metrics of FL diameter and volumes, is unaffected by the position of the aortic dissection membrane. The FL-CE itself is defined as the angular distance between the two FL insertion points in the intimal flap - an angle more than 249° was found to be an independent predictor of adverse aortic events.

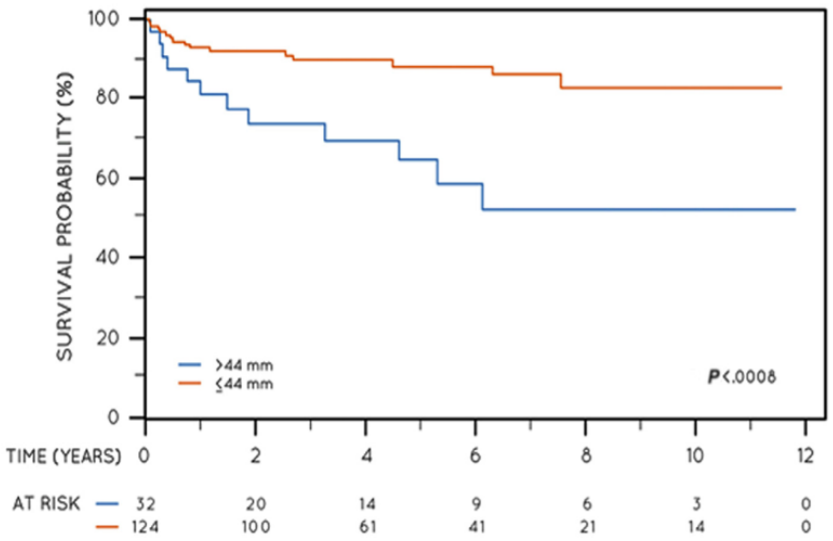

Figure 1. Kaplan-Meier curve for overall survival of 156 unTBAD patients retrospectively reviewed between 2000-2014, shown for total aortic diameter. Reproduced from Ray et. al (19) with permission from Elsevier.

4.2 Entry tear

Entry tears are to be considered based on their size, whether they are located at the outer (convexity) or inner (concavity) circumference of the distal aortic arch, and their proximity to the LSA. Entry tears >10mm have been shown to have a greater aortic growth rate and incidence of dissection-related complications (HR = 5.8) than patients with an entry tear of < 10mm (16). Intuitively, larger entry tears mean more blood flow in the FL (discussed later) which is a well-documented predictor of wall growth (12-14, 18, 24, 25). A single entry tear produces a more pressurised avenue than multiple tears for blood which accelerates aortic growth (21). Specifically, patients with one entry tear showed an aortic growth rate of 5.6 ± 8.9mm/year versus those with two tears (2.1± 1.7 mm/year) and three tears (2.2 ± 4.1 mm/year) (21). The actual mechanism for this finding is misunderstood, although a haemodynamic ex-vivo study suggested that inner tears (IT) (**Fig. 2A**) cause significant elevation of the diastolic pressure (DP) in the FL (26).

Greater FL volumes are more likely to retrogradely propagate and manifest as acute coTBAD. Literature suggests that this manifestation is more common with ITs (**Fig. 2A**) than outer tears (OT) (**Fig. 2B**) (21, 26-29). Loewe et. al (27) reported 61% (IT; n = 23) vs 21% (OT; n = 42) incidences of primary coTBAD (p = 0.003). Tolenaar et. al (21) similarly compared differences in aortic growth rates. Growth rates in the IT group (n = 53) was 4.6 ± 9.6 (SD) mm/yr versus 2.9 ± 5.1 mm/yr in the OT group (n = 187) (21). Nonetheless, OTs appear to be of particular risk when within 5cm of the LSA based on the likelihood of forming an additional false lumen (30). Patients with 1 entry tear located within 5 cm of the LSA showed significantly more growth than their counterparts (5.8 ± 7.7 versus 2.5 ± 2.7 mm/yr; p = 0.003) (30). LSA coverage is a significant independent risk factor for dissection related death (HR = 5.6) (25) and more recently, for failing OMT (16, 31). Codner et. al (31) showed that aortic growth (n = 72) versus non-aortic growth (n = 49) patients had their median entry tears respectively located 27mm [9 to 66 mm] versus 77mm [26 to 144 mm] from the LSA; 53% of the growth group required open surgical repair/TEVAR vs 0% in the non-growth group (31).

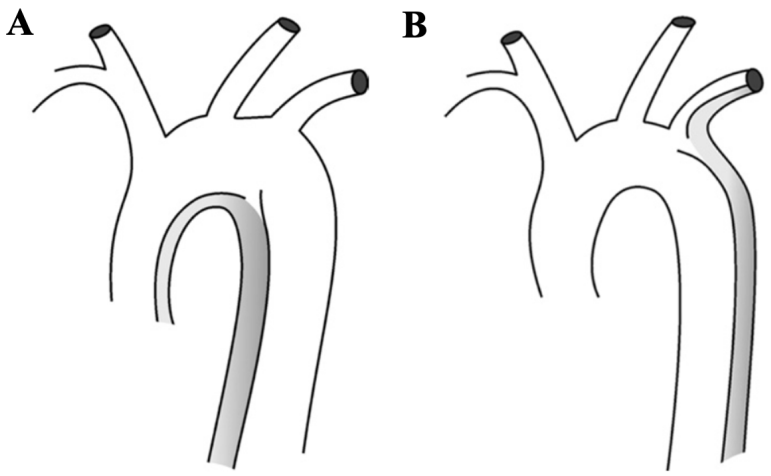

Figure 2. Main classification of the acute TBAD primary entry tear positions. Inner (concave) (**A**) primary entry tears (IT) occur at the inner circumference of the distal aortic arch and are unopposed to retrograde sequelae in the aortic arch and ascending aorta. Outer (convex) (**B**) primary entry tears (OT) occur at the outer circumference of the distal aortic arch and are opposed to retrograde sequelae by the left subclavian artery. Adapted and reproduced from Loewe et. al (27) with permission from Elsevier.

4.3 False Lumen

Based on the proven risk of complications and death associated with the position of the entry tear, FL position is also an important consideration alongside it's diameter, configuration, and whether it is thrombosed. A FL diameter of 22mm at the upper descending thoracic aorta is a significant ($p < 0.001$) independent risk for aneurysmal change and subsequent death. Furthermore, it is able to predict late aneurysm formation with 100% sensitivity and 76% specificity (22), although some studies have found it to be an insignificant marker of mortality (**Figure 3**) (19). Further growth of the FL is more likely in cases where there are many patent FL segments, which are typically found in younger patients with longer segments of the dissected aorta (32). A patent FL inflicts hemodynamic stress onto the aortic wall, which is likely to cause progressive growth of the segment. False lumen patency is therefore widely accepted as an independent predictor of aortic growth (12-14, 17, 18), and there appear to be corroborating findings which suggest that aortic growth is reduced when the FL is completely thrombosed FL (14, 25). Partial thrombosis is predictive of aortic growth (21, 33) and post-discharge mortality (outlined earlier in the context of FDPs) (8).

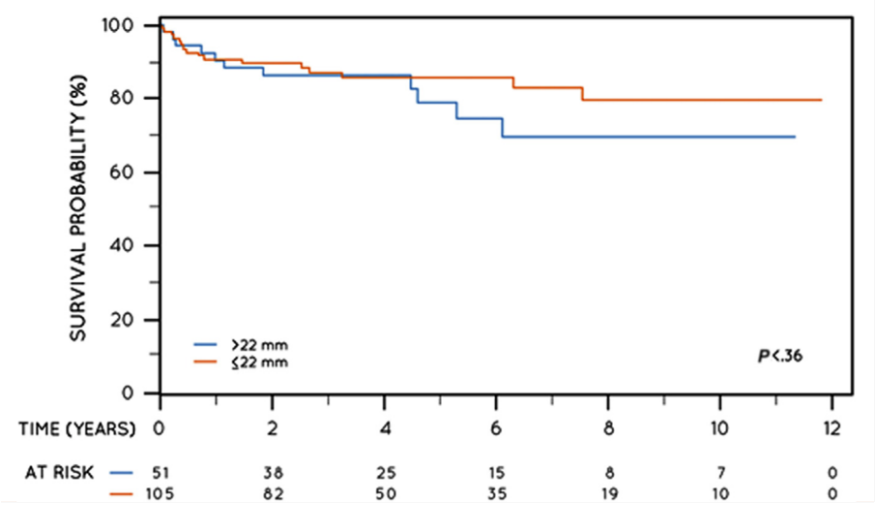

Figure 3. Kaplan-Meier curve for overall survival of 156 unTBAD patients retrospectively reviewed between 2000-2014, shown for false lumen diameter. Reproduced from Ray et. al (19) with permission from Elsevier.

Risk stratification

5.1. TEVAR risks and risk mitigation

TEVAR remains an imperfect procedure. Early survival in unTBAD patients receiving OMT is ± 90% (34, 35) which is appreciably lower than patients managed with TEVAR (36-40). Nonetheless, 30-40% (36, 41, 42) of surviving patients will experience aneurysmal dilatation or dissection progression despite being under cautious clinical surveillance which could mandate downstream TEVAR intervention. TEVAR is associated with moderate risk of ischemic stroke and spinal cord ischemia (SCI) / paraplegia even when preventative actions (e.g., CSF drainage) are taken (43, 44). A recent multicentre study found an incidence of SCI post-TEVAR is ± 3.6% with ± 60% being permanent (45). In addition to CSF drainage (46), other potentially more effective strategies to reduce paraplegia risk include permissive hypertension and left subclavian re-vascularization (47). Further trials using these strategies to characterise TEVAR outcomes are warranted. Accordingly, a list of significant SCI-predicting factors (45) is shown in**Table 1** .

The aetiology and management of TEVAR-related stroke is still not fully understood and further evidence can add value to the management approach. The MOTHER registry reports a higher incidence of TEVAR-related strokes in procedures where endovascular stent extends to cover the left subclavian artery (LSA), which usually manifests as posterior territory strokes and may cause left upper limb ischemia (48, 49). However, other aetiologies may be present due to the increased embolic load during the procedure which usually leads to middle cerebral artery territory strokes (50). Nonetheless, a recent observational study showed non-significant association between LSA revascularization and SCI ($p = 0.58$) or stroke ($p = 0.37$) incidence in patients undergoing TEVAR (51).

Other less commonly reported post-procedural complications include acute myocardial infarction, left upper limb ischemia, acute kidney injury, malperfusion, access-related pseudoaneurysm, or groin hematoma (38, 51). Aorta-specific TEVAR-related complications including retrograde type A aortic dissection (RTAD) and stent graft-induced new entry (SINE) are serious TEVAR-related complications. RTAD has an incidence of 2.5% and a mortality rate of up to 37.1%. (52). The incidence of RTAD is higher in patients undergoing acute procedures, whereas SINE occurs with higher incidence in chronic procedures. Incidence of these TEVAR-related complications and associated mortality can be reduced with using more appropriately sized stents and better developed aortic facilities (53, 54). Other aorta specific post-TEVAR complications include rupture, endoleaks, or stent failure, which are usually managed by reinterventions (55). Therefore, pre-procedural risk identification, interventional expertise, choice of appropriate stents, and the availability of well-developed

aortic centres are necessary factors when offering early TEVAR for patients with unTBAD to achieve optimal outcomes.

Table 1 . Predictor of increased risk for spinal cord ischemia for TEVAR procedures reported by Mousa et. al (45).

Predictor of increased risk	Hazard Ratio [95% CI]
Age by decade	1.2 [1.0-1.4]
Current smoker	1.6 [1.1-2.2]
Emergent or urgent surgery	1.5 [1.1-2.1]
Adjunct aorta-related procedure	2.5 [1.6-3.8]
Adjunct procedure not aortic related	2.6 [1.4-4.9]
ASA Class $>$ 3	1.6 [1.1-2.3]
Total estimated length of the aortic device	(19-31 cm, HR 1.9) ([?] 32 cm, HR 3.0)
Procedure time [?] 154 minutes	1.8 [1.3-2.5]
eGFR [?] 60 * High-volume centres *	0.6 [0.4-0.8]
CI; Confidence Interval, ASA; American Society of Anaesthesiologists, eGFR; estimated Glomerular Filtration Rate, *Associated with lower risk of SCI	CI; Confidence Interval, ASA; American Society of Anaesthesiologists, eGFR; estimated Glomerular Filtration Rate, *Associated with lower risk of SCI

5.2. Proposed management of unTBAD patients

TEVAR is the treatment of choice in coTBAD (ESC guidelines: class I, level of evidence C) (46) for patients with appropriate anatomy (46, 56, 57). It is most effective for aortic remodelling unTBAD in the acute and subacute stages (1), and is also recognised as a treatment to prevent future aortic complications in unTBAD (ESC guidelines: class IIA, level of evidence B) (46). Therefore, the most pertinent risk factors for unTBAD progression to coTBAD should inform candidate selection for TEVAR. At present, there are no clear clinical guidelines using risk-stratification to inform the selection of unTBAD patients for TEVAR. A recent systematic review and meta-analysis (58) has also highlighted that reporting of false lumen status, aortic diameters and growth, and demographic data has not always been congruent with the most recent recommendations published by the Society for Vascular Surgery and Society of Thoracic Surgeons (59). Given that these recommendations are adhered to in future, discussion on optimal unTBAD patient selection for TEVAR will become increasingly granular and allow for the formulation of strong evidence-based guidelines.

In the interim, we reason that the risks quantified in **Tables 2 & 3** could inform the execution of the treatment algorithm shown in**Figure 4** . It is imperative to focus on high-risk imaging features identified in **Table 3** (e.g., primary entry tear diameter $>$10 mm, initial total aortic diameter [?] 40 mm, false lumen [?] 22 mm, patent false lumen) which are likely to indicate unstable disease in apparently clinically stable patients (36, 42, 60). If the imaging features are present, demographic, clinical and laboratory parameters (**Table 2**) could then be considered.**Figure 4** outlines a pragmatic approach towards TEVAR intervention given its procedural risks and requirement for specialist aortic care; thorough patient imaging could guide the selection of patients at high risk of disease progression for deferred endovascular treatment within the 14 to 90 day subacute phase which appears to be the optimal TEVAR intervention time (1). Moreover, specific subsets of acute unTBAD patients that are at high risk of developing downstream aortic complications may qualify for early prophylactic endovascular therapy.

Table 2. Clinical and laboratory parameters in unTBAD patients that could be used to predict need for intervention. HR: Hazard ratio; OR: Odds Ratio; FDP: Fibrin degradation products.

Category	**Predictive feature, with parameter change (where applicable)**	**Predictive feature, with parameter change (where applicable)**	**Magnitude of Risk**	**[95% CI]**	**P value(s)**	**References**
Clinical	Heart rate > 60 beats per minute	Heart rate > 60 beats per minute	HR, 0.25 ++	[0.08 to 0.77]	0.01	(2)
	Refractory pain	Refractory pain	HR, 3.31 *	[1.04 to 10.45]	0.041	(3)
	Refractory hypertension	Refractory hypertension				
Laboratory	Peak CRP level (mg/dL)	> 9.6	HR, 3.25 ***	[1.37 to 7.71]	0.01	(4)
		> 14.9	HR, 6.02 ***	[2.44 to 14.87]	0.0001	
	FDPs	FDPs	OR, 7.8 ¥	[1.4 to 43.3]	—-	(5)
	Elevated cardiac troponin	Elevated cardiac troponin	OR, 2.57 **	[1.66 to 3.96]	—-	(9)
Predictive of in-hospital mortality; **	* Predictive of in-hospital mortality; **	* Predictive of in-hospital mortality; **	* Predictive of in-hospital mortality; **	* Predictive of in-hospital mortality; **	* Predictive of in-hospital mortality; **	* Predictive of in-hospital mortality; **
Predictive of post-discharge mortality; ***	Predictive of post-discharge mortality; ***	Predictive of post-discharge mortality; ***	Predictive of post-discharge mortality; ***	Predictive of post-discharge mortality; ***	Predictive of post-discharge mortality; ***	Predictive of post-discharge mortality; ***
Predictive of adverse events; ++	Predictive of adverse events; ++	Predictive of adverse events; ++	Predictive of adverse events; ++	Predictive of adverse events; ++	Predictive of adverse events; ++	Predictive of adverse events; ++
Predictive of dissection-related event; ¥	Predictive of dissection-related event; ¥	Predictive of dissection-related event; ¥	Predictive of dissection-related event; ¥	Predictive of dissection-related event; ¥	Predictive of dissection-related event; ¥	Predictive of dissection-related event; ¥
Predictive of aortic intervention at > 6 months.	Predictive of aortic intervention at > 6 months.	Predictive of aortic intervention at > 6 months.	Predictive of aortic intervention at > 6 months.	Predictive of aortic intervention at > 6 months.	Predictive of aortic intervention at > 6 months.	Predictive of aortic intervention at > 6 months.

Table 3 . Morphological / radiographic parameters in unTBAD patients that could be used to predict need for intervention. HR: Hazard ratio; OR: Odds ratio; RR: Relative risk; FL: False lumen; OT: Outer tear; LSA: Left subclavian artery.

Category	Predictive feature, with parameter change (where applicable)	Magnitude of risk	[95% CI]	P value(s)	References
Tear-related	Large entry tear located in the proximal part of the dissection	OR, 2.1 ¥	[1.5 to 3.8]	0.03	(15)
		HR, 5.8 ++	[3.3 to 10]	< 0.001	(16)
	Single entry tear	—-	—-	—-	(21, 30)
	False lumen/intimal tear located in the inner aortic curvature	HR, 1.8 §	[1.0 to 3.2]	—-	(27)
	OTs within 5cm of the LSA	—-	—-	—-	(25)
Aortic anatomy related	Aortic diameter during acute phase	HR, 3.18 ¥	[2.12 to 5.05]	< 0.001	(12)
		HR, 8.6 (> 44 mm) **	—-	< 0.01	18
	Circular-shaped TL (vs elliptic)	HR, -2.83 ¶	[-5.35 to -0.321]	0.027	(21, 22)
	Fusiform index	HR, 2.73 ¥	[1.85 to 4.60]	0.13	(12)
False lumen related	Proximal descending thoracic aorta FL diameter ≥ 22 mm on initial imaging	—-	—-	—-	(22)
	Patency	HR, 2.59 + HR, 1.87 ++	[1.01 to 6.12] [1.12 to 3.13]	—- —-	(14)
		HR, 5.6 + HR, 7.6 ++	[1.1 to 28] [2.7 to 22]	0.038 0	(24)
	Partial thrombosis	RR, 2.69 **	[1.45 to 4.98]	0.002	(8)
	FL circumferential extent (FL-CE)	HR, 1.03 ++ per degree	[1.01 to 1.04]	0.003	(23)

Category	Predictive feature, with parameter change (where applicable)	Magnitude of risk	[95% CI]	P value(s)	References
	Vessels originating from the false lumen	HR, 22.1 ¤	[1.01 to 481.5]	0.049	(32)
	Initial large thickness	HR, 1.50 ***	[1.15 to 1.95]	< 0.001	(61)
	Saccular false lumen	—-	[2.07 to 7.81] ¶¶	0.001	(21)

Category	**Predictive feature, with parameter change (where applicable)**	**Magnitude of risk**	**[95% CI]**	**P value(s)**	**References**
Predictive of in-hospital mortality; ** Predictive of post-discharge mortality; *** Predictive of adverse events; + Predictive of dissection-related death; ++ Predictive of dissection-related event; § Predictive of primary or secondary development of acute coTBAD; ¥ Predictive of aortic intervention at > 6 months; ¤ Predictive of false lumen growth; ¶ Predictive of significantly decreased aortic growth rate; ¶¶ Predictive of significantly increased aortic growth rate.	* Predictive of in-hospital mortality; ** Predictive of post-discharge mortality; *** Predictive of adverse events; + Predictive of dissection-related death; ++ Predictive of dissection-related event; § Predictive of primary or secondary development of acute coTBAD; ¥ Predictive of aortic intervention at > 6 months; ¤ Predictive of false lumen growth; ¶ Predictive of significantly decreased aortic growth rate; ¶¶ Predictive of significantly increased aortic growth rate.	* Predictive of in-hospital mortality; ** Predictive of post-discharge mortality; *** Predictive of adverse events; + Predictive of dissection-related death; ++ Predictive of dissection-related event; § Predictive of primary or secondary development of acute coTBAD; ¥ Predictive of aortic intervention at > 6 months; ¤ Predictive of false lumen growth; ¶ Predictive of significantly decreased aortic growth rate; ¶¶ Predictive of significantly increased aortic growth rate.	* Predictive of in-hospital mortality; ** Predictive of post-discharge mortality; *** Predictive of adverse events; + Predictive of dissection-related death; ++ Predictive of dissection-related event; § Predictive of primary or secondary development of acute coTBAD; ¥ Predictive of aortic intervention at > 6 months; ¤ Predictive of false lumen growth; ¶ Predictive of significantly decreased aortic growth rate; ¶¶ Predictive of significantly increased aortic growth rate.	* Predictive of in-hospital mortality; ** Predictive of post-discharge mortality; *** Predictive of adverse events; + Predictive of dissection-related death; ++ Predictive of dissection-related event; § Predictive of primary or secondary development of acute coTBAD; ¥ Predictive of aortic intervention at > 6 months; ¤ Predictive of false lumen growth; ¶ Predictive of significantly decreased aortic growth rate; ¶¶ Predictive of significantly increased aortic growth rate.	* Predictive of in-hospital mortality; ** Predictive of post-discharge mortality; *** Predictive of adverse events; + Predictive of dissection-related death; ++ Predictive of dissection-related event; § Predictive of primary or secondary development of acute coTBAD; ¥ Predictive of aortic intervention at > 6 months; ¤ Predictive of false lumen growth; ¶ Predictive of significantly decreased aortic growth rate; ¶¶ Predictive of significantly increased aortic growth rate.

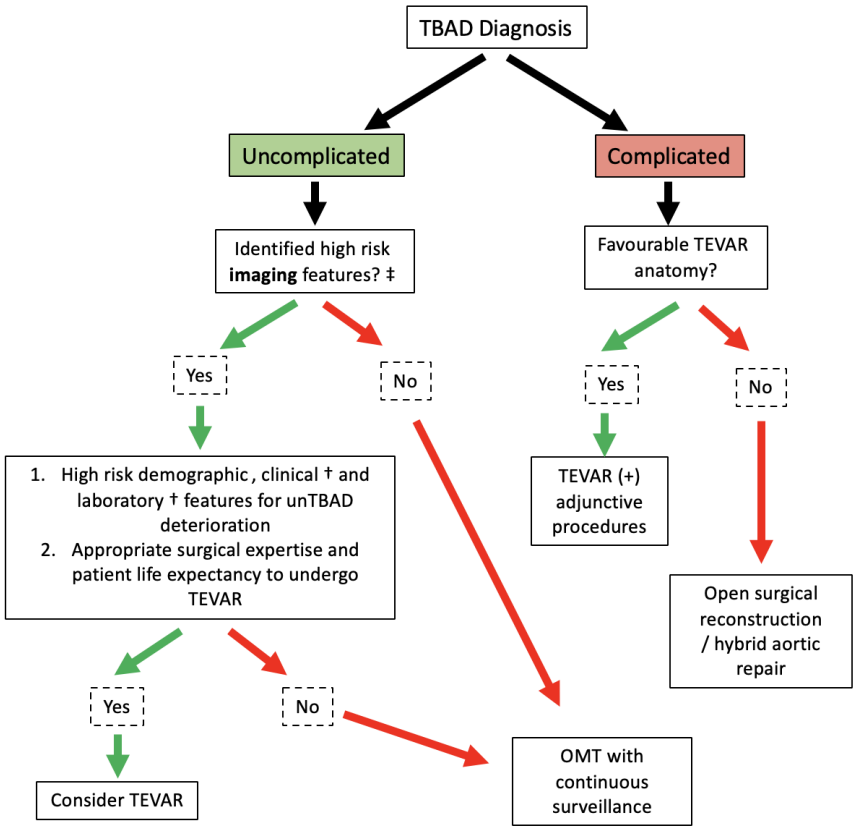

Figure 4. Possible TBAD treatment selection algorithm. Similarly reported by Tadros et. al (60). + Based on data summarised in**Table 2** ; ++ Based on data summarised in **Table 3** .

Future directions

Next-generation biomarkers

Specific biomarkers for tissue inflammation, matrix degeneration, and fibrinolysis are increasingly encroaching into the clinical realm to better inform current predictors of unTBAD deterioration. There seems to be growing evidence for a role for these novel serologic markers in predicting the development of thoracic aortic aneurysm – many of these are elusive based on small sample studies although CRP and TGF-β appear to be of value (62). More recently, immunological metrics - such as neutrophil-lymphocyte ratio (NLR) (63), monocyte to high-density lipoprotein ratio (64, 65), and long ncRNAs (e.g. LUCAT1) (66) - have all been suggested and further study in these areas is warranted.

Imaging advances

There are known limitations to measuring the diameters of true and false lumens as a means of characterising aortic remodelling and predicting aortic enlargement. The index CT scans could be volumetrically analysed and the ratio of true lumen volume (TLV) to false lumen volume (FLV) used to predict eventual aortic intervention (67). TLV/FLV < 0.8 (OR $= 12.2$; 95% CI 5-26; $p < 0.001$) is reportedly highly predictive of aortic intervention whereas > 1.6 is highly predictive of freedom from aortic intervention (67). Incorporating personalized hemodynamic parameters in patient risk stratification could also offer prognostic value. 4D-MRI could detect flow patterns in both lumens and the entry tear, and recent clinical guidelines suggest that these non-invasive methods can be used to deliver additional quantitative information about the flow in the entry tear, and on whether or not arterial vessels are involved (56, 68, 69). 4D-MRI imaging has

demonstrated that flow velocity, configuration (e.g., helical), and stroke volume are related to the rate of aortic expansion (70, 71). Since it is challenging to accurately standardize these parameters between patients based on differential aortic morphologies, patient-specific approaches must be used to utilize 4D-MRI for both predicting aortic expansion and as a tool for selecting patients who may require additional procedures based on their clinical conditions (e.g. organ malperfusion) (72, 73). Other innovations such as multidetector CT, high-resolution ultrasound including color-coded Doppler, and contrast-enhanced B-mode imaging have also been reported that may be used to enhance aortic care (74).

Conclusions

TEVAR has enriched the surgical armamentarium to treat acute and subacute TBAD and has reached maturation as a baseline strategy in many patients. A specific subset of acute unTBAD patients at greater risk for developing aortic complications should be strongly considered for timely prophylactic TEVAR intervention. There are a variety of demographic, clinical, laboratory and radiographic/morphological risk factors to consider when selecting these patients. To guide selection, the principles of the treatment scheme presented in this review could be used. The scheme could be refined in future as the reporting of risk quantification ratios across the literature becomes more consistent, and advances in biochemistry and imaging techniques progressively enter the clinical arena. Selection of high-risk patients should also be further informed with randomized controlled trials and multicentre registry analyses.

References

1. Jubouri M, Bashir M, Tan S, Bailey D, Anderson R, Nienaber CA, et al. What is the optimal timing for thoracic endovascular aortic repair in uncomplicated Type B aortic dissection? J Card Surg. 2022;37(4):993-1001.

2. Kodama K, Nishigami K, Sakamoto T, Sawamura T, Hirayama T, Misumi H, et al. Tight heart rate control reduces secondary adverse events in patients with type B acute aortic dissection. Circulation. 2008;118(14 Suppl):S167-70.

3. Trimarchi S, Eagle KA, Nienaber CA, Pyeritz R, Jonker FH, Suzuki T, et al. Importance of refractory pain and hypertension in acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). Circulation. 2010;122(13):1283-9.

4. Sakakura K, Kubo N, Ako J, Wada H, Fujiwara N, Funayama H, et al. Peak C-reactive protein level predicts long-term outcomes in type B acute aortic dissection. Hypertension. 2010;55(2):422-9.

5. Kitada S, Akutsu K, Tamori Y, Yoshimuta T, Hashimoto H, Takeshita S. Usefulness of Fibrinogen/Fibrin Degradation Product to Predict Poor One-Year Outcome of Medically Treated Patients With Acute Type B Aortic Dissection. The American Journal of Cardiology. 2008;101(9):1341-4.

6. Kashiwagi Y, Komukai K, Suzuki K, Oi Y, Tominaga M, Nakata K, et al. Predictors of oxygenation impairment in medical treatment for type B acute aortic dissection. Heart and vessels. 2018;33(12):1463-70.

7. Nagaoka K, Sadamatsu K, Yamawaki T, Shikada T, Sagara S, Ohe K, et al. Fibrinogen/fibrin degradation products in acute aortic dissection. Intern Med. 2010;49(18):1943-7.

8. Tsai TT, Evangelista A, Nienaber CA, Myrmel T, Meinhardt G, Cooper JV, et al. Partial Thrombosis of the False Lumen in Patients with Acute Type B Aortic Dissection. New England Journal of Medicine. 2007;357(4):349-59.

9. Vrsalovic M. Prognostic effect of cardiac troponin elevation in acute aortic dissection: A meta-analysis. International Journal of Cardiology. 2016;214:277-8.

10. Winnerkvist A, Lockowandt U, Rasmussen E, Rådegran K. A Prospective Study of Medically Treated Acute Type B Aortic Dissection. European Journal of Vascular and Endovascular Surgery. 2006;32(4):349-55.

11. Kato M, Bai H-z, Sato K, Kawamoto S, Kaneko M, Ueda T, et al. Determining Surgical Indications for Acute Type B Dissection Based on Enlargement of Aortic Diameter During the Chronic Phase. Circulation. 1995;92(9):107-12.

12. Marui A, Mochizuki T, Koyama T, Mitsui N. Degree of fusiform dilatation of the proximal descending aorta in type B acute aortic dissection can predict late aortic events. J Thorac Cardiovasc Surg. 2007;134(5):1163-70.

13. Marui A, Mochizuki T, Mitsui N, Koyama T, Kimura F, Horibe M. Toward the best treatment for uncomplicated patients with type B acute aortic dissection: A consideration for sound surgical indication. Circulation. 1999;100(19 Suppl):Ii275-80.

14. Kunishige H, Myojin K, Ishibashi Y, Ishii K, Kawasaki M, Oka J. Predictors of surgical indications for acute type B aortic dissection based on enlargement of aortic diameter during the chronic phase. Jpn J Thorac Cardiovasc Surg. 2006;54(11):477-82.

15. Schwartz SI, Durham C, Clouse WD, Patel VI, Lancaster RT, Cambria RP, et al. Predictors of late aortic intervention in patients with medically treated type B aortic dissection. Journal of Vascular Surgery. 2018;67(1):78-84.

16. Evangelista A, Salas A, Ribera A, Ferreira-González I, Cuellar H, Pineda V, et al. Long-term outcome of aortic dissection with patent false lumen: Predictive role of entry tear size and location. Circulation. 2012;125(25):3133-41.

17. Erbel R, Oelert H, Meyer J, Puth M, Mohr-Katoly S, Hausmann D, et al. Effect of medical and surgical therapy on aortic dissection evaluated by transesophageal echocardiography. Implications for prognosis and therapy. The European Cooperative Study Group on Echocardiography. Circulation. 1993;87(5):1604-15.

18. Onitsuka S, Akashi H, Tayama K, Okazaki T, Ishihara K, Hiromatsu S, et al. Long-term outcome and prognostic predictors of medically treated acute type B aortic dissections. Ann Thorac Surg. 2004;78(4):1268-73.

19. Ray HM, Durham CA, Ocazionez D, Charlton-Ouw KM, Estrera AL, Miller CC, III, et al. Predictors of intervention and mortality in patients with uncomplicated acute type B aortic dissection. Journal of Vascular Surgery. 2016;64(6):1560-8.

20. Trimarchi S, Jonker FHW, van Bogerijen GHW, Tolenaar JL, Moll FL, Czerny M, et al. Predicting aortic enlargement in type B aortic dissection. Annals of cardiothoracic surgery. 2014;3(3):285-91.

21. Tolenaar JL, van Keulen JW, Jonker FHW, van Herwaarden JA, Verhagen HJ, Moll FL, et al. Morphologic predictors of aortic dilatation in type B aortic dissection. Journal of Vascular Surgery. 2013;58(5):1220-5.

22. Song JM, Kim SD, Kim JH, Kim MJ, Kang DH, Seo JB, et al. Long-term predictors of descending aorta aneurysmal change in patients with aortic dissection. J Am Coll Cardiol. 2007;50(8):799-804.

23. Sailer AM, van Kuijk SM, Nelemans PJ, Chin AS, Kino A, Huininga M, et al. Computed Tomography Imaging Features in Acute Uncomplicated Stanford Type-B Aortic Dissection Predict Late Adverse Events. Circ Cardiovasc Imaging. 2017;10(4).

24. Akutsu K, Nejima J, Kiuchi K, Sasaki K, Ochi M, Tanaka K, et al. Effects of the patent false lumen on the long-term outcome of type B acute aortic dissection. Eur J Cardiothorac Surg. 2004;26(2):359-66.

25. Sueyoshi E, Sakamoto I, Hayashi K, Yamaguchi T, Imada T. Growth rate of aortic diameter in patients with type B aortic dissection during the chronic phase. Circulation. 2004;110(11 Suppl 1):Ii256-61.

26. Tsai TT, Schlicht MS, Khanafer K, Bull JL, Valassis DT, Williams DM, et al. Tear size and location impacts false lumen pressure in an ex vivo model of chronic type B aortic dissection. Journal of Vascular Surgery. 2008;47(4):844-51.

27. Loewe C, Czerny M, Sodeck GH, Ta J, Schoder M, Funovics M, et al. A New Mechanism by Which an Acute Type B Aortic Dissection Is Primarily Complicated, Becomes Complicated, or Remains Uncomplicated. The Annals of Thoracic Surgery. 2012;93(4):1215-22.

28. Kitamura T, Torii S, Oka N, Horai T, Nakashima K, Itatani K, et al. Key success factors for thoracic endovascular aortic repair for non-acute Stanford type B aortic dissection+. European Journal of Cardio-Thoracic Surgery. 2014;46(3):432-7.

29. Weiss G, Wolner I, Folkmann S, Sodeck G, Schmidli J, Grabenwoger M, et al. The location of the primary entry tear in acute type B aortic dissection affects early outcome. Eur J Cardiothorac Surg. 2012;42(3):571-6.

30. Tolenaar JL, van Keulen JW, Trimarchi S, Jonker FH, van Herwaarden JA, Verhagen HJ, et al. Number of entry tears is associated with aortic growth in type B dissections. Ann Thorac Surg. 2013;96(1):39-42.

31. Codner JA, Lou X, Duwayri YM, Chen EP, Binongo JN, Moon R, et al. The distance of the primary intimal tear from the left subclavian artery predicts aortic growth in uncomplicated type B aortic dissection. J Vasc Surg. 2019;69(3):692-700.

32. Kamman AV, Brunkwall J, Verhoeven EL, Heijmen RH, Trimarchi S. Predictors of aortic growth in uncomplicated type B aortic dissection from the Acute Dissection Stent Grafting or Best Medical Treatment (ADSORB) database. J Vasc Surg. 2017;65(4):964-71.e3.

33. Trimarchi S, Tolenaar JL, Jonker FH, Murray B, Tsai TT, Eagle KA, et al. Importance of false lumen thrombosis in type B aortic dissection prognosis. J Thorac Cardiovasc Surg. 2013;145(3 Suppl):S208-12.

34. Suzuki T, Mehta RH, Ince H, Nagai R, Sakomura Y, Weber F, et al. Clinical profiles and outcomes of acute type B aortic dissection in the current era: lessons from the International Registry of Aortic Dissection (IRAD). Circulation. 2003;108 Suppl 1:Ii312-7.

35. Elefteriades JA, Lovoulos CJ, Coady MA, Tellides G, Kopf GS, Rizzo JA. Management of descending aortic dissection. Ann Thorac Surg. 1999;67(6):2002-5; discussion 14-9.

36. Nienaber CA, Kische S, Rousseau H, Eggebrecht H, Rehders TC, Kundt G, et al. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. Circ Cardiovasc Interv. 2013;6(4):407-16.

37. Brunkwall J, Lammer J, Verhoeven E, Taylor P. ADSORB: A Study on the Efficacy of Endovascular Grafting in Uncomplicated Acute Dissection of the Descending Aorta. European Journal of Vascular and Endovascular Surgery. 2012;44(1):31-6.

38. Jia X, Guo W, Li TX, Guan S, Yang R, Liu X, et al. The results of stent graft versus medication therapy for chronic type B dissection. Journal of vascular surgery. 2013;57(2):406-14.

39. Xiang D, Kan X, Liang H, Xiong B, Liang B, Wang L, et al. Comparison of mid-term outcomes of endovascular repair and medical management in patients with acute uncomplicated type B aortic dissection. The Journal of thoracic and cardiovascular surgery. 2021;162(1):26-36.e1.

40. Qin YL, Wang F, Li TX, Ding W, Deng G, Xie B, et al. Endovascular Repair Compared With Medical Management of Patients With Uncomplicated Type B Acute Aortic Dissection. Journal of the American College of Cardiology. 2016;67(24):2835-42.

41. Kreibich M, Siepe M, Berger T, Beyersdorf F, Soschynski M, Schlett CL, et al. Intervention rates and outcomes in medically managed uncomplicated descending thoracic aortic dissections. The Journal of Thoracic and Cardiovascular Surgery. 2021.

42. Nienaber CA, Rousseau H, Eggebrecht H, Kische S, Fattori R, Rehders TC, et al. Randomized Comparison of Strategies for Type B Aortic Dissection. Circulation. 2009;120(25):2519-28.

43. Amabile P, Grisoli D, Giorgi R, Bartoli JM, Piquet P. Incidence and determinants of spinal cord ischaemia in stent-graft repair of the thoracic aorta. Eur J Vasc Endovasc Surg. 2008;35(4):455-61.

44. Riambau V, Capoccia L, Mestres G, Matute P. Spinal cord protection and related complications in endovascular management of B dissection: LSA revascularization and CSF drainage. Ann Cardiothorac Surg. 2014;3(3):336-8.

45. Mousa AY, Morcos R, Broce M, Bates MC, AbuRahma AF. New Preoperative Spinal Cord Ischemia Risk Stratification Model for Patients Undergoing Thoracic Endovascular Aortic Repair. Vascular and endovascular surgery. 2020;54(6):487-.

46. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). Eur Heart J. 2014;35(41):2873-926.

47. Weissler E, Voigt S, Raman V, Jawitz O, Doberne J, Anand J, et al. Permissive Hypertension and Collateral Revascularization May Allow Avoidance of Cerebrospinal Fluid Drainage in Thoracic Endovascular Aortic Repair. The Annals of thoracic surgery. 2020;110(5):1469-74.

48. Patterson B, Holt P, Nienaber CA, Cambria RP, Fairman R, Thompson M. Aortic pathology determines midterm outcome after endovascular repair of the thoracic aorta: report from the Medtronic Thoracic Endovascular Registry (MOTHER) database. Circulation. 2013;127(1):24-32.

49. Patterson B, Holt P, Nienaber CA, Fairman R, Heijmen RH, Thompson M. Management of the left subclavian artery and neurologic complications after thoracic endovascular aortic repair. Journal of vascular surgery. 2014;60(6):1491-8.e1.

50. Bismuth J, Garami Z, Anaya-Ayala J, Naoum J, El Sayed H, Peden E, et al. Transcranial Doppler findings during thoracic endovascular aortic repair. Journal of vascular surgery. 2011;54(2):364-9.

51. Janczak D, Ziomek A, Kobecki J, Malinowski M, Pormańczuk K, Chabowski M. Neurological complications after thoracic endovascular aortic repair. Does the left subclavian artery coverage without revascularization increase the risk of neurological complications in patients after thoracic endovascular aortic repair? Journal of Cardiothoracic Surgery. 2019;14(1).

52. Chen Y, Zhang S, Liu L, Lu Q, Zhang T, Jing Z. Retrograde Type A Aortic Dissection After Thoracic Endovascular Aortic Repair: A Systematic Review and Meta-Analysis. Journal of the American Heart Association. 2017;6(9).

53. Ma T, Dong Z, Fu W, Guo D, Xu XY, Chen B, et al. Incidence and risk factors for retrograde type A dissection and stent graft-induced new entry after thoracic endovascular aortic repair. Journal of vascular surgery. 2018;67(4):1026-33.e2.

54. Jang H, Kim MD, Kim GM, Won JY, Ko YG, Choi D, et al. Risk factors for stent graft-induced new entry after thoracic endovascular aortic repair for Stanford type B aortic dissection. Journal of vascular surgery. 2017;65(3):676-85.

55. Kamman AV, de Beaufort H, van Bogerijen GHW, Nauta F, Heijmen RH, Moll FL, et al. Contemporary Management Strategies for Chronic Type B Aortic Dissections: A Systematic Review. PloS one. 2016;11(5).

56. Riambau V, Böckler D, Brunkwall J, Cao P, Chiesa R, Coppi G, et al. Editor's Choice; Management of Descending Thoracic Aorta Diseases: Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS). European Journal of Vascular and Endovascular Surgery. 2017;53(1):4-52.

57. Grabenwöger M, Alfonso F, Bachet J, Bonser R, Czerny M, Eggebrecht H, et al. Thoracic Endovascular Aortic Repair (TEVAR) for the treatment of aortic diseases: a position statement from the European

Association for Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association of Percutaneous Cardiovascular Interventions (EAPCI). Eur Heart J. 2012;33(13):1558-63.

58. Romeiro AB, Nogueira C, Coelho A, Mansilha A. Predictors of adverse events in uncomplicated type B aortic dissection: a systematic review with meta-analysis. Int Angiol. 2021;40(5):416-24.

59. Lombardi JV, Hughes GC, Appoo JJ, Bavaria JE, Beck AW, Cambria RP, et al. Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) reporting standards for type B aortic dissections. Journal of Vascular Surgery. 2020;71(3):723-47.

60. Tadros RO, Tang GHL, Barnes HJ, Mousavi I, Kovacic JC, Faries P, et al. Optimal Treatment of Uncomplicated Type B Aortic Dissection: JACC Review Topic of the Week. Journal of the American College of Cardiology. 2019;74(11):1494-504.

61. Chang C-P, Liu J-C, Liou Y-M, Chang S-S, Chen J-Y. The Role of False Lumen Size in Prediction of In-Hospital Complications After Acute Type B Aortic Dissection. Journal of the American College of Cardiology. 2008;52(14):1170-6.

62. van Bogerijen GHW, Tolenaar JL, Grassi V, Lomazzi C, Segreti S, Rampoldi V, et al. Biomarkers in TAA—The Holy Grail. Prog Cardiovasc Dis. 2013;56(1):109-15.

63. Li S, Yang J, Dong J, Guo R, Chang S, Zhu H, et al. Neutrophil to lymphocyte ratio and fibrinogen values in predicting patients with type B aortic dissection. Scientific reports. 2021;11(1):11366-.

64. Xie E, Yang F, Luo S, Liu Y, Xue L, Huang W, et al. Association Between Preoperative Monocyte to High-Density Lipoprotein Ratio on In-hospital and Long-Term Mortality in Patients Undergoing Endovascular Repair for Acute Type B Aortic Dissection. Frontiers in Cardiovascular Medicine. 2022;8.

65. Jubouri M, Tan SZCP, Bashir M.Monocyte to high-density lipoprotein ratio as a predictivebiomarker for in-hospital mortality following surgery for typeA aortic dissection: reality or myth?J Card Surg. 2022;37:1203-1205.

66. Patamsytė V, Žukovas G, Gečys D, Žaliaduonytė D, Jakuška P, Benetis R, et al. Long Noncoding RNAs CARMN, LUCAT1, SMILR, and MALAT1 in Thoracic Aortic Aneurysm: Validation of Biomarkers in Clinical Samples. Dis Markers. 2020;2020:8521899.

67. Lavingia KS, Larion S, Ahanchi SS, Ammar CP, Bhasin M, Mirza AK, et al. Volumetric analysis of the initial index computed tomography scan can predict the natural history of acute uncomplicated type B dissections. Journal of Vascular Surgery. 2015;62(4):893-9.

68. François CJ, Markl M, Schiebler ML, Niespodzany E, Landgraf BR, Schlensak C, et al. Four-dimensional, flow-sensitive magnetic resonance imaging of blood flow patterns in thoracic aortic dissections. The Journal of Thoracic and Cardiovascular Surgery. 2013;145(5):1359-66.

69. Liu D, Fan Z, Li Y, Zhang N, Sun Z, An J, et al. Quantitative Study of Abdominal Blood Flow Patterns in Patients with Aortic Dissection by 4-Dimensional Flow MRI. Scientific Reports. 2018;8(1):9111.

70. Clough RE, Waltham M, Giese D, Taylor PR, Schaeffter T. A new imaging method for assessment of aortic dissection using four-dimensional phase contrast magnetic resonance imaging. J Vasc Surg. 2012;55(4):914-23.

71. Clough RE, Hussain T, Uribe S, Greil GF, Razavi R, Taylor PR, et al. A new method for quantification of false lumen thrombosis in aortic dissection using magnetic resonance imaging and a blood pool contrast agent. J Vasc Surg. 2011;54(5):1251-8.

72. Zilber ZA, Boddu A, Malaisrie SC, Hoel AW, Mehta CK, Vassallo P, et al. Noninvasive Morphologic and Hemodynamic Evaluation of Type B Aortic Dissection: State of the Art and Future Perspectives. Radiol Cardiothorac Imaging. 2021;3(3):e200456.

73. Adriaans BP, Wildberger JE, Westenberg JJM, Lamb HJ, Schalla S. Predictive imaging for thoracic aortic dissection and rupture: moving beyond diameters. Eur Radiol. 2019;29(12):6396-404.

74. Richter GM. State-of-the-Art Imaging of the Aorta with Respect to Endoluminal Intervention. Cardio-Vascular and Interventional Radiology. 2020;43(12):1745-55.