Acute Type B Dissection

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Abstract

Aortic dissections are catastrophic vascular emergencies, and early recognition and appropriate interventions can be crucial to survival. Research has changed the way aortic dissections are managed over the past two decades and will continue to contribute to the evolution of treatment modalities. Early treatment for uncomplicated type B dissections still remains controversial but certain characteristics may benefit from early intervention.

Keywords

Aortic dissection, complicated dissection, TEVAR, thoracic endovascular aortic repair, type B aortic dissection, uncomplicated dissection

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The first classification of aortic dissection was made by Debakey et al. in 1965.[1] The widely accepted Stanford classification was coined by Daily et al. 5 years later; in this classification type B signifies that the intimal tear is distal to the left subclavian artery (similar to Debakey type 3).[2] The basis of these classifications was predicated on the vastly different outcomes and treatment modalities for the different presentations. Additionally, the timeline from onset of symptoms can divide presentation into acute (<2 weeks), subacute (2 weeks to 3 months) and chronic (>3 months). It was recognised early on that medical management of type B aortic dissection (TBAD) resulted in good initial results with a review of published literature suggesting a 5-year mortality of 75–88%.[3,4] TBAD can quickly become catastrophic – in-hospital mortality is as high as 10–14%.[5,6]

Presentation and Diagnosis

The classic presentation of TBAD is an abrupt onset of severe sharp tearing or ripping pain in the chest or back. Pain is the presentation in 70–72.4% of cases of TBAD, with people most often experiencing pain in the back or occasionally the abdomen. Hypertension is the second most common presentation according to the International Registry of Aortic Dissection (IRAD) for TBAD, occurring in 66–70.1% of cases.[5,7] Hypertension is also a predictor for complications, with blood pressure control representing an important goal for management. Early high inpatient mortality represents a subset of TBAD due to malperfusion and/or rupture.[7] However, this may only represent the tip of the iceberg because estimates from autopsies suggest that 20–30% cases of TBAD do not make it to hospital.[8]

Malperfusion may be the presenting feature and occurs in up to 10% of TBAD cases.[4] End organ dysfunction must be recognised early and is indicative of impending organ failure. This may be static or dynamic for flow, which can be corroborated by laboratory findings and imaging. Compromised spinal, visceral, renal or iliac blood flow may result in paralysis, paraplegia, lower limb ischaemia, abdominal pain or diarrhoea with raised liver function, amylase, lactate, urea, creatinine and coupled with absence (thrombosis) or delay in contrast enhancement in the target organ.

The role of contrast enhanced imaging cannot be overemphasised as it gives useful information about the predictors of failure, malperfusion, pre-treatment sizing and – most importantly – aortic rupture. The diagnosis of impending rupture as defined as haemorrhagic pleural effusion or expanding peri-aortic haematoma can only be made with imaging.[9] Historically, retrograde aortography was considered the gold standard for diagnosis; fortunately, this is only used during interventions. CT angiography (CTA) has become widely available with rapid image acquisition. CTA has a 93.8% sensitivity and a specificity of 87.1% which compares well with other modalities such as MRI or transoesophageal echocardiogram for acute aortic emergencies.[10] MRI has sensitivity rates of 95–100% but is limited by its long examination time and availability.[11] Transoesophageal echocardiogram for TBAD is limited by the need for general anaesthesia and it loses sensitivity in the aortic arch which is in the vicinity of the proximal tear. Intravascular ultrasound is now considered the standard intervention for TBAD.

Natural History and Aortic Remodelling

In 80% of TBAD cases, the intimal tear is in a posterior-lateral aspect to the aorta, a few centimetres distal to the left subclavian artery with the trajectory of the false lumen taking various configurations but most commonly involving the left renal artery.[12] Debakey et al. first reported that up to 40% of TBAD will degenerate over time.[13] Since the founding of IRAD in 1995, there is a better understanding of the long-term outcomes. Five-year analysis suggests a better long-term survival in thoracic endovascular repair versus best medical management for

TBAD shown by the IRAD registry.[14] The ideology behind this is that aortic-related pathology will develop with time and the patients will eventually succumb to it. Only 41% of TBAD remain intervention-free at 6 years follow-up with 65% of the aortic-related interventions related to aneurysmal degeneration. Note that this is highly predicated on time, as these benefits were not realised at the 2-year follow-up. A significant reduction in all-cause mortality and aortic-specific mortality at 5 years was shown by the first randomised trial for uncomplicated TBAD with thoracic endovascular repair of the aorta (TEVAR) versus best medical management.[15,16]

There is a correlation with false lumen patency and progression to aneurysmal progression in TBAD.[17] Tsai et al. showed mortality post-discharge based on thrombosis of false lumen was 13.7% in complete thrombosis, 31.6% in partial thrombosis and 22.6% in complete thrombosis.[18] The concept of aortic remodelling with inducing false lumen thrombosis by sealing the aortic tear with TEVAR and redirecting flow to the true lumen has the potential to reduce complications. Additionally, Tolennar et al. showed patients with fully patent false lumen at presentation and branch vessel involvement are less likely to develop thrombosis and may benefit from TEVAR.[19] This was corroborated in the INvestigation of Stent Grafts in Aortic Dissection (INSTEAD) trial with positive remodelling showing expansion of the true lumen from a mean of 19.4 mm to 32.4 mm at 2 years and shrunken false lumen from a mean of 29.3–8.6 mm at 2 years in subacute to early chronic, uncomplicated TBAD. This was maintained at 5 years and was reproducible at different levels of the aorta.[15,16]

The real-world experience from the VIRTUE Registry – a prospective, non-randomised, multicentre European registry – shows promisingly similar results for complicated TBAD.[20] The true lumen increased across all levels of aorta involved with reduction in the false lumen after TEVAR but interestingly, this reduction was more marked in the acute and subacute groups. This adds insight into the plasticity of the intermembrane, suggesting the more acute the dissection, the more likely it is that aortic remodelling will be successful. The majority of the changes in the lumen size occurs by 6 months.

Management

Medical management guidelines recommend a target systolic blood pressure of 100–120 mmHg with heart rate <60 BPM.[21] Beta-blockers should be the first-line treatment and should be used in preference to vasodilators unless contraindicated. It is not unusual for patients to need multiple medications for blood pressure control. Continuous monitoring in an intensive setting is preferred because up to 10–12% of these patients will have complications that occur during the initial encounter.

Acute Complicated Type B Dissection

Details of stent grafting for a complicated TBAD was first published in 1999.[22,23] This was the start of endovascular treatment of TBAD, which resulted in TEVAR and it was initially only used in cases that were not amenable for open surgery. The definition of complicated was standardised by the interdisciplinary expert group to include malperfusion, persistent or uncontrolled hypertension despite full medical therapy, and rupture or impending rupture.[4] Open repair has traditionally offered the best chance of survival in complicated TBAD with the necessity for thoracotomy and left heart bypass adding to its risk of mortality. A high 30-day mortality of 19% and combined neurological complication of 9.8% in a meta-analysis by Moulakakis et al. made this a daunting procedure.[24] This is compared to mortality in TEVAR of 10.2% and combined neurological complications at 9.1%.[4] There are now several measures to improve neurological outcomes.[25] Long-term outcomes of TEVAR now include 5-year freedom from aortic-related interventions of 45–77% and survival ranging from 62 to 100%.[4] This changing landscape of treatment has led an expert consensus to recommend TEVAR as the first-line treatment for complicated TBAD. Other modalities for treatment, such as fenestrations, have fallen out of favour.

Interventions may be multiple; in addition to TEVAR, it may be necessary to ensure flow to the renal, visceral, iliac and left subclavian artery with stenting, fenestration or open surgical adjuncts. Complicated TBAD may also include increasing size, progression of dissection or refractory pain. Trimarchi et al. reviewed the IRAD data and found that recurrent pain or refractory hypertension appeared to be clinical features associated with increased inpatient mortality when managed medically (35.6% versus 1.5%).[26]

The real-world data from IRAD is encouraging. TEVAR confers a similar mortality benefit compared with medical management despite TEVAR being more suitable for the treatment of complicated TBAD.[14] This led to a few TEVAR trials that were specific for acute complicated TBAD. The GORE TAG 08-01 study used the new conformable design placed in 50 patients, resulting in an 8% mortality, 18% stroke rate and 6% paraplegia rate at 30 days, and 88% survival at 1 year; there was a reduced mean false lumen area and increased true lumen diameter up to 3 years post-implantation.[27] The Zenith TX2 was a 'pathology-specific' graft with a combination of proximal covered stent and distal uncovered bare metal stent. It was implanted in 86 patients with acute and subacute complicated TBAD. There was stability or an increase in the true lumen size and a reduction of the false lumen in the thoracic aorta with a 30-day mortality of 4.7%, 7% stroke rate and 88.3% freedom from all-cause mortality at 1 year.[28] However, the false lumen of the abdominal aorta continued to expand. The Valiant Captiva was implanted in 50 patients for acute complicated TBAD with 8% mortality, 6% stroke rate and 6% spinal ischaemia at 30 days; however, 47% of the patients had serious adverse events by 1 year. The true lumen was stable or increased in 93.1%, the false lumen was stable or decreased in 44% with partial or complete thrombosis in 91%.[29]

Overall, this represents the past two decades of a new alignment of treatment of stent graft treatment for complicated TBAD, with trials still ongoing. Continued efforts are needed to lower mortality and neurological complications in this catastrophic disease that would otherwise be fatal. Fenestrated/branched devices may have a role for the future. Open repair is super selective and only reserved for a minority of cases without proximal seal with rupture or impending rupture at the intimal tear in a patient who can tolerate an open procedure. Short segment proximal descending aortic replacement with left ventricular bypass would be the most appropriate procedure.

Acute Uncomplicated Type B Dissection

There is mounting evidence that uncomplicated TBAD will become aneurysmal even with best medical therapy and will be responsible for up to 30% of late mortality combined with up to 66% of late aortic interventions.[13,30] Controversy exists over the decision to intervene or to watch and wait. It is without question that those who develop aneurysmal disease and require intervention have a much higher mortality.

The INSTEAD and Acute Dissection Stent Grafting or Best Medical Treatment (ADSORB) trials are the first and only two randomised trials for uncomplicated TBAD.[15,31] The INSTEAD trial was a randomised study comparing 68 patients with optimal medical management versus 72 patients with TEVAR in addition to optimal medical management treated >14 days after onset of symptoms. This provided evidence of aortic remodelling at 2 years but without any impact on either all-cause or aortic-specific mortality. Early complications included one paraplegia in both study groups, with the intervention group having one transient paraplegia and one stroke. True lumen recovery and false lumen thrombosis occurred in 91.3% of the intervention group compared with 19.4% of the optimal medical management group at 2 years.[15] It was not until 2013 that data emerged to show that TEVAR with optimal medical management reduced aortic-specific mortality (6.9% versus 19.3%) and reduced disease progression (27.0% versus 46.1%) on an intention-to-treat basis.[16] This study has been critiqued for not including acute TBAD and extending its inclusion criterion to early chronic phase (2–52 weeks) when it is thought the inter-membrane plasticity is lost and remodelling is less than ideal.

The ADSORB trial was also randomised to similar comparative groups to include only acute, uncomplicated TBAD. It attempted to accrue 30 patients in the best medical therapy and 31 in the TEVAR plus best medical therapy group. ADSORB used 10 cm increments from the left subclavian artery to interrogate the lumens of sections of the dissection. A unique analysis dividing groups into false lumen growth versus no false lumen growth showed number of branch vessels and longer dissection lengths predicted growth of the false lumen. Additionally, TEVAR plus best medical therapy showed changes from partial to complete or patent to partial thrombosis in 90.3% compared with 31.0% in the best medical therapy group analysing the proximal 20 cm of the descending thoracic aorta.[31] The trial was limited by being underpowered, there was a lack of long-term follow-up, and recruitment was restricted to 21 versus 16 (best medical versus TEVAR with best medical therapy) with a 9.6% crossover to the TEVAR group. With these trials and other encouraging data, the Food and Drug Administration first approved a thoracic device for dissection in 2013. There are now two approved devices in the US.

The same year as the use of the first TEVAR was documented for use in complicated TBAD, Marui et al. suggested that patients with acute uncomplicated TBAD who show a maximum aortic diameter of ≥40 mm and a patent false lumen should undergo surgery earlier before aneurysmal enlargement occurs.[32] Promising results from retrospective studies predict unique characteristics that will allow for treatment of a subset of uncomplicated TBAD that will progress to aneurysmal dilation, preventing its long-term sequelae. Grommes et al. noted that mortality in TBAD was more associated with total aortic diameter ≥41 mm and age >66 years with a 2-year cumulative survival of 68.5%, versus 95.7% for patients without these risk factors.[33] Kudo et al. echoed these results, with their cohort showing aortic diameter >40 mm and ulcer-like projection from the false lumen were associated with late aortic events.[34] Evangelista et al. noted that the larger the intimal tear, the greater likelihood of complications.[35] Bogerijen et al. corroborated all these features, among others, after a systemic review and analysis in 2014.[36]

In 2016, Ray et al. carried out a retrospective review of 156 patients from a single institution with stratified analysis that suggested that an aortic diameter of >44 mm and false lumen diameter of >22 mm were both associated with a decrease in intervention-free survival. Additionally, age over 60 was a predictor of mortality.[37] Schwartz et al. also carried out a retrospective review of a single centre with a larger cohort of 254 patients of whom 38% required intervention. Predictors of late aortic intervention at presentation included total aortic diameter of >40 mm, false lumen of >20 mm, entry tear of >10 mm and an increase in >5 mm between imaging studies. Thrombosis of the false lumen was deemed protective.[38] These studies have put forward convincing data that best medical therapy may not be adequate in all cases and there is a role for TEVAR where the risk of procedure outweighs the risk of aortic-specific mortality.

Conclusion

TBADs are a complex physiological and anatomical pathology. Early recognition, blood pressure control and appropriate interventions can minimise mortality and morbidity. Poor outcomes have driven the evolution of treatment for acute complicated TBAD, and TEVAR is now the standard of care. It is evident that there are predictors of tendency towards long-term consequences for acute uncomplicated TBAD. Continued research with randomised trials with precise and objective data to produce firm consensus guidelines is required. Nevertheless, the practice of TEVAR to allow aortic remodelling and prevent long-term sequalae will continue to be individualised in patients with uncomplicated TBAD. ■

1. Debakey ME, Henly WS, Cooley DA, et al. Surgical management of dissecting aneurysms of the aorta. *J Thorac Cardiovasc Surg* 1965;49:130–49. PMID: 14261867.
2. Daily PO, Trueblood HW, Stinson EB, et al. Management of acute aortic dissections. *Ann Thorac Surg* 1970;10:237–47. https://doi.org/10.1016/S0003-4975(10)65594-4; PMID: 5458238.
3. Wheat MW Jr, Palmer RF, Bartley TD, Seelman RC. Treatment of dissecting aneurysms of the aorta without surgery. *J Thorac Cardiovasc Surg* 1965;50:364–73. PMID: 14346544.
4. Fattori R, Cao P, De Rango P, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. *J Am Coll Cardiol* 2013;61:1661–78. https://doi.org/10.1016/j.jacc.2012.11.072; PMID: 23500232.
5. Pape LA, Awais M, Woznicki EM, et al. Presentation, diagnosis, and outcomes of acute aortic dissection: 17-year trends from the International Registry of Acute Aortic Dissection. *J Am Coll Cardiol* 2015;66:350–8. https://doi.org/10.1016/j.jacc.2015.05.029; PMID: 26205591.
6. Suzuki T, Mehta RH, Ince H, 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:II312–7. https://doi.org/10.1161/01.cir.0000087386.07204.09; PMID: 12970252.
7. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA* 2000;283:897–903. https://doi.org/10.1001/jama.283.7.897; PMID: 10685714.
8. Tsai TT, Fattori R, Trimarchi S, et al. Long-term survival in patients presenting with type B acute aortic dissection: insights from the International Registry of Acute Aortic Dissection. *Circulation* 2006;114:2226–31. https://doi.org/10.1161/CIRCULATIONAHA.106.622340; PMID: 17101856.
9. Scott AJ, Bicknell CD. Contemporary management of acute type B dissection. *Eur J Vasc Endovasc Surg* 2016;51(3):452–9. https://doi.org/10.1016/j.ejvs.2015.10.026; PMID: 26684594.
10. Nienaber CA, von Kodolitsch Y, Nicolas V, et al. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. *N Engl J Med* 1993;328:1–9. https://doi.org/10.1056/NEJM199301073280101; PMID: 8416265.
11. Moore AG, Eagle KA, Bruckman D, et al. Choice of computed tomography, transesophageal echocardiography, magnetic resonance imaging, and aortography in acute aortic dissection: International Registry of Acute Aortic Dissection (IRAD). *Am J Cardiol* 2002;89:1235–8. https://doi.org/10.1016/S0002-9149(02)02316-0; PMID: 12008187.
12. Crawford ES. The diagnosis and management of aortic dissection. *JAMA* 1990;264:2537–41. https://doi.org/10.1001/jama.1990.03450190069031; PMID: 2232021.
13. DeBakey ME, McCollum CH, Crawford ES, et al. Dissection and dissecting aneurysms of the aorta: twenty-year follow-up of five hundred twenty-seven patients treated surgically. *Surgery* 1982;92:1118–34. PMID: 7147190.
14. Fattori R, Montgomery D, Lovato L, et al. Survival after endovascular therapy in patients with type B aortic dissection: a report from the International Registry of Acute Aortic Dissection (IRAD). *JACC Cardiovasc Interv* 2013;6:876–82. https://doi.org/10.1016/j.jcin.2013.05.003; PMID: 23968705.
15. Nienaber CA, Rousseau H, Eggebrecht H, et al. Randomized comparison of strategies for type B aortic dissection: the INvestigation of STEnt Grafts in Aortic Dissection (INSTEAD) trial. *Circulation* 2009;120:2519–28. https://doi.org/10.1161/CIRCULATIONAHA.109.886408; PMID: 19996018.
16. Nienaber CA, Kische S, Rousseau H, 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;6:407–16. https://doi.org/10.1161/CIRCINTERVENTIONS.113.000463; PMID: 23922146.
17. Sueyoshi E, Sakamoto I, Hayashi K, et al. Growth rate of aortic diameter in patients with type B aortic dissection during the chronic phase. *Circulation* 2004;110:II256–61. https://doi.org/10.1161/01.CIR.0000138386.48852.b6; PMID: 15364872.
18. Tsai TT, Evangelista A, Nienaber CA, et al. Partial thrombosis of the false lumen in patients with acute type B aortic dissection. *N Engl J Med* 2007;357:349–59. https://doi.org/10.1056/NEJMoa063232; PMID: 17652650.
19. Tolenaar JL, Kern JA, Jonker FH, et al. Predictors of false lumen thrombosis in type B aortic dissection treated with TEVAR. *Ann Cardiothorac Surg* 2014;3:255–63. doi: 10.3978/j.issn.2225-319X.2014.05.17; PMID: 24967164.

20. Investigators VR. Mid-term outcomes and aortic remodelling after thoracic endovascular repair for acute, subacute, and chronic aortic dissection: the VIRTUE Registry. *Eur J Vasc Endovasc Surg* 2014;48:363–71. https://doi.org/10.1016/j.ejvs.2014.05.007; PMID: 24952999.
21. Estrera AL, Miller CC, Safi HJ, et al. Outcomes of medical management of acute type B aortic dissection. *Circulation* 2006;114:I384–9. https://doi.org/10.1161/CIRCULATIONAHA.105.001479; PMID: 16820605.
22. Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med* 1999;340:1546–52. https://doi.org/10.1056/NEJM199905203402004; PMID: 10332016.
23. Nienaber CA, Fattori R, Lund G, et al. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. *N Engl J Med* 1999;340:1539–45. https://doi.org/10.1056/NEJM199905203402003. PMID: 10332015.
24. Moulakakis KG, Mylonas SN, Dalainas I, et al. Management of complicated and uncomplicated acute type B dissection. A systematic review and meta-analysis. *Ann Cardiothorac Surg* 2014;3:234–46. doi: 10.3978/j.issn.2225-319X.2014.05.08; PMID: 24967162.
25. Hnath JC, Mehta M, Taggert JB, et al. Strategies to improve spinal cord ischemia in endovascular thoracic aortic repair: Outcomes of a prospective cerebrospinal fluid drainage protocol. Journal of vascular surgery. 2008;48:836–40. https://doi.org/10.1016/j.jvs.2008.05.073; PMID: 18723308.
26. Trimarchi S, Eagle KA, Nienaber CA, 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;122:1283–9. https://doi.org/10.1161/CIRCULATIONAHA.109.929422; PMID: 20837896.
27. Cambria RP, Conrad MF, Matsumoto AH, et al. Multicenter clinical trial of the conformable stent graft for the treatment of acute, complicated type B dissection. *J Vasc Surg* 2015;62:271–8. https://doi.org/10.1016/j.jvs.2015.03.026; PMID: 26211376.
28. Lombardi JV, Cambria RP, Nienaber CA, et al. Aortic remodeling after endovascular treatment of complicated type B aortic dissection with the use of a composite device design. *J Vasc Surg* 2014;59:1544–54. https://doi.org/10.1016/j.jvs.2013.12.038; PMID: 24560244.
29. Bavaria JE, Brinkman WT, Hughes GC, et al. Outcomes of thoracic endovascular aortic repair in acute type b aortic dissection: results from the Valiant United States Investigational Device Exemption Study. *Ann Thorac Surg* 2015;100:802–8; discussion:8–9. https://doi.org/10.1016/j.athoracsur.2015.03.108; PMID: 26209487.
30. Durham CA, Cambria RP, Wang LJ, et al. The natural history of medically managed acute type B aortic dissection. *J Vasc Surg* 2015;61:1192–8. https://doi.org/10.1016/j.jvs.2014.12.038; PMID: 25659458.
31. Kamman AV, Brunkwall J, Verhoeven EL, et al. 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;65:964–71e3. https://doi.org/10.1016/j.jvs.2016.09.033; PMID: 27876516.
32. Marui A, Mochizuki T, Mitsui N, et al. Toward the best treatment for uncomplicated patients with type B acute aortic dissection: A consideration for sound surgical indication. *Circulation* 1999;100:II275-80. https://doi.org/10.1161/01.CIR.100.suppl_2.II-275; PMID: 10567316.
33. Grommes J, Greiner A, Bendermacher B, et al. Risk factors for mortality and failure of conservative treatment after aortic type B dissection. *J Thorac Cardiovasc Surg* 2014;148:2155–60. https://doi.org/10.1016/j.jtcvs.2014.03.053; PMID: 24793648.
34. Kudo T, Mikamo A, Kurazumi H, et al. Predictors of late aortic events after Stanford type B acute aortic dissection. *J Thorac Cardiovasc Surg* 2014;148:98–104. https://doi.org/10.1016/j.jtcvs.2013.07.047; PMID: 24029294.
35. Evangelista A, Galuppo V, Gruosso D, et al. Role of entry tear size in type B aortic dissection. *Ann Cardiothorac Surg* 2014;3:403–5. https://doi.org/10.3978/j.issn.2225-319X.2014.07.03. PMID: 25133104.
36. van Bogerijen GH, Tolenaar JL, Rampoldi V, et al. Predictors of aortic growth in uncomplicated type B aortic dissection. *J Vasc Surg* 2014;59:1134–43. https://doi.org/10.1016/j.jvs.2014.01.042; PMID: 24661897.
37. Ray HM, Durham CA, Ocazionez D, et al. Predictors of intervention and mortality in patients with uncomplicated acute type B aortic dissection. *J Vasc Surg* 2016;64:1560–8. https://doi.org/10.1016/j.jvs.2016.07.111; PMID: 27707621.
38. Schwartz SI, Durham C, Clouse WD, et al. Predictors of late aortic intervention in patients with medically treated type B aortic dissection. *J Vasc Surg* 2018;67:78–84. https://doi.org/10.1016/j.jvs.2017.05.128; PMID: 28912006.