The Journal of Tehran University Heart Center

Review Article

Type B Aortic Dissection: Management Updates

Mehrab Marzban, MD*, Naghmeh Moshtaghi, MD, Nasim Mirhosseini, MD

Tehran Heart Center, Tehran University of Medical Sciences, Tehran, Iran.

Abstract

Acute aortic dissection (AAD) is the most frequent catastrophic event of the aorta; it occurs nearly three times as frequently as the rupture of abdominal aortic aneurysm. Sixty percent of dissection cases are classified as proximal or type A and 40% as distal or type B, according to the Stanford Classification. The most frequent causes of death in acute type B dissection are aortic rupture and malperfusion syndrome.

We herein review recent data suggesting different management modalities of type B aortic dissection, including medical, surgical, and endovascular treatments. Although medical therapy is still the standard approach in uncomplicated cases, there are subgroups of patients who may benefit from endovascular management. Endovascular techniques or surgery are valuable options for complicated cases. Hybrid suites, multidisciplinary approaches, and good imaging techniques can be considered as the key to success in this regard.

J Teh Univ Heart Ctr 4 (2009) 218-221

Keywords: *Aortic diseases • Dissection • Mortality • Treatment outcome*

"The tragedies of life are largely arterial."

"Sir William Osler"[1]

Introduction

The term "acute aortic syndrome" refers to a variety of pathological-clinical entities affecting the aorta; they include rupture of aortic aneurysm, acute aortic dissection (AAD), intramural hematoma, penetrating atherosclerotic ulcer, and traumatic aortic transection.[2]

AAD is the most frequent catastrophic event of the aorta; it occurs nearly three times as frequently as the rupture of abdominal aortic aneurysm.[2] In one autopsy series, the antemortem diagnosis, made in only 15% of patients, revealed that many immediately fatal events remain undiagnosed.[3] Men are twice as often found to suffer AAD as are women, with 60% of dissection cases classified as proximal or type A and 40% as distal or type B according to the Stanford classification.[4]

The historical data of untreated aortic dissection of the ascending aorta show a mortality rate of 1% per hour in the first 2 days, resulting in a mortality rate of 50% in the first 48 hours and 75% in the first two weeks.[3]

Uncomplicated acute type B dissection is less frequently fatal with survival rates of 89% in medically treated patients at 1 month, 84% within 1 year, and up to 80% within 5 years.[5, 6] However, other variable long-term prognoses have also been reported with 5-year survival rates as low as 48%.[7, 8] The most frequent causes of death in acute type B dissection are aortic rupture and malperfusion syndrome.

The indications for surgical or interventional repair include contained or free aortic rupture, acute aortic expansion, intractable pain, uncontrollable hypertension, and progression of dissection despite maximal medical management.

Medical treatment

Currently, the primary treatment of uncomplicated acute

*Corresponding Author: ***Mehrab Marzban,*** *Assistant Professor of Cardiac Surgery, Tehran University of Medical Sciences, Tehran Heart Center, North Karegar Street, Tehran, Iran. 1411713138. Tel: +98 21 88029256. Fax: +98 21 88029256. E-mail: mehrabmarzban@yahoo.com.*

type B dissection is medical.

Most of the guiding tenets for the management of AAD, derived from the early experience of Wheat and Austen, suggest a better outcome with medical management.[9, 10]

Patients with an impression or diagnosis of AAD should be admitted to the intensive care unit for hemodynamic monitoring and diagnostic evaluation and be provided with good IV-line, invasive arterial pressure monitoring, urine output monitoring, supplemental oxygen, and Electrocardiogram (ECG). In addition, blood samples should be obtained for routine laboratory tests cardiac enzymes and type- cross match for 10 units of packed cells. It is recommended that any painful or stressful procedure such as central venous line placement or transesophageal echocardiography (TEE) be avoided in waking patients and that pain be treated with appropriate analgesics like morphine.

Kodama and his colleagues demonstrated that tight heart rate control with β-blockers reduced secondary adverse events in patients with type B AAD.[11] Target systolic blood pressure should be between 90-110 mmHg. β-blockers are the first choice drugs because they control the maximal force of left ventricular contraction (dp/dt max) in addition to heart rate and blood pressure, which can avert the extension of dissection, rupture, or malperfusion. Common β-blockers include Labetolol, Esmolol, Metoprolol, and Atenolol.

Calcium channel blockers like Diltiazem or Nicardipine may be alternative in patients with potential intolerance to β-blockers as is the case with asthmatics.

Vasodilators such as Nitroglycerine, Sodium Nitroprusside, or Hydralazine can be added to β-blockers to control blood pressure, but some of these vasodilators may give rise to reflex tachycardia and so should be used cautiously.

Surgical treatment

The modern surgical treatment for AAD, including those for acute type B aortic dissection, began after the landmark aortic operation by Drs Cooley and Debakey in 1950s.[12]

Complications such as contained or free rupture, acute aortic expansion, malperfusion (visceral or limb), intractable pain, and uncontrollable hypertension may necessitate interventional management; the goal of surgery is the prevention of malperfusion or rupture of the aorta, which are the most frequent causes of death in complicated type B dissection.

The results of surgery are currently suboptimal, with reported in-hospital mortality rates of 25-50%.[13] Improved imaging modalities have made faster recognition of the disease possible, and streamlined techniques of cardiopulmonary bypass, spinal cord protection, hemostatic vascular graft, and intensive care of these critically ill patients should all be credited for the recent decrease (down to 10%) in operative mortality rates in most experienced centers. There are reports of no operative mortality in 34 consecutive patients at a single center[14] or in specific series, not including patients with pre-operative thoracoabdominal malperfusion syndromes. For all these promising improvements, however, the overall surgical mortality in the International Registry of Aortic Dissection (IRAD) stands at 29.3%.

The ideal operation is the replacement of as little of the descending aorta as is necessary when resecting the segment that contains the primary tear site, which is most often located in the proximal 1/3 of the descending aorta. This strategy is based on the fact that an increase in the size of the resected segment adds to the risk of paraplegia (as high as 19%).[15]

In recent years, adjunct measures such as CSF drainage and cardiopulmonary bypass have decreased the risk of early neurological deficit[16] Dr. Safi and his co-workers demonstrated the safety and efficacy of these adjuncts in a large series.[17, 18]

Whereas most centers advocate surgical treatment for complications of type B dissection, some centers consider the large diameter of the false lumen as a predictor of late complication and, therefore, recommend surgery in the acute phase for patients with a large aortic diameter (≥ 40 mm) and a patent primary entry site in the thorax.[19] In one study, branch vessel involvement and a large maximal false lumen area were shown to be powerful predictors of in-hospital complications.[20]

Hemodynamic instability in type B dissection is due to hemorrhagic shock (rupture) or visceral or limb malperfusion. Intubation and fluid resuscitation should be commenced prior to the patient's transfer to the operating room or hybrid suite, depending on the hospital's facilities. As was mentioned before, renal or mesenteric ischemia correlates with a high surgical mortality rate.[21-23]

Endovascular treatment

The latest additions to the armamentarium to treat dissection have been based on percutanous interventional techniques. The minimally invasive nature of these techniques makes them an attractive alternative to open surgical intervention; be that as it may, the exact role and long-term durability of these procedures remain to be determined. The earliest endovascular therapies were directed toward the complications of aortic dissections and included angioplasty of an obstructed aorta, angioplasty and stenting of branch vessels, and fenestration of the intimal flap to treat the malperfusion.[24-26]

More recently with newer generations of endografts, surgeons, interventional cardiologists, and radiologists have extended the application of thoracic endovascular aortic repair (TEVAR). The concept is that covering the primary intimal tear in the thoracic aorta depressurizes the false lumen, accelerates the thrombosis of the false lumen, and prevents the aortic expansion or progression of the dissection.

Table 1. Endovascular Stent-Graft Repair of Type B Aortic Dissection[30]

Study	Year		Technical success %	Retrograde %	Stroke %	Paraplegia %	30-day mortality %
Eggebrech (meta-analysis)	2006	609 (Type B: 96%; retrograde type A: 4%)	2006	19	19	0.8	5.3
Leurs (EUROSTAR/UK)	2004	131 (Type A: 7; type B: 106: N/A:18)	2004	N/A	1.5	0.8	8.4
Bortone	2004	43 (Acute type B: 24; chronic type:19)	2004	5-7	0	0	7
Dialetto	2005	28 (All type B)	2005	4	0	0	10.7
Nathanson	2005	40 (All type B)	2005	N/A	2.5	2.5	2.5
Eggebrecht	2005	38 (Acute type B: 10; chronic type B: 28)	2005	N/A	2.6	0	2.6
Xu	2006	63 (All type B)	2006	4.8	16	0	3.2
Chen	2006	62 (All type B)	2006	4.8	3.2	0	4.8
Schoder	2007	28 (All type B)	2007	7.1	0	3.6	10.7

N/A, Not applicable

There are two other endovascular applications in complicated cases: flap fenestration and branch vessel stenting.

In some cases endovascular techniques may obviate the need for surgical management, whereas in other cases, endovascular techniques are complementary to surgical repair. As in endovascular treatment of abdominal aorta, TEVAR needs a proximal and distal landing zone of enough length to seal the blood flow to the false lumen and prevent the transmission of systemic blood pressure to the false lumen; this means redirection of blood to the true lumen to relieve the dynamic obstruction of the branches supplied by a diminutive true lumen.

A very important point in these cases of TEVAR is the sizing of the stent graft; the segment between the left common carotid and left subclavian artery (normal aorta at proximal) is used to estimate the original size of the involved aorta before dissection. This measurement is oversized up to 10% to select the stent graft diameter (In usual cases of thoracic aneurysm or chronic dissection, a 20% oversizing is recommended).

The other endovascular treatment, fenestration seeks to create a distal reentry to the true lumen so as to effect equalization of pressure between the true and false lumens and thus relieve the dynamic obstruction of the branch vessels.[27, 28]

Because the stent graft treatment is a more definite treatment directed at correcting the pathology, fenestration is reserved for those in whom anatomical constraints preclude stent grafting or for those who still bear the stigmata of organ or limb malperfusion after surgical repair or endovascular treatment.

Percutaneous fenestration can be performed under fluoroscopic guidance or with intravascular ultrasound or both, and, needless to say, it requires a high level of interventional skill.

Sometimes, the mechanism of the obstruction of the aorta or side branches is static (compression). In such cases, attempts are made to restore normal blood flow to the involved branches by placing uncovered stents via the lumen of the aorta into the true lumen of the side branches. It also can be employed to reopen the collapsed aorta and support the true lumen.[29]

Outcomes of endovascular management

A meta-analysis by Eggebrecht et al.[30] reviewed all data published between January 1999 and May 2004 involving stent-graft placement for aortic dissection. A total of 609 patients were reviewed (Table 1).[30] Procedural success rate was 98.2%, neurological complications including stroke or paraplegia occurred in 2.9%, total in-hospital conversion rate reached 2.3%, overall in-hospital mortality rate amounted to 5.2%, and the 30–day mortality rate stood at 5.3%. In the IRAD data, the 30-day mortality rates are 10.7% and 31.4% for the medically treated group and surgical group, respectively.[31]

A composite Kaplan- Meier table, encompassing all the three interventions, shows worse survival outcomes in surgical groups and similar outcomes in medical and endovascular groups. A mean follow-up time of 19.5 months was available for 561 patients. During the follow-up, false lumen thrombosis was observed in 75.5%, late surgical conversion in 2.5%, and supplemental endovascular stent-graft procedure in 4.6% of the patients. The total reintervention rate was 11.9%. It is deserving of note that there are more contemporary studies the results of which are comparable to those of this meta- analysis.[32-34]

These collected observational data demonstrate that the endovascular treatment of type B dissection is feasible with high initial success and acceptable short-term outcomes. In contrast to the general consensus about endovascular treatment for complications of type B dissection, however, the role of this type of treatment in uncomplicated cases is uncertain. Some experts believe that there are subgroups of patients like those with large diameters of the false lumen who may benefit from endovascular intervention in the acute phase. The results of future trials will help shed further light on this dilemma.

Conclusion

Enhanced imaging techniques have ushered in the feasibility of faster diagnosis of this disease entity. In

uncomplicated cases, medical therapy is still the standard but there are subgroups of patients who may benefit from endovascular management.

For complications, surgery or endovascular techniques are valuable options. Hybrid suites, multidisciplinary approaches, and good imaging techniques are the key to success.

References

1. Rogers RL. Don't miss this diagnosis: aortic dissection. www.aorticdissection.com/Aortic%20Diagnosis.htm (15 February 2009).
2. Coady MA, Rizzo JA, Goldstein LJ, Elefteriades JA. Natural history, pathogenesis, and etiology of thoracic aortic aneurysms and dissections. Cardiol Clin 1999;17:615-635.
3. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, Rakowski H, Struyven J, Radegran K, Sechtem U, Taylor J, Zollikofer C, Klein WW, Mulder B, Providencia LA; Task force on aortic dissection, European society of cardiology. Diagnosis and management of aortic dissection. Eur Heart J 2001;22:1642-1681.
4. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, Evangelista A, Fattori R, Suzuki T, Oh JK, Moore AG, Malouf JF, Pape LA, Gaca C, Sechtem U, Lenferink S, Deutsch HJ, Diedrichs H, Marcos Y Robles J, Llovet A, Gilon D, Das SK, Armstrong WF, Deeb GM, Eagle KA. The International registry of acute aortic dissection (IRAD): new insights into an old disease. JAMA 2000;283:897-903.
5. Estrera AL, Miller CC 3rd, Safi HJ, Goodrick JS, Keyhani A, Porat EE, Achouh PE, Meada R, Azizzadeh A, Dhareshwar J, Allaham A. Outcomes of medical management of acute type B aortic dissection. Circulation 2006;114:I384-389.
6. Glower DD, Fann JI, Speier RH, Morrison L, White WD, Smith LR, Rankin JS, Miller DC, Wolfe WG. Comparison of medical and surgical therapy for uncomplicated descending aortic dissection. Circulation 1990;82:IV39-46.
7. Gysi J, Schaffner T, Mohacsi P, Aeschbacher B, Althaus U, Carrel T. Early and late outcome of operated and non-operated acute dissection of the descending aorta. Eur J Cardiothorac Surg 1997;1:1163-1169.
8. Glower DD, Speier RH, White WD, Smith LR, Rankin JS, Wolfe WG. Management and long-term outcome of aortic dissection. Ann Surg 1991;214:31-41.
9. Austen WG, DeSanctis RW. Dissecting aneurysm. Surg Clin North Am 1966;46:573-586.
10. Wheat MW Jr, Palmer RF, Bartley TD, Seelman RC. Treatment of dissecting aneurysms of the aorta without surgery. J Thorac Cardiovasc Surg 1965;50:364-373.
11. Kodama K, Nishigami K, Sakamoto T, Sawamura T, Hirayama T, Misumi H, Nakao K. Tight heart rate control reduces secondary adverse events in patients with type B acute aortic dissection. Circulation 2008;118:S167-170.
12. DeBakey ME, Beall AC Jr, Cooley DA, Crawford ES, Morris GC Jr, Garrett HE, Howell JF. Dissecting aneurysms of the aorta. Surg Clin North Am 1966;46:1045-1055.
13. Stone C, Borst H. Dissecting aortic aneurysm. In: Edmunds LH Jr, ed. Cardiac surgery in the adult. New York: McGraw-Hill; 1997. p. 1153-1157.
14. Lansman SL, Hagel C, Fink D, Galla JD, Spielvogel D, Ergin MA, Griepp RB. Acute type B aortic dissection: Surgical therapy. Ann Thorae Surg 2002:74:SI833-1835.
15. Coselli JS, LeMaire SA, de Figueiredo LP, Kirby RP. Paraplegia after thoracoabdominal aortic aneurysm repair: is dissection a risk factor? Ann Thorac Surg 1997;63:28-35.
16. Le Maire SA, Koksoy C, Schmittling ZC, Miller CC III, Oberwalder PJ, Curling PF, Coselli JS. Cerebrospinal fluid drainage during thoracoabdominal aortic aneurysm repair prevents spinal cord ischemia. Ann Thorac Surg 2000;70:1790.
17. Estrera AL, Sheinbaum R, Miller CC, Azizzadeh A, Walkes JC, Lee TY, Kaiser L, Safi HJ. Cerebrospinal fluid drainage during thoracic aortic repair: safety and current management. Ann Thorac Surg 2009;88:9-15.
18. Estrera AL, Rubenstein FS, Miller CC 3rd, Huynh TT, Letsou GV, Safi HJ. Descending thoracic aortic aneurysm: surgical approach and treatment using the adjuncts cerebrospinal fluid drainage and distal aortic perfusion. Ann Thorac Surg 2001;72:481-486.
19. Kato M, Bai H, Sato K, Kawamoto S, Kaneko M, Ueda T, Kishi D, Ohnishi K. Determining surgical indications for acute type B dissection based on enlargement of aortic diameter during the chronic phase. Circulation 1995;92:II107-112.
20. Chang CP, Liu JC, Liou YM, Chang SS, Chen JY. The role of false lumen size in prediction of in-hospital complications after acute type B aortic dissection. J Am Coll Cardiol 2008;52:1170-1176.
21. Cambria RP, Brewster DC, Gertler JG, Moncure AC, Gusberg R, Tilson MD, Darling RC, Hammond G, Mergerment J, Abbott WM. Vascular complications assoiated with spontaneous aortic dissection. J Vasc Surg 1988;7:199-209.
22. Fann JI, Sarris GE, Mitchell RS, Shumway NE, Stinson EB, Oyer PE, Miller DC. Treatment of patients with aortic dissection presenting with peripheral vascular complications. Ann Surg 1990;212:705-713.
23. Borst HG, Laas J, Heinemann M. Type A aortic dissection: diagnosis and management of malperfusion phenomena. Semin Thorac Cardiovasc Surg 1991;3:238-241.
24. Shimshak TM, Giorgi LV, Hartzler GO. Successful percutanous transluminal angioplasty of on obstructed abdominal aorta secondary to a chronic dissection. Am J Cardiol 1998;61:486-487.
25. Walker PJ, Dake MD, Mitchell RS, Miller DC. The use of endovascular techniques for the treatment of complications of aortic dissection. J Vasc Surg 1993;18:1042-1051.
26. Williams DM, Brothers TE, Messina LM. Relief of mesenteric ischemia in type III aortic dissection with percutaneous fenestration of the aortic septum. Radiology 1990;174:450-452.
27. Slonim SM, Nyman U, Semba CP, Miller DC, Mitchell RS, Dake MD. Aortic dissection: percutaneous management of ischemic complications with endovascular stents and balloon fenestration. J Vasc Surg 1996;23:241-251.
28. Vedantham S, Picus D, Sanchez LA, Braverman A, Moon MR, Sundt T 3rd, Kim P, Rubin B, Sicard GA. Percutaneous management of ischemic complications in patients with type-B aortic dissection. J Vasc Interv Radiol 2003;14:181-194.
29. Williams DM, Lee DY, Hamilton BH, Marx MV, Narasimham DL, Kazanjian SN, Prince MR, Andrews JC, Cho KJ, Deeb GM. The dissected aorta: percutaneous treatment of ischemic complications-principles and results. J Vasc Interv Radiol 1997;8:605-625.
30. Eggebrecht H, Nienaber CA, Neuhäuser M, Baumgart D, Kische S, Schmermund A, Herold U, Rehders TC, Jakob HG, Erbel R. Endovascular stent-graft placement in aortic dissection: a meta-analysis. Eur Heart J 2006;27:489-498.
31. Tsai TT, Fattori R, Trimarchi S, Nienaber C, Myrmel T, Hutchison S, Sechtem U, Cooper JV, Fang J, Pape L, Januzzi J, Eagle KA, Isselbacher E. Long-term survival in patients with type B acute aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). Circulation 2005; 112:535.
32. Bortone AS, De Cillis E, D'Agostino D, de Luca Tupputi Schinosa L. Endovascular treatment of thoracic aortic disease: four years of experience. Circulation 2004;110:II262-267.
33. Xu SD, Huang FJ, Yang JF, Li ZZ, Wang XY, Zhang ZG, Du JH. Endovascular repair of acute type B aortic dissection: early and mid-term results. J Vasc Surg 2006;43:1090-1095.
34. Nathanson DR, Rodriguez-Lopez JA, Ramaiah VG, Williams J, Olsen DM, Wheatley GH, Diethrich EB. Endoluminal stent-graft stabilization for thoracic aortic dissection. J Endovasc Ther 2005;12:354-359.