29 Complicated and Uncomplicated Acute Type B Aortic Dissection: Definitions and Approach in the Light of IRAD and INSTEAD

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Abstract

Type B aortic dissection is defined by an entry tear beyond the origin of the left subclavian artery. The acute phase is determined by the length of 2 weeks. The overall hospital mortality of this disease is in the 12–15 % range. In uncomplicated type B aortic dissections a primarily conservative approach with aggressive antihypertensive therapy is favored, which is supported by the INSTEAD trial. Aortic rupture and malperfusion (kidney, limbs, visceral organs, spinal cord) are the most important contributors to mortality and give the disease a "complicated" course. Risk factors such as aortic expansion, recurrent pain, refractory pain/hypertension, and periaortic hematoma are present in patients with a complicated course which increases hospital mortality to a 20 % rate. Interventional therapy (fenestration, stenting, stentgraft placement) as well as surgical therapy in combination with optimal medical therapy are options for patients with complicated type B aortic dissections.

Keywords

Aortic dissection • Descending aorta • Type B aortic dissection • Complicated aortic dissection • Uncomplicated aortic dissection

Introduction

Acute Type B Dissection is defined as acute aortic dissection affecting the descending aorta—with potential extension to the abdominal aorta—with an entry tear beyond the origin of the left subclavian artery. The phase of acute aortic dissection was defined in IRAD as 14 days following the onset of the disease [1]. The disease has the potential of a complicated or uncomplicated course, both requiring distinct therapeutic pathways.

Expansion of dissection may occlude a branch vessel or cause a rupture of the vessel wall. Complications include aortic rupture, limb ischemia malperfusion of kidneys or other visceral organ systems, persistent or recurrent intractable pain, progression of dissection, aneurysm

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expansion, and uncontrolled hypertension [2]. IRAD shows that definitions of complicated type B aortic dissection have changed over time. This is on the one hand due to an increased therapeutic armamentarium (esp. interventional strategies). On the other hand the knowledge about risk factors increased over time, and important risk factors such as refractory pain and/or hypertension emerged during the last decade of therapeutic management. Thus in the year 2002 the rate of intervention (which was surgery at that time) was 20 % and in a study of the year 2012 45 % of the patients were classified as complicated. Despite of all developments the early mortality in IRAD has not dramatically changed with 14.9 % in the year 2000 and 12.4 % in 2012 [1, 3].

Two distinct morphological subgroups of the IRAD cohort with acute type B aortic dissection were separately analysed: (1) Isolated abdominal aortic dissections and (2) Involvement of the aortic arch in the dissection process.

Isolated abdominal aortic dissection (below the diaphragm) was found in 18/532 (1.3 %) of patients with type B aortic dissection. In 11 % the etiology was iatrogenic (cardiac catheterization), whereas it was only 2 % in all type B aortic dissections ($p=0.07$). Two thirds of these patients were treated conservatively, five out of the remaining six patients underwent surgery. The hospital mortality in this subgroup of type B aortic dissections was 1/18 (6 %, in the conservative therapy group). The mean survival rate was 93 % at 1 year and 73 % at 5 years, and all of the patients who died during follow up were from the conservative therapy group. The authors conclude that aggressive surgical or endovascular management seems justified in patients with isolated abdominal aortic dissection [4].

In 2007 Tsai et al. investigated 127 out of 498 (26 %) patients with type B aortic dissection had the aortic arch involved in the dissection process. The long term follow-up analysis included 232 patients discharged alive from the referral centers with >80 % follow-up. The median follow-up time was 2.6 years. The hospital mortality was 12 %, without a significant difference between aortic arch involvement and no arch involvement. Interestingly there was no difference in the rate of neurological events whether the aortic arch was involved (12.6 %) in the dissection process or not (12.1 %, $p=0.88$). Furthermore, in patients who survived to hospital discharge, the mortality curves were similar in patients without aortic arch involvement with a 3-year follow-up mortality of 21 % versus 20 % in patients with AAI (log rank $P=0.82$) [5].

Uncomplicated Type B Aortic Dissections

An uncomplicated type B aortic dissection is defined by the absence of end-organ perfusion deficits, clinical symptoms, and morphologically progressive aneurismal disease.

Uncomplicated type B aortic dissections are primarily treated by medical therapy. This approach is supported by a landmark study in this field: The INSTEAD trial (Investigation Of Stent Grafts in Patients with type B Aortic Dissection) is a prospective randomized study where 68 patients were treated with optimal medical therapy and 72 patients underwent TEVAR. All 140 patients had a chronic (>2 weeks) uncomplicated type B aortic dissection. After 2 years the survival was 96 % with medical therapy and 89 % with TEVAR ($p=0.15$), paraplegia/paraparesis occurred in 1.4 % with medical therapy and 2.8 % with TEVAR ($p=0.90$), and major stroke occurred in 0 % with medical therapy and 2.8 % with TEVAR ($p=0.53$).

However, lifelong follow up of these patients is required. In the INSTEAD trial 22 % of patients of the conservative therapy group needed intervention or surgery within 2 years. The major pathology for the secondary complicated course was aortic expansion >60 mm, whereas only one case (2 %) of late malperfusion occurred.

With regard to the best medical therapy IRAD data found that beta-blocker and calcium channel blocker intake was associated with a better long term survival, whereas angiotensin converting enzyme inhibitors or diuretics did not show a benefit [6].

Complicated Type B Aortic Dissections

Complicated type B aortic dissections remain a challenging therapeutic problem. The International Registry of aortic dissections (IRAD) provides a large number of patients and a multi institutional scientific gain of knowledge. Important contributions have been made for the definition of complicated type B aortic dissection:

In 2003 IRAD outcome data on 384 acute type B aortic dissections out of 1,007 patients with aortic dissection (38 %) were published. In this series the hospital mortality was 13 %, with 85 % of hospital deaths occurring during the first week of stay. The most common causes of hospital death were rupture in 70 %, visceral ischemia in 19 %, and neurological causes in 8 %. This study found the "deadly triad" as independent risk factors for hospital mortality in acute type B aortic dissection: First the absence of chest/back pain which occurred in 14 % of patients, second hypotension/shock which occurred in 3 % of patients, and third a branch vessel involvement which occurred in 21 % of the patients. Other risk factors for hospital mortality were an aortic diameter ≥6 cm which occurred in 16 % of the patients, and coma/altered consciousness which occurred in 5 % of patients [2].

Henke et al. report on 26 out of 458 patients (6 %) with acute type B aortic dissection with acute limb ischemia. Acute limb ischemia was defined clinically as lack of peripheral pulses and concomitant limb symptoms such as pain and paresthesias. Patients with acute limb ischemia had a more complex disease with significantly increased rates of mesenteric ischemia/infarction (25 % vs 5 %, p = 0.001), acute renal failure (25 % vs. 11 %, p = 0.05), myocardial infarction (12 % vs. 2 %, p = 0.03), and stroke (12 % vs. 2 %, p = 0.01) compared with patients without acute limb ischemia. This resulted in a threefold hospital mortality of patients with acute limb ischemia (31 % with acute limb ischemia vs. 11 % without limb ischemia, p = 0.009) [7].

These data impressively show that rupture and malperfusion (visceral, renal, cerebral, spinal, limb) are contributing to a lethal course in patients with type B aortic dissection. Thus signs of a contained rupture such as periaortic hematoma or pleural hemorrhagic effusion, as well as any malperfusion, and progressive aneurismal dilatation which leads to a rupture are "classical" indications for intervention or surgery.

In 2006 Trimarchi et al. published the results of 82 out of 476 (17 %) patients with acute type B aortic dissection who underwent surgical therapy. The patients for this study were enrolled from January 1996 to April 2003, and at that time surgery was the main intervention for type B aortic dissection. In the surgery group the mean age was 60 years, 83 % had male gender, and 11 % Marfan syndrome. The indications for surgery were aortic rupture in 23 % of patients, visceral ischemia in 24 %, limb ischemia in 16 %, extension of dissection beyond the intimal tear compromising additional arteries in 52 %, recurrent pain in 27 %, refractory pain in 14 %, and untreatable hypertension in 16 %. In almost one third of patients, more than one indication for surgery was noted. The surgical procedures were: replacement of the descending aorta in 69 %, partial arch replacement in 21 %, complete arch replacement in 8 %, surgical fenestration in 9 %, surgical approaches to the peripheral vessels in 20 %, and visceral or iliac stenting in association with surgical procedures in 9 %. Hypothermic circulatory arrest was used in 48 % of patients. Interestingly the percentage of hypothermic circulatory arrest has higher in the patients who survived surgery than in the mortality group (55 % vs. 32 %, p = 0.07). The mean hypothermic circulatory arrest time was 40 min (range, 29–61), and it was significantly shorter in patients who survived surgery compared to patients who died in hospital (37 min versus 54 min, P = 0.01). Reoperation for bleeding was required in 7.1 %. The hospital mortality for patients undergoing surgery for acute type B aortic dissection was 29 %. The following risk factors were associated with hospital mortality: age >70 years, aortic rupture, partial thrombosis of the false lumen, periaortic hematoma at the diagnostic examinations, descending aortic diameter >6 cm, preoperative coma or altered consciousness, and preoperative shock/hypotension.

The postoperative complications were: new neurological deficits in 23 %, cerebrovascular accident in 9 %, coma in 7 %, and paraplegia in 4.5 %, visceral ischemia/infarction in 7 %, acute renal failure in 18 %, hypotension/shock in 21 %, cardiac tamponade in 3 %, and limb ischemia in 3 % [8].

An actual study examined the aortic expansion following acute type B aortic dissection. In 191 patients, who survived the initial hospitalization and who were treated medically imaging studies were available at distinct intervals of 6, 12, and 24 months after onset of the disease. Aortic expansion during a median follow-up time of 2 years was observed in 59 % of patients. The mean annual expansion rate was 1.7 ± 7.6 mm/year. Risk factors for aortic expansion were white race and male gender whereas the intake of calcium channel blockers was protective against aortic expansion. Interestingly, an initially increased aortic diameter was associated with a decreased annual aortic expansion rate. Overall, the expansion rate for patients with an initial aortic diameter of less than 4 cm was 3.8 mm/year, as compared with 0.2 mm/year for aortas of 4 cm or larger ($p = 0.001$). The authors of the study discuss that patients with relatively normal aortic measurements exhibiting aortic dissection may have more severe aortic wall abnormalities, possibly caused by connective tissue disorders, as compared with those experiencing aortic dissection in enlarged aortas. The poor condition of the aortic wall in these patients with small aortic diameters may theoretically also result in increased risks of aortic enlargement during follow-up after dissection. Unfortunately, the IRAD does not contain data regarding connective tissue disorders other than Marfan syndrome which was no risk factor for aortic expansion within 2 years in this study. However, a selection bias may have affected the results of the analysis, because large dissected aortas generally more often undergo aortic intervention, and therefore more frequently may have been excluded from analysis. Another finding of this study was that presence of a patent false lumen showed a strong trend towards increased aortic expansion with 3.3 mm/year compared with absence of a patent false lumen with aortic expansion rate of 0.9 mm/year ($p = 0.063$) [9]. In this study partial false lumen thrombosis was no risk factor for aortic expansion. This is in contrast to the findings published in 2007 by Tsai et al. who found partial false lumen thrombosis (defined as both flow and thrombus in the false lumen) a risk factor for mortality in the long term. In this study the false lumen was found to be patent in 114 patients (56.7 %), partially thrombosed in 68 (33.8 %), and completely thrombosed in 19 (9.5 %) patients. The mortality rate was highest in patients with partial false lumen thrombosis, with a 3-year mortality rate of 32 %, versus 14 % in patients with a patent false lumen and 23 % in patients with complete thrombosis of the false lumen (overall $p = 0.003$). It is, however, important to know that there was a significant difference of age between the three groups with a mean age of 63 years for partial thrombosis, 58 years for patent lumina, and 71 years for the group with complete thrombosis ($p < 0.001$) [10].

In 2010 another IRAD study revealed that refractory pain and/or hypertension was an independent risk factor of hospital mortality in patients with acute type B aortic dissection. Patients with refractory pain and/or hypertension as a sole complication of the aortic dissection had a 17 % hospital mortality, and completely uncomplicated type B aortic dissections showed a mortality of 4 % ($p = 0.0003$) [11].

In 2012 IRAD data compared patients with complicated versus uncomplicated type B aortic dissections. Complicated dissections were defined as: presence of shock, periaortic hematoma, spinal cord ischemia, mesenteric ischemia/infarction, acute renal failure, limb ischemia, recurrent pain, refractory pain or refractory hypertension. Out of 550 patients 46 % were classified as complicated. With regard to demographical data Marfan syndrome, and hypertension were more frequently found in complicated cases, whereas previous coronary revascularization was more frequent in the uncomplicated cases. Interestingly age was not different between the two groups. Complicated type B aortic dissections exhibited a larger mean descending aortic diameter compared with uncomplicated cases (4.8 cm vs. 4.4 cm, $p = 0.02$). In this study neither

the rate of partial false lumen thrombosis (39 % vs. 38 %, p = 0.74) nor the rate of complete false lumen thrombosis (11 % vs. 16 %, p = 0.15) was different between complicated and uncomplicated cases. The overall hospital mortality in this series was 12.4 %, with 20 % in complicated cases and 6.1 % in uncomplicated cases (p < 0.01). Independent risk factors for hospital mortality were age >70 years, preoperative limb ischemia, and periaortic hematoma. The management of the complicated type B aortic dissections was surgery in 28 %, medical therapy in 44 %, percutaneous intervention in 28 %, stenting in 26 %, and fenestration in 7 %. The hospital mortality of complicated cases treated by surgery was 28.6 %, it was 20.7 % in cases with medical therapy, and 10.1 % in patients with percutaneous intervention. There is, however a strong selection bias. The authors of the study state that patient with aortic rupture and shock were usually submitted to surgery. Thus 45 % of aortic ruptures in this cohort were in the surgical group. Furthermore surgery is the last therapeutic option if both medical therapy and intervention fail. The IRAD authors conclude that "patients with such extreme conditions identify a cohort which, regardless of treatment, have a very high mortality" [3].

Long Term Follow Up

In 2006 midterm follow up data were published from 317 out of 532 patients (60 %) with acute aortic type B dissection from the referral centers with >80 % follow-up. The hospital mortality was 12 % and the remaining 242 comprised the study population. The median follow-up time was 2.3 years. The unadjusted survival rate at 3 years for patients discharged from the hospital alive according to in hospital therapy was 78 % for medical therapy alone, 83 % for surgery, and 76 % for endovascular treatment (log-rank P = 0.63). Significant independent predictors of follow-up mortality were: female gender, a history of prior aortic aneurysm, a history of atherosclerosis, in-hospital renal failure, pleural effusion on chest radiograph, and in-hospital hypotension/shock [12].

References

1. 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(7):897–903.
2. Suzuki T, Mehta RH, Ince H, Nagai R, Sakomura Y, Weber F, Sumiyoshi T, Bossone E, Trimarchi S, Cooper JV, Smith DE, Isselbacher EM, Eagle KA, Nienaber CA, International Registry of Aortic Dissection. 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.
3. Trimarchi S, Tolenaar JL, Tsai TT, Froehlich J, Pegorer M, Upchurch GR, Fattori R, Sundt 3rd TM, Isselbacher EM, Nienaber CA, Rampoldi V, Eagle KA. Influence of clinical presentation on the outcome of acute B aortic dissection: evidences from IRAD. J Cardiovasc Surg (Torino). 2012;53(2):161–8.
4. Trimarchi S, Tsai T, Eagle KA, Isselbacher EM, Froehlich J, Cooper JV, Rampoldi V, Upchurch Jr GR, International Registry of Acute Aortic Dissection (IRAD) investigators. Acute abdominal aortic dissection: insight from the International Registry of Acute Aortic Dissection (IRAD). J Vasc Surg. 2007;46(5):913–9.
5. Tsai TT, Isselbacher EM, Trimarchi S, Bossone E, Pape L, Januzzi JL, Evangelista A, Oh JK, Llovet A, Beckman J, Cooper JV, Smith DE, Froehlich JB, Fattori R, Eagle KA, Nienaber CA, International Registry of Acute Aortic Dissection. Acute type B aortic dissection: does aortic arch involvement affect management and outcomes? Insights from the International Registry of Acute Aortic Dissection (IRAD). Circulation. 2007;116(11 Suppl):I150–6.
6. Suzuki T, Isselbacher EM, Nienaber CA, Pyeritz RE, Eagle KA, Tsai TT, Cooper JV, Januzzi Jr JL, Braverman AC, Montgomery DG, Fattori R, Pape L, Harris KM, Booher A, Oh JK, Peterson M, Ramanath VS, Froehlich JB, IRAD Investigators. Type-selective benefits of medications in treatment of acute aortic dissection (from the International Registry of Acute Aortic Dissection [IRAD]). Am J Cardiol. 2012;109(1):122–7.
7. Henke PK, Williams DM, Upchurch Jr GR, Proctor M, Cooper JV, Fang J, Nienaber CA, Isselbacher EM, Fattori R, Dasika N, Gemmete J, Stanley JC, Wakefield TW, Eagle KA. Acute limb ischemia associated with type B aortic dissection: clinical relevance and therapy. Surgery. 2006;140(4):532–9.
8. Trimarchi S, Nienaber CA, Rampoldi V, Myrmel T, Suzuki T, Bossone E, Tolva V, Deeb MG, Upchurch Jr GR, Cooper JV, Fang J, Isselbacher EM, Sundt 3rd TM, Eagle KA, IRAD Investigators. Role and results

of surgery in acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). Circulation. 2006;114(1 Suppl):I357–64.

9. Jonker FH, Trimarchi S, Rampoldi V, Patel HJ, O'Gara P, Peterson MD, Fattori R, Moll FL, Voehringer M, Pyeritz RE, Hutchison S, Montgomery D, Isselbacher EM, Nienaber CA, Eagle KA, International Registry of Acute Aortic Dissection (IRAD) Investigators. Aortic expansion after acute type B aortic dissection. Ann Thorac Surg. 2012;94:1223–9.
10. Tsai TT, Evangelista A, Nienaber CA, Myrmel T, Meinhardt G, Cooper JV, Smith DE, Suzuki T, Fattori R, Llovet A, Froehlich J, Hutchison S, Distante A, Sundt T, Beckman J, Januzzi Jr JL, Isselbacher EM, Eagle KA, International Registry of Acute Aortic Dissection. Partial thrombosis of the false lumen in patients with acute type B aortic dissection. N Engl J Med. 2007;357(4):349–59.
11. Trimarchi S, Eagle KA, Nienaber CA, Pyeritz RE, Jonker FH, Suzuki T, O'Gara PT, Hutchinson SJ, Rampoldi V, Grassi V, Bossone E, Muhs BE, Evangelista A, Tsai TT, Froehlich JB, Cooper JV, Montgomery D, Meinhardt G, Myrmel T, Upchurch GR, Sundt TM, Isselbacher EM, International Registry of Acute Aortic Dissection (IRAD) Investigators. 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.
12. Tsai TT, Fattori R, Trimarchi S, Isselbacher E, Myrmel T, Evangelista A, Hutchison S, Sechtem U, Cooper JV, Smith DE, Pape L, Froehlich J, Raghupathy A, Januzzi JL, Eagle KA, Nienaber CA, International Registry of Acute Aortic Dissection. Long-term survival in patients presenting with type B acute aortic dissection: insights from the International Registry of Acute Aortic Dissection. Circulation. 2006;114(21):2226–31.