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Management of Uncomplicated Stanford Type B Aortic Dissection: A Literature Review

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Introduction

Type B aortic dissections are approximately 33% of all dissection cases. Clinically, type B aortic dissection procedure can be divided into the complicated and uncomplicated dissection. Complicated aortic type B dissections are patients with one of the symptoms: hypotension, shock, malperfusion, acute renal failure, hypertension and retractable pain, aortic rupture, and aortic dilatation, an increase in the size of dissection. An Uncomplicated type B aortic dissection are patient without these symptoms during an in-hospital stay. This classification determines the prognosis and survival of patients ongoing treatment for complicated aortic dissection, which is lower (50%) compared to the uncomplicated (90%) dissection.[1,2]

Hypertension is more prevalent in type B aortic dissection (70%)[1]; therefore, the primary treatment of type B aortic dissection is a therapy that is aimed at lowering blood pressure and cardiac output. Pharmacological therapy alone can increase the survival rate per year by ≥80%, but with a high long-term morbidity rate (25%-50%). In comparison, open surgical therapy increases mortality by 30% instead of pharmacological therapy (10%). Thus, pharmacological therapy is preferable to treat an uncomplicated type B aortic dissection.[3,4] Traditionally, Thoracic Endovascular Aortic Repair (TEVAR) is only for complicated dissection. However, in the last few decades, with the advancement of imaging modality and minimally invasive endovascular intervention, the paradigm for managing uncomplicated type B aortic dissection has gone through many changes.[5]

Globally, the incidents are estimated at 3 per 100,000 lives.[2] Meanwhile, the cases of type B aortic dissection at the National Center General Hospital dr. Cipto Mangunkusumo (RSCM) is found between 1 to 2 times per year. Though relatively infrequent, the mortality is quite high. Thus, a review of the current management of uncomplicated descending aortic dissection is required.

Classification

Acute Aortic Dissection (IRAD) is classified as four, i.e., hyperacute, acute, subacute, and chronic. The hyperacute referred to 0-24 hours, acute 2-7 days, subacute 8-30 days, and chronic ≥30 days. The division is related to the loss of elasticity of the intima septum as time progresses, and the success rate for manipulating dissection decreases. Moreover, the cumulative survival rate continues to lower across the four-time groups despite the therapeutic modality: 94-99%, 82-93%, 77-92%, and 73-91%. This division can help decide for early and late treatment in aortic dissection cases.[6]

Another classification of type B aortic dissection is complicated and uncomplicated, related to the presence of complication. Complicated type B dissection only represents 15-20% of cases; however, the mortality rate reaches 50% in this group. Meanwhile, the mortality rate of uncomplicated type dissection is only 10%. Complications of type B dissection include hemorrhaging, untreatable pain, uncontrollable hypertension, dissection enlargement, limb and visceral organ malperfusion, and spinal cord ischemia. Meanwhile, uncomplicated aortic dissection means a stable patient who shows none of those complications during the presentation and admission.[6]

Therapy in the management of aortic dissection

Medical therapy plays an essential role in managing aortic dissection, both in acute and chronic cases. The therapy's primary goal is to decrease the friction and pressure on the affected areas by lowering blood pressure and cardiac contractility.[7] The target for systolic blood pressure is 100-120 mmHg, which is called permissive hypotension. The therapies to lower the blood pressure are beta-blockers, angiotensin-converting enzyme inhibitors, or intravenous angiotensin receptor blockers for acute dissection.[2]

For patients with comorbidities such as diabetes or chronic kidney failure, the blood pressure target is lower than 130/80mmHg, decreasing heartbeat with a target of 60 beats per minute, and statin administration to achieve LDL cholesterol levels the blood of below 70mg/dL and stop smoking.[8,9] For chronic phases, the goal of medical therapy is to restrain the dissection enlargement, control blood pressure below 140/90 mmHg, change lifestyle, and antihypertensives if needed.[7] The administration of statins, calcium channel blockers, and renin-angiotensin inhibitors can prevent complications and avoid aortic enlargement.[2]

Open surgery

Open surgery on aortic dissection is related to morbidity rate (paraplegia 30-36%) and high mortality (29-50%). For type B aortic dissection, complications, and the highest mortality of open surgery are at the acute phase. The mortality rate depends on the patient's age and organ

dysfunctions, primarily the renal and visceral, caused by ischemia.[2,11] The goal of open surgery on aortic dissection is to charge up and replace dissected segments. Open surgery tends to be left out due to its high mortality rate, and pharmacological therapy alone can lower the chance of morbidity and mortality.[4,5,11]

Hybrid technique

Open surgery technique requires cardiopulmonary bypass and stopping circulation using hypothermia to identify and cut the intima tear, and then the re-approximation of intima and adventitia. This technique still leaves residue and patented false lumen on about 50% of the patients. This hybrid technique is a combination of surgery and endovascular, which is expected to resolve the challenges of open surgery. This technique is recommended on acute type A aortic dissection with malperfusion. The hybrid technique for type B aortic dissection is the revascularization of supra-aortic branches/TEVAR and stent graft repair without surgery (stenting/fenestrated/branched TEVAR).

The revascularization of supra-aortic branches is followed by endovascular stent graft on an aortic arch, with or without TEVAR procedure on the descending aorta. Meanwhile, the stent-graft repair technique has three types of the stent that can be customized, while the endovascular treatment can be done during surgery (branch cutting) or after surgery. This hybrid technique poses similar risks to open surgery, with a mortality rate on 30 days of 30%. The long-term outcome remains unknown, but this technique may be an alternative for patients who cannot undergo open surgery and endovascular intervention.[12,13]

Endovascular treatment

In 1999, TEVAR was an option for the management of type B aortic dissection but, in 2008, it became the standard for complicated type B aortic dissection. TEVAR has shown its superiority in managing complicated type B aortic dissection compared to open surgery as it lowers the mortality rate significantly on the first 30 days from 29.3% to 2.8%. The TEVAR procedure benefits are that it can be performed on patients that don't fit the criteria for surgery, has a short procedure time, causes much less bleeding compared to open surgery, and faster recovery time. The main goal of TEVAR on aortic dissection is to stop blood flow to the false lumen by blocking the entrance tear and redirecting the blood flow to the aortic lumen. The false lumen will gradually close up and prompts the thrombosis to recover the aorta.[2,13]

Some notes need to be taken into consideration in the management of aortic dissection are the time frame (acute, subacute, and chronic), the location of the intima tear, the size of the aorta, the extent of the segment of the aortic involvement, the presence of complications and false lumen status (patent, partial or complete thrombosis). A classification is developed to keep up with the endovascular action development, called DISSECT classification system. This classification identifies six characteristics that influence the choice of therapy, particularly for endovascular procedures. Characteristics that are considered necessary include duration, intimal tear, size of the aorta, the segmental extent of involvement, clinical complications, and thrombosis of the false lumen. This classification system makes it easy for practitioners to image the anatomical features that are considered necessary in deciding the treatment of patients with aortic dissection.[14]

One of the complications that often occur after TEVAR is endoleak. Endoleak is defined as a continuous flow of blood that "leaks" into the saccus aneurisma but outside the graft endoluminal. Endoleak occurs in 10-40% (average 26%) of patients undergoing TEVAR. If undetected, the endoleak can progress and cause the expansion of the saccus aneurisma to rupture; the rupture can be fatal. Endoleak is classified into four types based on the source of the leak between the systemic circulation and the saccus aneurisma. Type I endoleak is divided into type Ia if the leak comes from the endograft's proximal end and Ib if the leak originates from the distal end of the endograft. Endoleak type I is a type of endoleak which is very dangerous and requires immediate intervention. Endoleak type II is the most common endoleak and occurs when there is retrograde blood flow from patent collateral vessels to the saccus aneurisma. Intervention in type II endoleak is carried out when expanding the saccus aneurisma by more than 5 mm. Endoleak type III is divided into IIIa (endograft modular component leak) and IIIb (endograft fabric leak). Type IV endoleak is the mildest endoleak and can heal itself without additional treatment, only involving an increase in the endograft fabric's porosity. Type V endoleak is an endoleak that is indeterminate with an unidentifiable source of leakage. The management protocol for type V endoleak is unclear but generally requires intervention. Type I and III endoleak are defined as high-flow endoleak and have a high risk of causing saccus aneurysmal rupture. Endoleak type II is defined as endoleak low-flow. If left untreated, the endoleak type can lead to large aortic aneurysms that can rupture and cause heavy bleeding.[19,20,21]

Based on the study of validity, importance, and ability in the application of literature, it was found that five studies were suitable and met the rules of study in answering research questions.

The study by Afifi (2015) published a retrospective cohort study in patients with acute type B aortic dissection (ATBAD) who received pharmacological therapy and opened surgical intervention.[22] This study was conducted to analyze the outcome in type B acute aortic dissection after treatment. All ATBAD patients are divided into complicated and uncomplicated according to clinical and radiological conditions. The therapeutic modalities were pharmacology, open surgery, endovascular intervention, and peripheral vascular bypass. This study was conducted for thirteen years at Texas Houston Medical School, from January 2001 to June 2014. All uncomplicated and complicated ATBAD patients received pharmacological therapy. The pharmacological treatment goal was to lower systolic blood pressure between 100 and 120mmHg with early symptoms resolution. All patients were subjected to CT or MRI scans of the chest, abdomen, and pelvis before discharge to assess for aortic enlargement. Early death was defined as death within 30 days of admission.

The early mortality rate was 7.6% (34 patients), with premature death in uncomplicated ATBAD (2.6%, seven patients). There were readmissions of 20.3%, namely 101 patients with a median time of 3.5 years for the uncomplicated ATBAD group. Of the 101 patients who underwent readmissions, 12 (11.8%) patients presented with acute or chronic type A dissection; 7 of them were uncomplicated ATBAD patients. In the uncomplicated group, 40 patients (15%) required reintervention. Partially due to hemothorax evacuation, open aortic aneurysm surgery, and type A aortic dissection repair. The mean follow-up time was 4.6 years. The survival rates without intervention in the uncomplicated groups at one year and five years were 84.8% and 62.7%, respectively. The uncomplicated group's overall survival rates at years 1, 5, and 10 were 91%, 76.6%, and 66.7%.

The study by Brunkwall (2014) published research comparing best medical treatment (BMT) and BMT + intervention endovascular stent graft Gore Tag in patients with uncomplicated acute aortic dissection.[23] Acute Dissection: Stent graft OR Best medical therapy (ADSORB) trial is a prospective randomized study. The study was conducted in 17 European centers from December 2008 to December 2010. All patients received BMT therapy and were randomized to receive either BMT

alone or BMT and Gore tag stents. BMT therapy is aimed at lowering blood pressure to <120/80mmHg and close monitoring. The outcome of this study assessed the status of thrombosis in the false lumen (incomplete, complete, no thrombosis) and dilatation or rupture of the aorta for one year. The assessment was carried out before therapy, three months, and one year after the complaint about maximum actual diameter, false lumen, and the transverse section's aortic diameter as a whole. Dissection-related mortality was defined as death within 30 days after therapy, during treatment, within 30 days after the endovascular intervention, or due to aortic dissection.

The BMT administration in the two groups was not much different; on average, they required four or more hypertension drugs. Patients given the BMT + Gore Tag stent received intervention within 1-14 days after symptoms. Incomplete, false lumen thrombosis was found in 13 patients (43%). BMT + Gore Tag and 30 patients (97%) from the BMT group ($p < 0.001$). Aortic dilatation was observed in 11 patients (37%) in the BMT + Gore tag group and 14 patients (45%) in the BMT group. There was no aortic rupture in either group, or there was one case of death with myocardial infarction, but it was not considered a dissection-related criterion.

Nienaber (2009) showed the effect of endovascular stent graft as an adjunct to pharmacological therapy.[15] The study was conducted in 7 German, Italian, and French medical centers between November 2003 and 2005. Uncomplicated type B aortic dissection patients of 2- 52 weeks were randomized to receive TEVAR therapy and optimal medical therapy (OMT) or OMT only. The instrument that was used was a TALENT stent graft (Medtronic, Inc, Santa Rosa, Calif) that was adjusted to the anatomy of each patient. Imaging evaluation was carried out at three months, one year, and two years after intervention. Patients with an aortic diameter of ≥6cm or with acute complications and anatomical abnormalities which could not undergo TEVAR >75° or had complete false lumen thrombosis were excluded from this study. After a 14-day observation period to see if there were any acute complications or the occurrence of spontaneous false lumen thrombosis, the patients were then offered randomization. One hundred forty patients met the criteria; 72 patients received medical and endovascular therapy, while 68 received medical therapy alone. The mean period from the complaint to randomization was 39 and 45 days (beginning of the chronic phase). The mean time from randomization to the endovascular intervention was 12 days (1 to 29 days). The TEVAR intervention was successfully performed in 70 patients without open surgical conversion. One stent-graft was placed in 58 patients (82.9%), 2 stent-grafts in 8 patients (11.4%), and 3 stent-grafts in 4 patients (5.7 %).

The outcome in 30 days was three vascular injuries requiring additional procedures, three neurological complications (one paraplegia, one transient paraparesis with left subclavian artery occlusion, and one stroke). Although most (74%) patients proceeded with ICU care for ± 24 hours, the mean hospital stay in the TEVAR group was eight days. In addition, a blood pressure of ≤120/80mmHg was achieved in all patients after one month of randomization and during outpatient visits. In the 2-year outcome, aortic dilation >60mm was more prevalent in the OMT group, which was then treated with TEVAR (16.2%) or to open surgery (4.4%). There were also three cases of spinal injury ischemia complications: two in the TEVAR group and one in the OMT group. All patients who were previously in the OMT group and then underwent the TEVAR intervention showed promising outcomes, without death and aortic remodeling. The thrombosis process in false lumen increased after stent-graft placement, with 91.3% complete false lumen thrombosis and accompanied by aortic remodeling ($p < 0.001$). On the other hand, OMT administration alone did not show the process of aortic remodeling and deflating the false lumen; false lumen thrombosis only occurs in a few cases.

The study by Nienaber (2013) carried out an extension of the INSTEAD trial to evaluate the long-term outcome of patients with uncomplicated type B aortic dissection who were treated with TEVAR and OMT and previously administered OMT therapy alone.[24] The endpoint at five years assessed all causes of death — deaths associated with aortic disease (rupture, malperfusion, proximal dissection or death within an hour of onset of symptoms, symptomatic patients without coronary disease or heart valve disease) and the development of aortic pathology (assessed by the incidence rate of open conversion or TEVAR group), endovascular measures or additional surgery, enlargement of the aorta >5.5cm and the remodeling process. Patients were monitored until September 30, 2010 (minimum five years, maximum eight years), and imaging at five years was obtained in 103 patients. Clinical monitoring showed that systolic blood pressure could be reduced to ≤130 mm Hg with OMT in 90% of patients. During the five years of monitoring, 93 TEVAR interventions were carried out. In patients who received OMT alone, there were 14 cases (five emergencies) that subsequently required TEVAR and four open surgery cases due to enlargement of the false lumen. In the TEVAR group, seven cases were requiring additional TEVAR intervention and three open surgery. At the end of 2010, twenty-seven patients had undergone aortic repair procedures, and 117 patients were survived.

The mortality of >5 years tended to be lower in patients receiving TEVAR and OMT compared to OMT alone (11.1 ± 3.7% versus 19.3 ± 4.8%; $p = 0.13$). The benefit of TEVAR at survival rates was seen between 2 and 5 years (100% versus 83.1 ± 4.7%; $p = 0.0003$), although at two years follow-up was not very significant (88.9 ± 3.7% versus 97.9 ± 2.0%; hazard ratio, 3.96; 95% CI, 0.84–18.6; $p = 0.082$). In addition, cases requiring further treatment (TEVAR or open surgery conversion) were more common in the OMT group than in TEVAR.

All patients previously in the OMT group who experienced rupturing during follow-up had a tear of >10mm (14 ± 4 mm), similar to patients who crossed into critical expansion (13 ± 4 mm). Meanwhile, on the TEVAR group, re-intervention rates were required in two cases. False lumen thrombosis was present in 90.6% with an aortic remodeling rate of 79.2% at year five after TEVAR. In contrast, OMT alone failed to demonstrate actual lumen enlargement or false lumen deflation but resulted in the aorta's enlargement from 43.6 ± 9.2 to 56.4 ± 6.8 mm ($p < 0.0001$).

The study by Wang (2019) published a retrospective analytical research to assess the effectiveness of TEVAR for uncomplicated type B aortic dissection by evaluating the 30-day outcome for patients with acute (<30 days) and chronic dissections who were divided into the complicated and uncomplicated aortic dissection.[25] Uncomplicated patients were then differentiated according to the time interval from symptom onset to receiving TEVAR therapy according to the IRAD distribution; ≤48 hours,> 48 hours to <7 days, ≥7 days to ≤14 days, and> 14 days to <30 days.

The TEVAR procedure successfully closes the entrance tears in 98% of acute patients and 99% of chronic patients. The 30-day mortality rate outcome was 7.3%, with a higher trend for acute versus chronic dissection (9.3% versus 5.2%; $p = 0.126$). Complications of procedure-related bone marrow ischemia occurred in 4.4% of acute dissection patients and in 2.1% of chronic dissection patients ($p = 0.261$), with a neurological deficit of 3.4% in acute dissection and 0.5% in chronic dissection ($p = 0.068$). Paralysis incidence due to stroke occurred in 2.5% of acute dissection patients and 1.6% in chronic dissection (p =

0.725). Type A retrograde dissection occurred in 1.1% of acute dissection patients and in 2.6% of chronic dissection patients (p = 0.412). There was also a lower free reintervention rate in acute dissection than in chronic dissection (90.7% versus 94.8%; p = 0.13). In acute dissection, rapid aortic enlargement was more frequent in the group ≥7 days to ≤14 days and >14 days to <30 days compared with that given therapy within seven days of onset of symptoms (p = 0.042). The 30-day reintervention rate for uncomplicated acute dissection was 5.8% at all time intervals.

There were five selected studies found in PubMed and Cochrane Library, four studies comparing the pharmacological therapy and TEVAR with pharmacological therapy alone in uncomplicated type B aortic dissection patients. One study comparing TEVAR in acute and chronic type B aortic dissection. To date, pharmacological therapy remains the primary treatment for uncomplicated type B aortic dissection. In the study by Afifi et al., pharmacological therapy alone can prolong long-term survival by 50-70%. The study also states that patients of uncomplicated type B aortic dissection receiving pharmacological therapy alone have an early mortality rate <2.2%, and those who survived to require further intervention at 15.3%.[22] In addition, in the ADSORB trial by Brunkwall et al., there was no early mortality at 30 days of monitoring uncomplicated dissection patients who only get pharmacological therapy. This study also states that at least three different antihypertensive drugs are needed to control this group's blood pressure.[23]

The INSTEAD trial by Nienaber et al. showed uncomplicated type B dissection patients who received pharmacological therapy alone had a reasonable survival rate (95.2 ± 2.5%) for up to two years. Those who had aortic enlargement >60mm who subsequently required intervention occurred at 22.1% of cases (15 of 68 patients), and only 1.4% (one patient) developed persistent paraplegia/paraparesis.[15] However, on the five-year observation of this study's extension INSTEAD-XL, the risk level of death due to all causes that were associated with the aortic and aortic disease progression was higher in patients receiving pharmacological therapy alone than in those receiving medicaments therapy and TEVAR, respectively: 11.1% versus 19.3%, 6.9% versus 19.3% and 27% versus 46.1%.[24]

Complete false lumen thrombosis is one of the indicators of a good prognosis. On the other hand, partial false lumen thrombosis is an indicator that can predict dilation of the aorta and eventually form an aneurysm. According to the ADSORB trial, patients receiving pharmacological therapy alone had a higher rate of incomplete, false lumen thrombosis at one year than patients receiving additional endovascular therapy (97% versus 43%), as well as aortic dilatation (45% versus 37%).[26] INSTEAD and the INSTEAD-XL trials also stated that the administration of pharmacological therapy alone failed to show significant signs of aortic healing and thrombus formation in the false lumen only in a minority of patients at two years of surveillance. Five years of partial thrombosis was still present in 78% of patients who received pharmacological therapy alone and aortic enlargement, which was seen in 66% of patients.
Conversely, the rate of false lumen thrombosis was increased by TEVAR intervention at 91.3% complete false lumen thrombosis, and signs of aortic remodeling at two years. At five years of observation, complete false lumen thrombosis was found in 90.6% of patients, and signs of aortic remodeling were found in 79.2% of patients after TEVAR. In addition, the long-term reintervention-free rate was lower in patients who had received TEVAR intervention.[15, 24]

The study by Wang et al. compared the outcome of 30 days at the TEVAR intervention based on the IRAD time classification. They found no significant difference between demographics, complaints, mortality, and complications. The 30-day reintervention rate in this study was 5.8% for acute dissection after early TEVAR, and no significant difference was found at any time of intervention. One case type A retrograde dissection in a case that underwent TEVAR intervention for <48 hours. However, in the ADSORB trial, the TEVAR procedure in uncomplicated type B dissection patients <2 weeks, the true lumen was enlarged, the false lumen was reduced, and the aortic diameter was reduced. In addition, in the INSTEAD-XL study, patients who received TEVAR were intervened with a median time of 12 days from randomization in patients with dissection who had been ≥14 days, reflecting the initial time of the chronic phase. This study revealed a reduced aortic specific mortality rate (6.9%) and a reduced disease progression rate (27%) in those receiving TEVAR.[23,24,25] Based on these data, TEVAR therapy is best performed at subacute time intervals; a procedure in the acute phase can be performed when the risk of complications is more significant, such as aortic rupture, but intervention in the acute phase carries a risk for retrograde dissection which may be due to the fragility of the blood vessels

Clinical Implication

Previously, the management of uncomplicated type B aortic dissection was only pharmacological therapy, and intervention was carried out when complications occurred, either endovascular or open surgery. Pharmacological therapy remains the mainstay of treatment for type B aortic dissection, with the primary aim of lowering and stabilizing systolic blood pressure <120mmHg. The current management of uncomplicated Stanford type B aortic dissection, based on the results of the literature search, found a trend in the use of endovascular therapy as a prevention against the incidence of complications of type B aortic dissection.

The management of TEVAR in particular cases of uncomplicated aortic dissection is a minimally invasive option with good results, reducing the early mortality rate to <2.2%, accelerating the occurrence of complete flagellum thrombosis (91.3%) at two years, and signs of the sign of aortic remodeling in 79.2% post-TEVAR at five years of observation. The results of the treatment of TEVAR in uncomplicated type B aortic dissection require further study

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