Acute type B aortic dissection: update on proper management

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ABSTRACT

This study aims to collect and present all current literature data on the diagnostic and therapeutic management of acute type B aortic dissection. It includes a comprehensive literature search utilizing the following keywords: 'acute aortic dissection', 'type B aortic dissection', 'conservative management', 'endovascular repair', 'open surgery' and 'diagnosis'. Uncomplicated acute type B aortic dissection can be effectively managed using conservative management, although open repair is indicated only for complicated cases. Endovascular repair shows promising results in selected patients with increased perioperative risk and without contraindications. Recent evidence supports endovascular repair even in uncomplicated cases, although more data on long−term outcomes are needed. Early risk stratification and evaluation of the patient is crucial for selection of optimal management.

1. Introduction

Acute aortic dissection (AAD) remains a potentially life−threatening condition that is followed by a 2−3 times higher risk for rupture compared to that of aneurysms[1]. Although aortic dissection is the most common etiology for acute aortic syndrome, other processes such as intramural hematoma and penetrating atherosclerotic ulcers are being increasingly recognized[2]. The incidence is approximately 3−5/100 000 in western countries with an observed increase during the past decades[3]. Moreover, men suffer more frequently than women do from this acute condition, showing almost a two times higher incidence[4]. Additionally, AAD seems to present in a particular circadian or seasonal pattern. A timeframe between 6.00 am and 12.00 pm as well as winter months seem to show an increased prevalence[5].

Regarding pathophysiology, an endothelial damage of the aortic wall allows the blood to flow through the different aortic wall and to form a false lumen that could apply pressure to the true lumen of the aorta[5]. This leads to a dissection of the aortic wall that could expand either proximally or distally. Acute type B aortic dissection (ATBAD) (identified within 2 weeks of symptom onset), as described using the Stanford classification, accounts for 25%−40% of all aortic dissections[6]. Stanford type B or DeBakey III aortic dissection originates in the descending thoracic aorta without retrograde extension into the ascending aorta, and involves the aorta distal to the left subclavian artery[7]. Acute type B dissections may be classified as uncomplicated or complicated. Approximately 25% of patients presenting with ATBAD are complicated at admission by malperfusion syndrome or hemodynamic instability, resulting in a high risk of early death when untreated. Complicated type B aortic dissection refers to malperfusion syndrome involving visceral, renal, or extremity ischemia, rupture or impending rupture, uncontrolled hypertension, persistent abdominal or chest pain, or findings of rapid expansion on computed tomography (CT) imaging[7].

The selection of optimal therapeutic strategy for ATBAD, in contrast to type A dissection, seems to still be under debate. Therefore, this review aims to collect and present current literature data on proper management of ATBAD as well as to make useful conclusions for all physicians.

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2. Risk factors and clinical presentation

Many risk factors have been identified to be associated with the development of acute aortic syndromes and AAD (Table 1). Almost 75% of these patients suffer from arterial hypertension[8]. Male sex, age and smoking have also been identified as major risk factors[9,10]. Although trauma and endovascular interventions are a leading cause for aortic dissection in patients of all ages[11], hereditary syndromes remain the commonest predisposing factor in children and younger individuals[12]. Marfan syndrome among other connective tissue disorders (e.g. Ehlers–Danlos) is an important risk factor for aortic dissection, especially in young patients and adolescents, and thresholds for prophylactic aortic replacement are typically lower for this specific patient group[13]. However, type B dissection is less frequent than type A in this particular group of patients[13]. Additionally, cocaine use is also implicated in 1.8% of patients with AAD, as underlined by Dean *et al*[14].

Table 1
Major risk factors for acute aortic syndromes and aortic dissection.

Hypertension, particularly uncontrolled	Inflammatory diseases
Genetic or hereditary conditions	Giant cell arteritis
Marfan syndrome	Takayasu arteritis
Bicuspid aortic valve	Behcet arteritis
Ehlers–Danlos syndrome	Other factors
Turner syndrome	Cocaine use
Loeys–Dietz syndrome	Pregnancy
Congenital aortic stenosis	Weight lifting or Valsalva maneuver
Fallot's tetralogy	Infections affecting the aortic wall (*e.g.* syphilis)
Familial thoracic aorta diseases	
Atherosclerosis	
Trauma or injury	
Catheterization	
Cardiovascular surgery	
Injury/trauma	
Coarctation of the aorta	

Recently, the International Registry of Acute Aortic Dissection (IRAAD) declared that about one fifth of these patients do not present with an aortic dilatation[15]. The risk of ATBAD was thought to increase with descending thoracic aortic diameter. However, the majority of patients with ATBAD present with a descending aortic diameter less than 5.5 cm before dissection. Therefore, aortic diameter measurements do not seem to be a useful parameter to prevent aortic dissection, and other methods are needed to identify patients at risk for ATBAD[16]. Furthermore, inflammatory or infectious diseases could also predispose to type B aortic dissection[17,18]. Finally, aortic atherosclerosis seems to play an important role in aortic dissection development, and is more associated with distal than with proximal aortic dissection, according to recent data[19].

Regarding clinical presentation, pain is the most commonly reported presenting symptom of AAD regardless of patient age, sex, or other associated clinical complaint[20]. It is usually described as tearing, stabbing, or sharp in character. Almost 17% of individuals will feel the pain migrate as the dissection extends down the aorta, while the location of pain is associated with the location of the dissection[21]. The combination of two or more high–risk features (Table 2) is strongly suggestive of AAD[20]. Up to 20% of these patients will suffer from syncope. Although acute aortic valve insufficiency and myocardial/cerebral ischemia are observed mainly in type A dissection, type B dissection could more often lead to paraplegia, acute renal insufficiency or even limb ischemia. In AADs, compromise of one or both renal arteries occurs in 5%–8% of cases, while mesenteric ischemia (ischemia of the large intestines) occurs 3%–5% of the time[21]. Approximately 30% of patients who present with ATBAD have a complicated dissection, making immediate treatment imperative to save the life or the limb of the patient[22]. Independent predictors of death in type B dissection have been jointly termed the 'deadly triad': hypotension/shock, absence of chest/back pain on presentation, and branch vessel involvement[23].

Table 2
High–risk features for aortic dissection.

Classification	Features
High risk conditions	Marfan Syndrome
	Family History Aortic Disease
	Known Aortic Valve Disease
	Recent Aortic Manipulation
	Known Thoracic Aortic Aneurysm
High risk pain features	Chest, back or abdominal pain described as any of the following:
	Abrupt onset
	Severe intensity
	Ripping or tearing
High risk exam features	Evidence of perfusion deficit
	– Pulse deficit
	– Systolic blood pressure differential
	– Focal Neurologic Deficit (combined with pain)
	Murmur or Aortic Insufficiency (new and with pain)
	Hypotension or Shock state

3. Diagnostic tools

Although imaging studies are sensitive and specific in general, timely diagnosis can be delayed because of variability in presenting symptoms and the relatively low frequency with which acute aortic syndromes are seen in the emergency setting. Therefore, utilization of proper diagnostic tools is imperative to set final diagnosis, although the indication for use of most imaging studies is of evidence level C.

3.1. Plain chest X–ray

Findings on plain chest radiographs of patients with aortic dissection are variable and often overlap those of patients

without dissection. In an older study by Jagannath *et al.*, a widened aortic knob, widened descending aorta, and widened mediastinum showed the greatest inter–observer agreement (*P*<0.001) although the overall inter–observer agreement was poor[24]. Although their utilization in the emergency setting is useful, this finding dictates that further definitive investigation should be undertaken. In another retrospective study by Lai *et al.*, postero–anterior (PA) chest radiography showed a higher diagnostic accuracy compared to antero–posterior (AP) imaging[25]. However, according to the authors, a lower threshold for proceeding to a computed tomography (CT) evaluation is recommended in the elderly and patients with widened mediastinum in the AP X–ray. Additionally, data indicate a limitation of plain chest radiography in discriminating between AAD and other acute coronary syndromes[26].

3.2. D–dimers

D–dimers are cleavage products of fibrin that occur during plasmin–mediated fibrinolysis of blood clots[27]. In the emergency setting, their measurement in serum represents a valuable and cost–effective tool in the differential diagnosis of acute chest pain including the main life–threatening entities: acute coronary syndrome, pulmonary embolism, and acute aortic syndrome[27]. However, because of limitations in specificity, d–dimer testing is only one component in the diagnosis of acute chest pain. It has been shown that a positive d–dimer test has a sensitivity of about 97%, a specificity of 56%, a positive predictive value of about 60%, and a negative predictive value of up to 96%[28]. Furthermore, d–dimer levels seem to correlate with the anatomic extension of the dissection as well[29]. In the recent meta–analysis of Shimony *et al.*[30], the authors conclude that plasma levels <500 ng/mL is a useful screening tool to identify patients who do not have AAD. Therefore, serum d–dimers could be used to identify subjects who are unlikely to benefit from further aortic imaging.

3.3. Ultrasound assessment

The combination of different ultrasound techniques such as transthoracic, suprasternal, subcostal, and transesophageal ultrasonography has a high sensitivity and specificity in the diagnosis of aortic dissection[31]. Main goals of this examination are a) to confirm the diagnosis by visualizing the intimal membrane, b) to differentiate the true or false lumen, c) to detect the intimal tear, d) to determine the extent of the dissection and classify it, and e) to detect wall motion abnormalities or side branch involvement[31]. There are studies showing that the transesophageal approach can identify specific important elements of the dissection (such as false lumen thrombosis or visualization of flap in the aorta) more accurately than the transthoracic approach[32]. Recently, there have been studies highlighting the potential value of intravascular ulrtasonography (IVUS) in the diagnosis of AAD as well as aortic intramural hematoma[33].

3.4. Computed tomography/angiography (CT/CTA)

The widespread use of 3–dimensional imaging such as CT has increased dramatically in the last decades, and the incidence of diagnosed aortic dissection cases has increased as well. Current methods to risk stratify patients with type B aortic dissection, rely upon static imaging, usually CT angiography[34]. In a study by Sommer *et al.*, spiral CT demonstrated a specificity of 100% compared to multiplanar transesophageal ultrasound (94%)[35]. Concerning time–resolved CT–angiography, data indicate that it is feasible at a reasonable effective radiation dose and adds significant diagnostic information with therapeutic consequences in patients with aortic dissection[36].

3.5. Magnetic resonance imaging (MRI)

Although MRI shows a high sensitivity and specificity for the diagnosis of AAD, this technique is not usually available in all institutions, and the examination can be very difficult in unstable patients[35]. MRI study allows multiplanar study of the lesions without contrast medium, and best visualization of sub–endothelial bleeding in dissections without intimal lesion[37]. Therefore, Liu *et al.* support that 3D contrast–enhanced MR angiography with post–processing is a fast, accurate, and noninvasive technique that may prove to be the optimal imaging modality in medically stable patients with aortic dissection[38].

3.6. 18F–flurodeoxyglucose positron emission tomography (18F–FDG PETCT)

This is a novel imaging technique, with preliminary data showing that it can visualize atherosclerotic plaques and that it has prognostic value concerning risk for rupture and progression of dissection[39,40]. Additionally, Reeps *et al.* have found that this imaging tool could probably differentiate acute from chronic aortic dissection in unclear cases[41]. However, more studies are needed to clarify its role in clinical scenario.

4. Treatment strategies

4.1. Conservative therapy

Patients suffering from acute distal aortic dissection are at significantly lower risk of early death from complications of the dissection than are those with proximal dissection[42]. Therefore, aggressive medical treatment has been recommended since almost 50 years and has been advocated by many authors[43]. The main goal is the reduction of systolic blood pressure (goal levels: 100–120 mmHg) and

diminution of the rate of left ventricular ejection (dP/dt). Primary concern is also the relief from pain using mostly morphine regimens[43]. The combination of beta–blockers along with another antihypertensive agent is usually recommended. However, it is suggested that nitrates should be given after beta–blockage, in order to avoid immediate vasodilatation, secondary catecholamine release, increase of left ventricular contractility and subsequent extension of dissection. This intensive medical therapy seems to reduce mortality in type B aortic dissection[44]. Promising results have been reported with angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARB) as well[45], although some authors have not found a significant prognostic value[42].

Heart rate reduction has been thought to be an important aspect of medical treatment as well. Kodama *et al.* conclude that tight heart rate reduction improved the outcome in patients with AAD who were conservatively managed[42]. A 'complication–specific' approach has been suggested, including medical management with 'anti–impulse therapy' for uncomplicated acute descending dissections[46]. Following initial stabilization with intravenous antihypertensives, most patients will require long–term antihypertensive treatment. As recommended by the latest guidelines, acute thoracic aortic dissection involving the descending aorta should be managed medically unless life–threatening complications develop (such as malperfusion syndrome, progression of dissection, enlarging aneurysm, inability to control blood pressure or symptoms; Level of Evidence: B)[20].

However, uncertainty still remains regarding the optimal management strategy for uncomplicated acute type B dissection. Best medical treatment is associated with a considerable risk for disease progression towards complicated dissection or aneurysm degeneration of the affected segment with an estimated incidence of almost 40%[47]. Furthermore, long–term outcomes after primary conservative treatment have been associated with a very high complication rate[48].

4.2. Open repair

Despite aggressive antihypertensive treatment, hospital mortality after primary conservative treatment is still high and a substantial proportion of patients require surgery during initial hospitalization[49]. Main goal of surgical management in ATBAD is the prevention of aortic rupture or the treatment of serious complications (Table 3). Carrel *et al.* underline that early surgery will be needed in the following cases, even for uncomplicated type B dissection: i) younger patients with 5 cm diameter of the aorta at initial evaluation, ii) those with Marfan syndrome, iii) patients with limited false aneurysm or retrograde dissection into the aortic arch, and iv) those with poor medical compliance or uncontrollable proximal hypertension[49].

Table 3
Major complications of acute aortic dissection.

Position	Complications
Cardiovascular	Aortic insufficiency
	Syncope
	Pericardial tamponade
	Myocardial ischemia/infarction
	Congestive heart failure
Neurologic	Ischemic stroke/transient ischemic attack
	Peripheral neuropathy
	Paraplegia/paraparesis
	Spinal ischemia
Pulmonary	Pleural effusion
	Aortopulmonary fistula with bleeding
Gastrointestinal	Mesenteric ischemia/infarction
	Aortoenteric fistula with bleeding
Renal	Renal failure
	Renal ischemia/infarction
Extremities	Limb ischemia

Perioperative mortality for patients treated for aortic dissection ranges from 5% to 10% and could reach 70% in complicated cases[50]. Independent prognostic factors for perioperative mortality in open repair include the presence of cardiac tamponade, the location of the intima tear, the duration of surgery, the presence of renal/visceral ischemia, renal dysfunction and the presence of pulmonary disease[51]. Although data reveal a superiority of endovascular techniques regarding early and midterm mortality, Moulakakis *et al.* conclude in their recent meta–analysis that open repair still has a significant role as endovascular repair is not applicable in all patients and there are still concerns regarding the durability of this technique[48]. However, the absence of randomized trials comparing endovascular with open repair treatments in complicated type B acute dissection remains a limitation.

Two recent meta–analyses seem to lead to controversial results. Luebke *et al.* suggest that the use of endovascular treatment in complicated type B aortic dissection leads to favourable early outcomes with lower neurologic and vascular complications, although there were no sufficient data for long–term outcomes[52]. However, Zhang *et al.* conclude that endovascular treatment reduces short–term mortality, although it does not improve postoperative complications or long–term mortality significantly[53].

4.3. Endovascular repair

Endovascular repair may be of particular value in patients with significant co–morbid conditions (older age, substantial cardiac, pulmonary or renal dysfunction) who would be considered poor or non–candidates for open surgery[20]. Data indicate that patients who are not considered candidates for open surgery but who have undergone endovascular grafting have substantially poorer long–term outcomes than patients who are reasonable candidates for open operation and are treated with endografts[20]. Endovascular treatment of aortic dissection includes three major therapeutic approaches:

1) placement of an aortic endograft, 2) fenestration and 3) stenting of aortic branches[54]. The main goal of endovascular management is to seal the tear of the intima and to cause the thrombosis of the false lumen. This will have a positive effect on aorta remodeling during the dissection and will decrease the risk of rupture[54].

Regarding the comparison between the two interventional methods, there are no firm data conclusively demonstrating that the prevalence of spinal cord ischemic injury (lower extremity paralysis or paresis) is less for endovascular approaches than for open surgical repair. Similarly, there are no firm data indicating that overall medical care costs are lower with endovascular repair[20,55]. Furthermore, some patients are not suitable candidates for endovascular grafting procedures. Absence of suitable "landing zones" above and below the aneurysm (usually 2 to 3 cm of normal diameter aorta without circumferential thrombus) as well as landing zone width exceeding the recommended width for the largest available endovascular grafts (generally 10% to 15% larger than the width of the aorta) are also contraindications[55]. Finally, lack of vascular access sites as well as severe atherosclerosis and intraluminal thrombus of the aorta may increase the risk of peripheral embolism during manipulation of guidewires and catheters[56].

Regarding prognosis, Desai *et al.* conclude in their study that delayed intervention appears to lead to lower complication risk after thoracic endovascular aneurysm repair (TEVAR) in patients who are stable enough to wait[57]. Additionally, Wilkinson *et al.* found that early aortic repair for complicated type B dissection leads to high mortality and re–intervention rates, with results of TEVAR being similar with that of open repair[58]. However, Tang *et al.* showed that emergency endovascular repair of complicated acute dissection within 24 h is associated with good results and decreases mortality[59].

The recent report of IRAAD underlines that TEVAR is associated with lower 5–year mortality than medical therapy for ATBAD, although more randomized trials with long–term follow–up are needed[60]. However, Hanna *et al.* found recently that TEVAR is associated with excellent short–term outcomes after acute dissection, with durable and sustained results over long–term follow–up[61]. Studies so far suggest that endovascular techniques may shift the risk of patients with acute complicated type B dissection from high to lower mortality, comparable to that seen in uncomplicated distal dissection[62]. The IRAAD database suggests a better outcome in patients treated with stent graft for acute dissection compared to open surgical repair, lowering short–term mortality to the level of medically managed uncomplicated type B dissection[62].

As aforementioned, there still remains a debate regarding the indicated strategy for uncomplicated type B aortic dissection. Recently, one year results of the ADSORB trial were published, concerning whether endovascular repair could be applied in uncomplicated ATBAD[63]. Conservative treatment of such cases is followed by a high 30–day mortality and intervention rate within 4 years. Therefore, endovascular treatment plus optimal medical therapy was compared with best medical treatment only in this multicenter randomized trial. Although early death and neurologic complications rates were low for both groups, 1–year aortic remodeling was better in the first group[63].

5. Conclusions

Conservative management with optimal medical therapy remains the first line strategy for patients with uncomplicated acute type B aortic dissection. Open repair is indicated only for complicated cases. Endovascular repair shows promising results in selected patients with increased perioperative risk, when there are no contraindications. There are promising results regarding the utilization of endovascular repair even in uncomplicated cases, although more data on long–term outcomes are needed. The decision on optimal strategy should always be based on the individual characteristics and risk factors of each patient.

Conflict of interest statement

The authors report no conflict of interest.

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