

Acute Dissection of the Descending Aorta: A Case Report and Review of the Literature

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ABSTRACT

Cardiovascular disease is the leading cause of death worldwide. Acute aortic syndromes, which include aortic dissection, intramural hematoma, and penetrating aortic ulcer, represent the most morbid presentations of aortic disease and can be difficult to diagnose. Recent advances in imaging have allowed for more rapid and accurate diagnosis of acute aortic syndromes and the options for management are expanding. This case report

and review presents the case of a 43-year-old man with acute type B aortic dissection who underwent two endovascular procedures for malperfusion syndrome. The review focuses on the presentation, diagnosis, medical management, and procedural options for acute dissection of the descending aorta.

Keywords: Aortic dissection; Endovascular; Hypertension; Type B aortic dissection

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INTRODUCTION

Cardiovascular disease is the leading cause of death worldwide [1]. Acute aortic syndromes, which include aortic dissection, intramural hematoma, and penetrating aortic ulcer, represent the most morbid presentations of aortic disease and can be difficult to diagnose [2, 3]. Approximately 40% of patients with acute aortic dissection die before reaching the hospital [4] with in-hospital early mortality of 1–2% per hour for those who do survive to hospital-level care [5]. Recent advances in imaging have allowed for more rapid and accurate diagnosis of acute aortic syndromes

and the options for management are expanding. This case report and review of the literature will focus on the presentation, diagnosis, medical management, and procedural options for acute dissection of the descending aorta.

The authors conformed with the Helsinki Declaration of 1975, as revised in 2000 concerning Human and Animal Rights. Springer's policy concerning informed consent has been followed. This article does not contain any studies with human or animal subjects performed by any of the authors.

CASE PRESENTATION

A 43-year-old man with a history of cocaine and amphetamine use and no medical contact since childhood presented to an outside hospital with severe, acute-onset, tearing back and flank pain. On initial evaluation his blood pressure was 240/130 mmHg with a heart rate of 100 beats per minute and he was noted to have an altered mental status. Computerized tomography (CT) angiography at the outside hospital demonstrated a descending aortic dissection. He received an intravenous labetalol bolus followed by continuous infusions of esmolol and nitroprusside. He was transferred to our institution for further management.

On arrival, the heart rate was 91 beats per minute and noninvasively measured blood pressure was 139/66 mmHg in both arms. The radial, dorsalis pedis, and posterior tibialis pulses were full and equal bilaterally. Laboratory evaluation was notable for potassium of 5.9 mmol/L, creatinine of 1.57 mg/dL, lactic acid of 2.9 mg/dL, white blood cell count of $20.17 \times 10^3/\mu\text{L}$, and a mild troponin-T elevation at 0.06 ng/mL (Table 1). The electrocardiogram showed sinus rhythm at a rate of 63 beats per

Table 1 Initial laboratory results

Test	Result	Reference range
Sodium (mmol/L)	136	135–145
Potassium (mmol/L)	5.9	3.5–5.0
Chloride (mmol/L)	99	98–108
Bicarbonate (mmol/L)	24	23–32
Blood urea nitrogen (mg/dL)	30	9–25
Creatinine (mg/dL)	1.57 ^a	0.7–1.3
Glucose (mg/dL)	207	54–118
Alanine aminotransferase (U/L)	80	10–50
Aspartate aminotransferase (U/L)	67	10–50
Alkaline phosphatase (U/L)	69	35–130
Total bilirubin (mg/dL)	0.5	0–1
Creatine kinase (units/L)	149	39–308
Creatine kinase-myocardial band (ng/mL)	6.7	0–6.6
Troponin-T (ng/mL)	0.06	0.00
Urine toxicology screen	POSITIVE: Amphetamines, tetrahydrocannabinol, opiates NEGATIVE: Benzodiazepines, barbiturates, cocaine, methadone, oxycodone	
White blood cells ($\times 1,000/\text{mL}$)	20.17	4–10
White blood cell differential (%)	84.2	Polymorphonucleocytes
	5.2	Lymphocytes
	10.4	Monocytes
	0.1	Eosinophils
	0.1	Basophils
Hematocrit (%)	46.1	40–54

Table 1 continued

Test	Result	Reference range
Platelet count ($\times 1,000/\text{mL}$)	290	150–450
International normalized ratio	1.0	
Partial thromboplastin time (s)	28.1	23.8–36.6
Urinalysis	Color: clear yellow Specific gravity: 1.059 pH: 6.0 1+ protein	
Urine sediment (number per high power field)	4 red blood cells 4 white blood cells 0 crystals 0 casts No bacteria	

^a No baseline creatinine was available

minute with left axis deviation as well as an R wave in aVL measuring 14 mm, consistent with left ventricular hypertrophy. Urine toxicology screen was positive for amphetamines, tetrahydrocannabinol, and opiates (the latter had been administered prior to transfer).

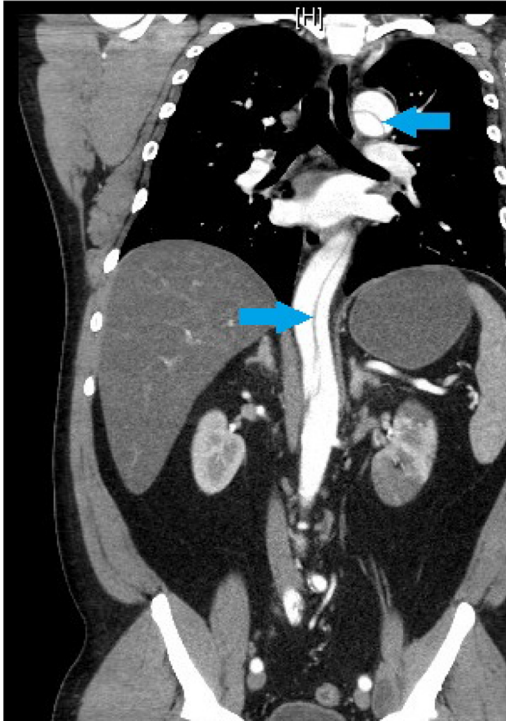
Review of the CT obtained at the hospital of first presentation revealed a descending aortic dissection that began distal to the origin of the left subclavian artery and extended to both iliac arteries (Fig. 1). The left renal artery received blood supply from the false lumen, which compressed the true lumen at the site of the right renal artery takeoff. There was lack of contrast enhancement of the left kidney consistent with malperfusion. Enhancement of the right kidney was normal.

Following multidisciplinary evaluation, consensus was reached that the time for surgical intervention to preserve the left kidney had passed. It was noted that the right

kidney appeared to be perfused and the left kidney had a warm ischemic time of approximately 12 h. Given the duration of warm ischemic time and lack of evidence of other limb or visceral malperfusion, surgical intervention was deemed unlikely to be beneficial and intensive medical management was recommended. Medical therapy was selected with careful monitoring of renal function and visceral and extremity perfusion. The infusions of esmolol and nitroprusside were uptitrated and the patient was given hydromorphone and fentanyl for pain. He was intubated for airway protection in the setting of altered mental status and was monitored in the cardiac intensive care unit with goal heart rate <60 beats per minute and goal systolic blood pressure 100–120 mmHg.

Despite control of heart rate and blood pressure, the urine output declined to <20 cc/h and creatinine rose to 2.9 mg/dL within the first 24 h. It was felt that expansion of the false lumen with compression of the true lumen was leading to malperfusion of the right kidney. In addition, he was noted to have diminished pulses in the right foot. After multidisciplinary consultation, the decision was made to proceed with aortic fenestration with the goal of decompressing the false lumen to restore flow to the right kidney (Fig. 2, Movie 1). The right renal artery pressure was lower than the proximal aorta pressure with a peak gradient of 50 mmHg and mean gradient of 22 mmHg. Intravascular ultrasound (IVUS) of the aortic true lumen showed it to be severely compressed in the suprarenal segment with a dynamic component. Fenestration was performed using a Pioneer[®] Plus catheter (Medtronic CardioVascular, Santa Rosa, CA) and dilations across the dissection flap were performed with 10 and 14 mm balloons. Intravascular ultrasound showed an expanded true lumen

Panel A



Panel B

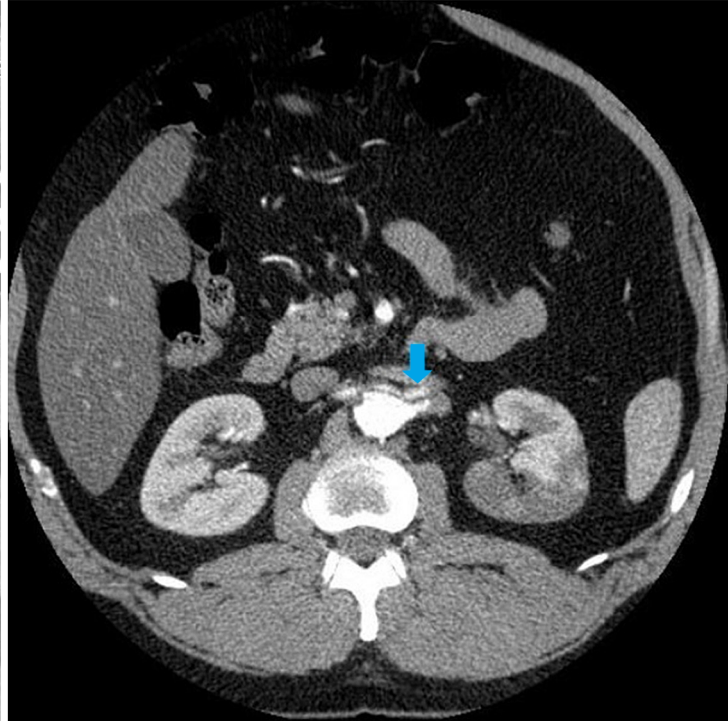


Fig. 1 CT angiogram. There is a type B aortic dissection (*Panel A, blue arrows*) extending from just below the left subclavian artery to the proximal iliac arteries. The left main renal artery arises from the false lumen. A supernumerary left

renal artery arises from the true lumen of the aorta and supplies a segment of that kidney (*Panel B, blue arrow*). The right renal artery is supplied by the true lumen, which is compressed by the false lumen at the site of the right renal artery takeoff

and a patent right renal artery. The urine output increased to 60 cc/h over the next 4 hours.

Despite the initial improvement in renal function, the urine output again declined to <30 cc/h with progression to anuria over the subsequent 8 h. The patient was taken back to the catheterization laboratory 12 h after the first procedure. IVUS of the aorta showed that in the thoracic aorta the true lumen was widely patent but in the sub-diaphragmatic segment at the level of the visceral vessels the true lumen became quite small before enlarging again in the infrarenal aorta. An endovascular endarterectomy from T11 to the aortic bifurcation was undertaken (*Fig. 3*) with restoration of flow to the right renal artery. Urine output post-procedure was 400 cc/h for the next hour and then returned to baseline.

The remainder of the hospital course was complicated by ventilator-associated pneumonia, right internal jugular vein thrombosis, and new-onset left arm weakness with magnetic resonance imaging of the brain demonstrating likely embolic infarcts in the right middle cerebral artery territory. The patient was successfully extubated and transitioned to oral medications to control the blood pressure and heart rate. No embolic source was found and the left arm weakness had markedly improved by discharge on hospital day 20. The creatinine, which had peaked at 3.36 mg/dL on hospital day three, had declined to 1.3 mg/dL by discharge. At follow-up, the patient was compliant with a stable regimen of metoprolol, hydrochlorothiazide, lisinopril, and amlodipine to maintain a normal blood pressure and had not used cocaine or amphetamines since discharge.

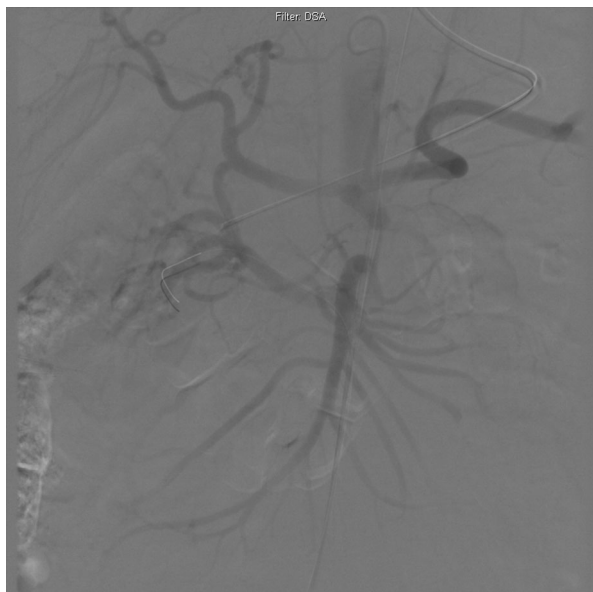


Fig. 2 Balloon fenestration of the descending aortic flap. Vascular access was obtained via the right common femoral artery. The right renal artery was first engaged and the pressure was 50 mmHg lower than in the proximal aorta, with a mean pressure gradient of 22 mmHg. The vessel itself was patent on selective right renal artery injection. Intravascular ultrasound (IVUS) of the aortic true lumen showed it to be severely compressed in the suprarenal segment with a dynamic component. Using a 6 F Pioneer[®] Plus catheter (Medtronic CardioVascular, Santa Rosa, CA) a puncture was made from the true lumen to the false lumen during visualization with IVUS. A coronary wire was advanced into the false lumen all the way to the origin of the left subclavian artery where it stopped. Fenestration with 10 and 14 mm balloons followed (*Movie 1*). Repeat IVUS of the true lumen showed it to be markedly expanded and a large gap in the flap just below the origin of the renal artery was visualized. IVUS of the right renal artery showed it to be patent. Repeat measurement of pressure in the renal artery showed that the mean gradient had decreased to 16 mmHg. Normal flow was seen in the celiac and mesenteric arteries after fenestration (*Panel A*)

The creatinine stabilized between 1.3 and 1.5 mg/dL. Contrast enhanced CT scan performed three and a half months after discharge (Fig. 4) demonstrated a stable dissection beginning immediately distal to the left subclavian artery and extending to the distal thoracic aorta without involvement of the great vessels. The upper pole of the left kidney was infarcted and atrophied. The

proximal abdominal aortic dimensions remained stable at 3.3×3.3 cm.

PATHOPHYSIOLOGY AND CLASSIFICATION

Aortic dissection is a disease of the media layer of the vessel. Bleeding into and along the media causes a separation of the layers of the aorta [4]. An intimal tear is typically present with a dissection flap separating the true lumen of the aorta from the newly created false lumen [6]; however, intramural hemorrhage may be present without intimal tear [4]. Spread of the dissection may proceed in an antegrade or retrograde direction, or both, and has the potential to involve branch vessels as well as cardiac structures.

Degeneration of the aortic media has long been considered the central underlying process in aortic dissection, intramural hematoma, and aneurysm formation [3, 7, 8]. Numerous factors lead to medial degeneration, including atherosclerosis, hypertension, tobacco use, genetic syndromes, and inflammatory aortopathies, with the end result of increased wall stress as indicated by the law of La Place. This relationship, written as $\sigma = P \times r/h$, where σ is wall stress, P is luminal pressure, r is vessel radius, and h is wall thickness [9], demonstrates how hypertension, aortic dilatation, and decreased wall thickness all contribute to increased levels of stress. The law of La Place additionally shows the particularly detrimental effect of atherosclerosis, which causes medial necrosis and thinning by limiting the supply of oxygen and nutrients and leads to increased radius through aneurysmal dilatation. When hypertension and atherosclerosis coexist, as they frequently do, all three variables contributing to wall stress have been detrimentally modified.



Aortic dissection is typically classified according to location. De Bakey's original system [10] based on the site of origin and extent of dissection has largely been supplanted by the Stanford system [11], which classifies according to involvement of the ascending aorta. Stanford type A includes all dissections involving the ascending aorta, regardless of the site of origin. Stanford type B dissections are confined to the descending aorta. A spectrum of aortic abnormality is now recognized including intramural hematoma and penetrating aortic ulcer in addition to classic aortic dissection; the Svensson system assigns acute aortic syndromes to one of five classes (Table 2) [12, 13]. The patient presented here had a Stanford type B, Svensson Class 1 aortic dissection.

EPIDEMIOLOGY AND RISK FACTORS

The incidence of aortic dissection in developed countries is approximately three events per

◀**Fig. 3** Endarterectomy and stenting of the descending aortic flap. Vascular access was obtained via the right common femoral artery. Wires were placed in both the true aortic lumen and the false lumen under intravascular ultrasound (IVUS) guidance via the external iliac artery. IVUS of the aorta showed that in the thoracic aorta the true lumen was widely patent and large in caliber. In the sub-diaphragmatic segment at the level of the visceral vessels the true lumen became quite small before enlarging again in the infrarenal aorta (*Panel A*). An endovascular endarterectomy was performed from T11 to the aortic bifurcation. A 6 F Pioneer® Plus IVUS reentry catheter (Medtronic CardioVascular, Santa Rosa, CA) was used to create a path from the true lumen to the false lumen at T11 (*Movie 1*). This wire spanning both lumens was snared in the false lumen using a catheter advanced from the right common femoral artery. Both ends of a 0.025" Terumo® guide wire (Terumo Europe N.V., Leuven, Belgium) were externalized and pulled together to the iliac bifurcation to perform the endarterectomy (*Movie 2*). IVUS of the aorta showed that the infra-diaphragmatic aorta now comprised a single lumen. The right renal artery and mesenteric vessels filled briskly but the aorta was occluded at the just below the inferior mesenteric artery as a result of functional endarterectomy of the infra-diaphragmatic aorta. Two 8 × 57 mm VisiPro™ balloon-expandable peripheral stents (Covidien, Dublin, Ireland) were deployed in a kissing fashion, extending from the infrarenal aorta into the right and left common iliac arteries. They were postdilated with 10 and 12 mm balloons. Further overlapping stenting was required to maintain adequate distal flow. Femoral and pedal pulses were bounding post-procedure

100,000 person-years [14]. Aortic dissection occurs with a bimodal age distribution. Among older patients, the most prevalent risk factors are hypertension, atherosclerosis, male gender, aortic aneurysm, smoking, and prior cardiovascular operations [15, 16] (Table 3). The greatest risk factor for aneurysm is atherosclerosis [17]. Among younger patients, genetic causes such as Marfan's syndrome, Ehlers–Danlos syndrome (type IV), Loeys–Dietz syndrome, Turner syndrome, aortic coarctation, bicuspid aortic valve, and non-syndromic familial dissection are the predominant risk factors [3, 18]. A full 50% of patients aged less than 40 years with aortic dissection have

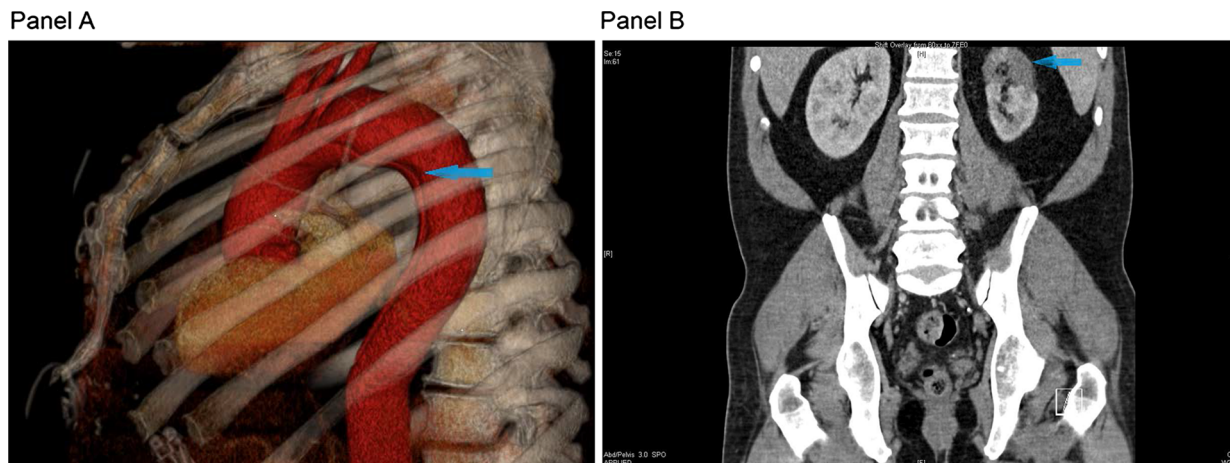


Fig. 4 Follow-up CT scan. CT scan obtained three and a half months after discharge demonstrated a Stanford type B aortic dissection with the intimal flap originating just distal to the origin of the left subclavian artery and extending to the descending portion of the thoracic aorta (*Panel A*, 3D reconstruction, *blue arrow*). There was no involvement of

the great vessels. A marked perfusion defect in the upper pole of the left kidney with associated interval atrophy of the left upper pole was seen (*Panel B*, *blue arrow*). Unchanged aneurysmal dilatation of the proximal abdominal aorta, measuring 3.3×3.3 cm, was present

Table 2 Svensson system for classification of aortic dissection

Class 1: Classic dissection with intimal flap separating true and false lumens

Class 2: Intramural hematoma

Class 3: Limited intimal tear with eccentric bulge

Class 4: Penetrating atherosclerotic ulcer

Class 5: Iatrogenic dissection

Classification of aortic dissection as outlined by Svensson et al. [12]

Marfan's syndrome [19]. Inflammatory aortopathies such as Takayasu arteritis and giant cell arteritis additionally predispose to aortic syndromes. Dilatation of the ascending aorta and aortic arch as well as aortic tortuosity are newly described anatomic predictors of type B dissection among patients with hypertension [20].

The International Registry of Acute Aortic Dissection (IRAD) was formed in 1996 to track the presentation, diagnosis, management, and

outcomes of patients with acute aortic dissection at 25 referral centers in 12 countries [21]. Among this cohort, 65% of patients were males with a mean age of 63 years. Nearly three quarters carried a diagnosis of hypertension, 31% had known atherosclerosis, and 18% had undergone prior cardiac surgery. Four percent had Marfan's syndrome [21]. Thirty-eight percent of dissections were Stanford type B. Overall mortality was approximately 23% in the IRAD registry, but was 40% among patients who had used cocaine prior to dissection [21]. Among patients with type B dissection, overall mortality was 12%, with 20% mortality among those with complications and 6% mortality in uncomplicated cases [22]. Mortality does not appear to be significantly different between acute type B dissection and type B intramural hematoma [23].

Based on these data, our patient was typical in his gender and predisposing hypertension, but was at increased risk for mortality given his renal injury and stimulant use. His relative

Table 3 Risk factors for aortic dissection

Conditions associated with increased aortic wall stress

Hypertension, particularly if poorly controlled
 Pheochromocytoma
 Cocaine or other stimulant use
 Weight lifting or other Valsalva maneuver
 Trauma
 Deceleration or torsional injury
 Coarctation of the aorta

Conditions associated with aortic media abnormalities

Genetic

Marfan's syndrome
 Ehlers–Danlos syndrome, vascular form
 Bicuspid aortic valve (including prior aortic valve replacement)
 Turner syndrome
 Loeys-Dietz syndrome
 Familial thoracic aortic aneurysm and dissection syndrome

Inflammatory vasculitides

Takayasu arteritis
 Giant cell arteritis
 Behçet arteritis

Other

Pregnancy
 Polycystic kidney disease
 Chronic corticosteroid or immunosuppressant administration
 Infections involving the aortic wall

Risk factors for aortic dissection as described by Hiratzka et al. [4]

youth raises the possibility of an underlying vasculopathy, but he had clear predisposing risk factors for dissection and no other evidence to suggest a syndromic process.

PRESENTATION

The spectrum of presentation of acute aortic dissection is protean, requiring vigilance on the part of the provider to arrive at the diagnosis promptly. Given the frequently non-specific presentation and higher pre-test probability of more common processes, acute aortic dissection is not diagnosed on initial evaluation in 38% of patients [24]. Common symptoms and signs include abrupt onset chest and/or back pain (86%) with radiation to the shoulders or neck that is typically described as tearing/sharp (64%), hypertension (69%), pulse deficit (20–30%), and syncope (13%) [16, 21, 24]. Less common presentations include heart failure symptoms, abdominal pain, spinal cord involvement with neurological deficits, Horner syndrome, and vocal cord paralysis [9]. Painless presentation is also observed in approximately 4–5% of patients and is associated with worse prognosis, perhaps related to delay in diagnosis [21].

The patient discussed here presented with acute, tearing back and flank pain as well as hypertension, allowing prompt consideration of the correct diagnosis.

DIAGNOSIS

The principle approach to the diagnosis of acute aortic syndromes is radiographic, though biochemical assays are an area of active research. The electrocardiogram (ECG) can be helpful in detecting rare complications such as coronary involvement; however, it is frequently abnormal and is not specific for acute aortic syndromes [24, 25].

The goals of aortic imaging are fourfold, namely to (1) establish the diagnosis, (2) localize the intimal tear, (3) determine the

extent of dissection, and (4) assess for indicators of pending emergency [9]. The modalities available for definitive imaging include computerized tomography angiography (CTA), magnetic resonance imaging (MRI), transesophageal echocardiography (TEE), and, less commonly, aortography. Among the IRAD cohort, CTA was performed in 93% of cases, TEE in 59%, MRI in 31%, and aortography in 24% [16]. Often times more than one study is necessary to confirm and fully characterize the diagnosis; thus the mean number of imaging studies other than plain film performed per patient is 1.8 [24].

Transthoracic echocardiography (TTE) provides a rapid tool for assessing cardiac complications of type A dissection such as wall motion abnormalities indicative of coronary artery involvement, pericardial tamponade, and aortic insufficiency [26]. TTE provides minimal to no imaging of the distal ascending, transverse, and descending aorta and is typically used in conjunction with TEE or another imaging modality. TEE can be rapidly obtained in many circumstances and has the advantages of excellent visualization of the distal ascending, transverse, and descending aorta, ability to differentiate between true and false lumens, and identification of intramural hemorrhage and penetrating atherosclerotic ulcer. TEE is limited by its invasive nature requiring sedation and a contraindication in significant esophageal disease, such as varices [27]. When combined with TTE, TEE achieves a sensitivity of 99% and specificity of 89% [28] for aortic dissection.

CT imaging with intravenous contrast can be rapidly performed in most Emergency Departments and has a sensitivity of at least 95% and specificity between 85% and 100% [9, 29, 30]. The disadvantages of CT are the contrast load in patients who already have or

may develop acute kidney injury as a result of the underlying process and limited ability to identify the site of intimal tear [9, 31].

MRI has perhaps the highest sensitivity and specificity, approaching 100% for each [27, 29, 32]. MRI has the additional advantages of not requiring intravenous contrast injection for vessel visualization and not exposing the patient to ionizing radiation [27]. Its use is constrained by the factors that limit MRI use in other settings, which are lack of availability, length of time for image acquisition in potentially unstable patients, patient discomfort/anxiety, and contraindication in patients with metallic implants (e.g., most pacemakers and implantable cardioverter-defibrillators).

Given that TEE, CT, and MRI all have favorable test characteristics, the choice of imaging modality largely depends on local availability and time. CT is nearly universally available in the developed world, is non-invasive, and is rapid, and, therefore, is the preferred modality in many institutions [16]. However, some institutions practice routine TEE in patients with proximal dissections. The American Heart Association (AHA) recommends TEE as the first imaging study in unstable patients to prevent patient transport out of the acute care setting [4].

Biochemical markers of aortic dissection remain largely investigational. Candidate markers include lactate dehydrogenase, d-dimer, white blood cell count, c-reactive protein, fibrinogen, and circulating smooth muscle myosin heavy chain. Currently, there is no clinically available biochemical assay specific for aortic dissection [4].

The patient described here had CT angiography performed on presentation to the referring hospital, which accurately identified the type B aortic dissection and branch vessel

involvement. Transesophageal echocardiography would have been a reasonable initial study as well given the need for close hemodynamic management and the elevated creatinine.

MANAGEMENT

The initial goals in management are to (1) decrease the shear stress on the aortic wall by limiting the force of left ventricular contraction; (2) minimize total wall stress by decreasing blood pressure to the extent allowed; and (3) maintain adequate end organ perfusion. Beta-blockers such as metoprolol, esmolol, propranolol, and labetalol are administered as first-line agents with goal heart rate <60 beats per minute and goal systolic blood pressure between 100 and 120 mmHg (Table 4) [4, 9, 26]. In patients who are intolerant of beta-adrenergic blockade, non-dihydropyridine calcium channel blockers may be an effective alternative [4, 9]. Vasodilators such as sodium nitroprusside, and nitroglycerin, or dihydropyridine calcium channel blockers are employed if beta blockade fails to adequately control blood pressure, but should not be used prior to heart rate control or as single agents given their propensity to increase the left ventricular contractile force and cause reflex tachycardia [4, 26]. Pain control is an important aspect of heart rate and blood pressure management and is typically achieved with morphine sulfate or another narcotic [4, 9]. There are no randomized trials comparing medical management strategies for acute type B dissection [4].

Uncomplicated type B dissection should be managed medically with close monitoring for the development of complications [33]. For patients with extant or impending complication, endovascular and/or surgical

Table 4 Initial medical management of heart rate and blood pressure in acute aortic dissection

Agent	Dose
Beta blockers	
Esmolol	0.5–1 mg/kg bolus followed by continuous infusion at 0.10–0.20 mg/kg/min
Propranolol	0.05–0.15 mg/kg bolus every 4–6 h
Metoprolol	1.25–15 mg bolus every 3–12 h
Combined alpha and beta blocker	
Labetalol	0.25–0.5 mg/kg bolus followed by continuous infusion at 2.0–6.0 mg/min
Non-dihydropyridine calcium channel blockers	
Verapamil	2.5–5 mg bolus followed by continuous infusion at 5–24 mg/h
Diltiazem	0.25–0.35 mg/kg bolus followed by continuous infusion at 5–15 mg/h
Vasodilators	
Sodium nitroprusside	0.25–10 µg/kg/min continuous infusion
Nitroglycerin	5–100 µg/min continuous infusion
Fenoldopam	0.01–1.6 µg/kg/min continuous infusion
Dihydropyridine calcium channel blocker	
Nicardipine	5–15 mg/h continuous infusion

Guidelines regarding the medical management of aortic dissection as described by Hiratzka et al. [4] and Erbel et al. [9]

management should be considered. In the IRAD registry, 73% of patients with acute type B dissection were managed medically, 15% received a surgical intervention, and 12% received a percutaneous intervention [16].

In-hospital mortality for patients with ischemic complications managed surgically was historically observed to be as high as 89%, prompting interest in less invasive techniques

[9, 34]. The first endovascular intervention for acute type B dissection was a balloon fenestration of the intimal flap in a patient with visceral ischemia performed in 1990 [35]. Endovascular techniques now provide an expanding range of interventions, including fenestration, covered stents at the site of dissection entry, ballooning and stenting at sites of complications, and stent-graft placement such as thoracic endovascular aortic repair (TEVAR). Often, a combination of these techniques is employed to provide rapid amelioration of static or dynamic obstruction of the true lumen with organ hypoperfusion. No completed randomized trials compare medical, endovascular, and/or surgical approaches among patients with acute type B dissection [36].

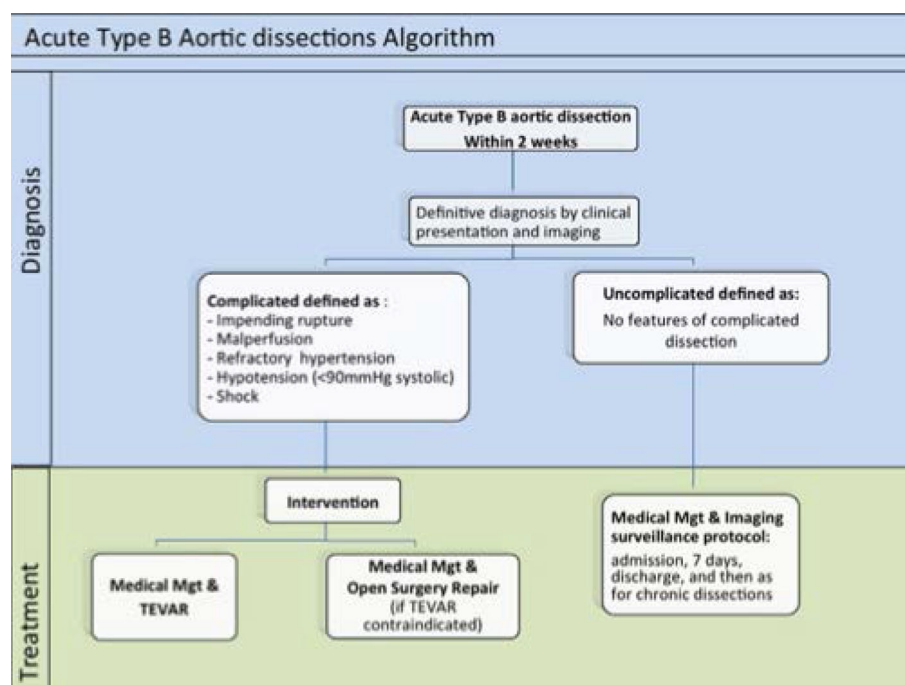
In a composite of 57 patients treated with an endovascular approach, adequate flow was restored in 90% of obstructed arteries with an average 30-day mortality of 10% [9]. A meta-analysis by Eggebrecht et al. [37] found a 98% success rate for endovascular stent-graft placement for acute and chronic type B dissection with 89% survival at 2 years and an 11% major complication rate. Patel et al. reviewed outcomes among 69 patients with acute type B dissection complicated by branch vessel hypoperfusion treated with flap fenestration and stenting of the true lumen and/or branch vessels. Adequate perfusion was restored to 96% of the affected vascular beds and mean survival was 84 months [38].

Rates of mortality and major morbidity appear to be lower with endovascular procedures compared with surgical intervention [39], but these data need to be cautiously interpreted due to the lack of large randomized trials and the tendency for the most complicated patients to be referred for surgical repair [36]. In a 2011 IRAD review,

patients with complicated type B dissections receiving percutaneous intervention had a 10% in-hospital mortality as compared with 29% for those receiving surgery and 21% for those managed medically alone [22]. Similarly, a case-control study by Brandt et al. among patients receiving either open surgery or endovascular stent-graft repair for dissections of the descending thoracic aorta found 5% 30-day mortality among the stent-graft group and 27% mortality among the open surgery cohort. The rate of post-procedure stroke or paraplegia was 5% in the stent-graft group and 9% in the surgery group [40]. Finally, in an IRAD cohort of 571 patients with acute type B dissection, Fattori et al. [33] observed a 20% in-hospital complication rate among those undergoing endovascular repair as opposed to a 40% in-hospital complication rate among those treated surgically. While these observational data show a consistent trend toward improved outcomes with endovascular repair, as was previously described, interpretation needs to account for the propensity for more complicated patients to be selected for surgery.

The Investigation of Stent Grafts in Aortic Dissection (INSTEAD) trial compared elective TEVAR plus optimal medical management in stable patients at least 2 weeks following an acute type B aortic dissection to optimal medical management alone. At 2 years there was no difference in all-cause mortality, aorta-related mortality, or neurologic complications, although the study was not powered to detect a difference in clinical events [41]. Interestingly, aortic remodeling with true lumen recovery and false lumen thrombosis was observed in 91% of TEVAR patients and only 19% of the medically managed cohort. Given the active remodeling observed, it is perhaps possible that a larger study population or longer follow-up period

Table 5 Acute type B aortic dissections algorithm



TEVAR thoracic endovascular aortic repair Reproduced with permission from Fattori et al. [36]

might reveal a clinical benefit to TEVAR in this population.

Expert consensus recommends medical management as the initial approach to uncomplicated acute type B aortic dissection. Endovascular or surgical repair should be considered in the setting of impending aortic rupture, end-organ hypoperfusion, dissection extension, hypotension, or failure to control hypertension or pain [4, 13], with TEVAR recommended as first line management of complicated acute type B dissection where available [33]. This treatment strategy is elaborated in algorithmic form from the 2013 American College of Cardiology consensus statement in Table 5 [36] and considerations for endovascular and surgical repair are summarized in Table 6 [34].

The patient presented here was initially managed medically and observed closely for the development of complications. When renal and limb hypoperfusion were noted, consideration was given to surgical and

percutaneous options for intervention. Because the issue of primary importance at the time of the first intervention was compression of the true lumen by the false lumen, and because endovascular management is associated with lower morbidity than open procedures, the decision was made to proceed first with fenestration with the goal of relieving the trans-membrane pressure gradient. When this approach succeeded initially but then failed to provide a lasting solution, it was felt that completion of the fenestration with what was functionally an endarterectomy would provide definitive relief of flow limitation in the true lumen. Open axillary-femoral bypass and TEVAR were also considered, but it was anticipated that adequate flow could be restored without the incumbent morbidity of a more involved open or endovascular intervention. Given the current landscape of numerous options for intervention and lack of definitive evidence for weighing the likelihood of success and risk of complication, randomized

Table 6 Considerations for surgical, medical, and interventional therapy in aortic dissection**Medical therapy**

Treatment of choice in uncomplicated type B dissection

Stable, isolated arch dissection

Stable type B dissection (chronic, ≥ 2 weeks after onset)

Interventional therapy

Stent-grafts to seal entry to false lumen and to enlarge compressed true lumen

Unstable type B dissection

Malperfusion syndrome

Balloon fenestration

Surgery

Treatment of choice for type A dissection

Consider for acute type B dissection complicated by Marfan's syndrome

Rupture or impending rupture (consider interventional therapy)

Progression or compromise of vital organs (consider interventional therapy)

Considerations for surgical, medical, and interventional therapy in aortic dissection as described by Nienaber et al. [34]

trials are needed comparing these techniques to guide management in situations such as these.

FOLLOW-UP

The most critical issue following the acute hospitalization is close outpatient management of blood pressure. Follow-up imaging is recommended after acute dissection at 7 days, hospital discharge, 6 weeks, 3 months, 6 months, and 12 months, and then every 12 months thereafter. Consideration of imaging modality is similar to that with acute dissection, save for the improved patient

stability and reduced urgency. Primary objectives of follow-up imaging are to assess for aortic expansion, aneurysm, and vascular leak at sites of intervention [9].

CONCLUSION

The aging populations of developed countries and the ongoing demographic transitions of developing nations will likely ensure that cardiovascular disease remains the leading cause of morbidity and mortality worldwide for the near future. Aortic pathology, including acute aortic dissection, remains an underappreciated yet important contributor to this disease burden. Progress continues to be made in diagnosis and management, particularly with respect to endovascular repair. As these techniques evolve, further comparisons of efficacy and safety will be needed to guide treatment decisions in this complicated and frequently unstable patient group. While population-level reduction of cardiovascular risk factors is ultimately required to prevent the development of aortic disease, high clinical suspicion and prompt diagnosis remain critical to limit the devastating sequelae of acute aortic syndromes.

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