BMJ Best Practice

Aortic dissection

The right clinical information, right where it's needed

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Summary

- A medical emergency resulting from a tear in the aortic wall intima, which causes blood flow into a new false channel composed of the inner and outer layers of the media. May propagate in an antegrade or retrograde direction, or both.
- Typically presents in men older than 50, with sudden onset of severe ripping or tearing substernal or interscapular pain.
- May present with syncope, heart/renal failure, or mesenteric or limb ischaemia; O2/ALS protocol and haemodynamic support should be instituted without delay when the condition is suspected.
- Diagnostic modalities include CT scan, MRI, or trans-thoracic/trans-oesophageal echocardiography.
- Involvement of the ascending aorta and/or arch warrants urgent surgical repair. Dissections of the descending aorta are managed medically with beta blockade; surgery in this group is reserved for those with end-organ malperfusion, persistent pain, or rupture.
- Lifelong surveillance is needed with regular imaging to detect aneurysmal degeneration of the remaining aorta, which may later require surgery.

Definition

Aortic dissection describes the condition when a separation has occurred in aortic wall intima, causing blood flow into a new false channel composed of the inner and outer layers of the media. Dissection most commonly occurs with a discrete intimal tear, but can occur without one. An aortic dissection is considered acute if the process is less than 14 days old.[1]

Epidemiology

The worldwide incidence of aortic dissection is 0.5 to 2.95 cases per 100,000 people yearly. The incidence in the US is 0.2 to 0.8 cases per 100,000 people yearly, resulting in about 2000 new cases each year. The highest rate is in Italy with 4.04 cases per 100,000 per year.[4] Men are predominantly affected, typically older than 50 years.

Aetiology

Aortic dissection results from an intimal tear that extends into the media of the aortic wall. Cystic medial degeneration predisposes to intimal disruption and is characterised by elastin, collagen, and smooth muscle breakdown in the lamina media. Bleeding from the vasa vasorum can also lead to this condition.

Inherited conditions that lead to medial degeneration provide a morphological substrate for developing aortic dissection. Marfan's syndrome and Ehlers-Danlos syndrome lead to weakening of the media, thus predisposing to aortic dilation and dissection. Bicuspid aortic valve may be associated with a non-specific connective-tissue disease and thus aortic aneurysms and dissection. Aortic atherosclerosis with dilation, and inflammatory or traumatic conditions or infections, may also predispose to aneurysmal degeneration and dissection. Although rare, iatrogenic causes include aortic manipulation associated with cardiac surgery or interventional procedures.[5] It is unclear if these iatrogenic complications occur in patients already predisposed by the aetiologies described above.
[Fig-4]

[Fig-5]

Pathophysiology

An intimal tear is the initial event, with subsequent degeneration of the medial layer of the aortic wall. Blood then passes through the media, propagating distally or proximally and creating a false lumen. As the dissection propagates, flow through the false lumen can occlude flow through branches of the aorta, including the coronary, brachiocephalic, intercostal, visceral and renal, or iliac vessels.

The intimal tears of dissection most commonly occur just above the sinotubular junction or just distal to the left subclavian artery.[6] Regardless of where tears occur in the aorta, there may be both a retrograde and antegrade extension of the dissection. Retrograde dissections starting in the ascending aorta can lead to aortic incompetence by separating the aortic valve from the aortic root.

Static narrowing of side-branches occurs when the line of dissection intersects the vessel origin and the aortic haematoma has propagated into the vessel wall, leading to stenosis or occlusion of the side-branch. Dynamic compression occurs when the dissection flap is on the opposite side of the side-branch

origin. Obstruction of the side-branch occurs during diastole, when the true lumen collapses and the intimal flap closes over the ostium of the branch vessel. Flow is restored during systole. Both static and dynamic compression of a side-branch or a combination of both can lead to total flow occlusion and end-organ ischaemia. Subsequent clinical manifestations occur depending on the extent of propagation with subsequent organ malperfusion.[1]

Laplace's law describes wall stress as directly proportional to pressure and radius, and inversely proportional to wall thickness. Thus, factors that weaken the aortic wall, particularly the lamina media, lead to increased risk of aneurysm formation and dissection, and a cycle of increasing wall stress.

Classification

Stanford[2]

- Type A: Dissection involves the ascending aorta with or without involvement of the arch and descending aorta.
  [Fig-1]
- Type B: Dissection does not involve the ascending aorta. Predominantly involves only the descending thoracic (distal to the left subclavian artery) and/or abdominal aorta.
  [Fig-2]

DeBakey[3]

- Type 1: Tear originates in the ascending aorta and involves the ascending and arch aorta, and variable amounts of the descending thoracic aorta.
- Type 2: Dissection is confined to the ascending aorta.
  [Fig-3]
- Type 3: Tear originates distal to the left subclavian artery and extends through the thoracic aorta (3A) or extends beyond the visceral segment (3B).

Primary prevention

Hypertension should be assessed for and treated appropriately in all patients. Other cardiovascular risk factors (e.g., dyslipidaemia and diabetes mellitus) should also be intensively managed. Smokers should be encouraged to cease smoking.

Secondary prevention

Patients with known Marfan's or Ehlers-Danlos syndrome should be regularly monitored with echocardiography for aortic root aneurysm (predisposing to dissection).

Blood pressure control to less than 150 mmHg (preferably less than 120 mmHg) systolic and less than 90 mmHg is recommended. No data support exact goals, but shear forces are excessive when systolic BP exceeds 150 mmHg. Heart rate should be maintained less than 80 bpm. Beta blockade is first-line treatment.

Case history

Case history #1

A 59-year-old man presents to the emergency department with a sudden onset of excruciating chest pain, which he describes as tearing. There is a history of hypertension. On physical examination, his heart rate is 95 bpm. BP is 195/90 mmHg in the right arm and 160/80 mmHg in the left arm. Pulses are absent in the right leg and diminished in the left.

Other presentations

The pain of aortic dissection may migrate through the thorax or abdomen. Symptoms of stroke or visceral or acute limb ischaemia may be present. Patients may be haemodynamically stable or in hypovolaemic shock. Occasionally, depressed mental status or neurological changes, limb pain, paraesthesias or weakness, paraplegia, or syncope are presenting symptoms. Infrequently, patients present without pain. There may be signs of heart failure, pericardial tamponade, or left pleural effusion. Younger patients can present with a recent history of heavy lifting or cocaine use. Patients with connective-tissue disorders such as Marfan's syndrome often present in their 30s.[1]
[Fig-4]

[Fig-5]

Step-by-step diagnostic approach

Aortic dissection should be suspected when an abrupt onset of tearing or ripping chest pain is reported. Ten per 100,000 Americans will have an acute aortic dissection, and missing the diagnosis in those with dissection may be catastrophic, hence the importance of further investigation.[13]

The usual presentation is a man in his 50s, but the condition may occur in younger patients who have Marfan's syndrome, Ehlers-Danlos syndrome, or overlap connective-tissue disorders. Because of the severity of the condition, the diagnosis should be considered in young patients, even when predisposing factors are absent.
[Fig-4]

[Fig-5]

Signs and symptoms

Most patients have prior HTN, often poorly controlled. Younger patients may have a connective-tissue disorder, or a recent history of heavy lifting or cocaine use. Family history may reveal aortic aneurysms, dissection, or connective-tissue disorder.

The pain may be located retrosternally, interscapularly, or in the lower back. Anterior chest pain is associated with an ascending dissection; interscapular pain occurs with a descending dissection. Pain may migrate through the thorax or abdomen, and the location of pain may change with time as the dissection extends. A minority of patients present with syncope or without pain.

Patients may be haemodynamically stable or in hypovolaemic shock. BP differences in the upper extremities or pulse deficits in the lower extremities should be sought. Neurological deficits may indicate involvement of cerebral or intercostal vessels. There may be depressed mental status, limb pain, paraesthesias or weakness, or paraplegia. Symptoms of visceral ischaemia may be present. Occasionally, a diastolic decrescendo murmur may be discovered, indicating aortic insufficiency. There may be symptoms or signs of heart failure, pericardial tamponade, or a left pleural effusion, such as dyspnoea.

Tests

Initial work-up includes CXR, ECG, and cardiac enzymes to exclude pneumonia or MI. Bloods including metabolic panel, FBC, and type and cross should also be requested. Despite a high sensitivity, D-dimer is not recommended as the sole screening tool for acute aortic dissection (while negative D-dimer may be helpful to rule out aortic dissection in low-risk patients, positive D-dimer lacks specificity, thereby limiting its clinical role).[14] [13] However, it will be of value when considering the differential diagnosis (e.g., pulmonary embolus).[13] Other biomarkers with the potential to assist in the diagnosis of aortic dissection include CRP, elastin degradation products (sELAF), calponin, and smooth muscle myosin heavy chain (smMHC), but none of these have been validated.[15]

If aortic dissection is suspected because of history or widened mediastinum on CXR, CT scan is the primary modality used for diagnosis, with a sensitivity greater than 90% and specificity greater than 85%.[16]
[Fig-2]

The diagnosis is made by imaging an intimal flap separating 2 lumens. If the false lumen is completely thrombosed, central displacement of the intimal flap, calcification, or separation of intimal layers are definitive signs of aortic dissection. CT also allows visualisation of the extent of dissection and involvement of side-branches.
[Fig-5]

[Fig-3]

Trans-thoracic echocardiography (TTE) and/or trans-oesophageal echocardiography (TEE) may be done in the emergency department, ICU, or theatre for acute proximal dissections if the patient is clinically unstable and there is any question about the diagnosis, or if CT is unavailable.[17]
[Fig-4]

Sensitivity and specificity are 77% to 80% and 93% to 96%, respectively.[16]

Also for type A dissections, trans-oesophageal echocardiography may be done in the ICU or theatre to confirm the diagnosis and better evaluate the aortic valve. Sensitivity and specificity are higher than for TTE.

MRI is the most accurate, sensitive, and specific test, but is rarely used in the acute setting because it is more difficult to obtain than CT.[16]

In the setting of type B dissections, if medical therapy fails and surgery is required, intravascular ultrasound helps define morphology of the dissection and assists in the treatment plan.

Risk factors

Strong

HTN

- The International Registry of Acute Aortic Dissection found that 72% of patients with aortic dissection had a history of HTN and 32% had a history of atherosclerosis.[7]

atherosclerotic aneurysmal disease

- Approximately 1% of sudden deaths are attributable to aortic rupture. Of these, two-thirds are due to dissection and one third to atherosclerotic aneurysms.[8]

Marfan's syndrome

- Predisposes to both aneurysms and/or dissections, presumably related to weakness of the aortic wall.[1]
  [Fig-4]

  [Fig-5]

Ehlers-Danlos syndrome

- Type IV predisposes to both aneurysms and/or dissections, presumably related to weakness of the aortic wall.[1]

bicuspid aortic valve

- Predisposes to both aneurysms and/or dissections, presumably related to weakness of the aortic wall.[1]

annulo-aortic ectasia

- Predisposes to both aneurysms and/or dissections, presumably related to weakness of the aortic wall.

coarctation

- Untreated coarctation in adults is associated with dissection and is probably related to longstanding HTN.

smoking

- Tobacco use is closely associated with atherosclerotic and vascular disease and therefore dissections.

FHx of aortic aneurysm or dissection

Weak

older age

- Usual presentation is a man in his 50s. However, aortic dissection can occur in younger patients, even in the absence of connective-tissue disorders, and should be considered given the severity of the process.[1]

giant cell arteritis

- Can weaken the media of the aorta and lead to expansion or dissection.

overlap connective-tissue disorders

- Clinical or laboratory features of several connective tissue diseases such as rheumatoid arthritis, SLE, systemic sclerosis, polymyositis, dermatomyositis, and Sjogren's syndrome, without meeting the criteria for a specific diagnosis.

surgical/catheter manipulation

- Manipulation of at-risk aortas: examples of procedures include cardiac catheterisation, aortic valve replacement, or thoracic stent-grafting.[5] [9]

cocaine/amfetamine use

- Acute HTN, vasoconstriction, increased stroke volume, and vasospasm as a result of the misuse of these agents may lead to aortic dissection. Case reports involving young patients have been described, and the increased risk associated with misuse of these substances has also been demonstrated using the Nationwide Inpatient Sample.[10] [11]

heavy lifting

- Typically confined to young patients and theoretically attributed to the elevated aortic pressure during straining.

pregnancy

- Case reports; for example, in conjunction with Marfan's syndrome.[12]

History & examination factors

Key diagnostic factors

features of Marfan's/Ehlers-Danlos syndromes (common)

- Patients may exhibit typical marfanoid features including tall stature, arachnodactyly, pectus excavatum, hypermobile joints, and narrow face. Type IV Ehlers-Danlos syndrome predisposes to both aneurysms and/or dissections.

acute severe chest pain (common)

- Acute onset of a severe tearing or ripping chest pain suggests aortic dissection.
- May change location with time as the dissection extends. Anterior pain occurs with dissection of ascending aorta.

interscapular pain (common)

- Occurs with dissection of descending aorta.

left/right BP differential (common)

- A BP differential between the 2 arms is suggestive and a hallmark feature. Pulse differences in the lower limbs may also be evident.

pulse differential/deficit (common)

- A pulse differential or deficit between the 2 legs is suggestive.

diastolic murmur (common)

- Crescendo pattern, indicating aortic incompetence. Common in proximal dissections, but uncommon in distal dissections.

syncope (uncommon)

- Up to 20% of patients may present with syncope and no pain.[4]

hypotension (uncommon)

- Associated with cardiac tamponade and/or hypovolaemic shock.

Other diagnostic factors

hypertension (common)

- Due to pre-existing hypertensive condition or increased sympathetic drive.

dyspnoea (uncommon)

- May indicate new-onset heart failure because of acute aortic insufficiency during proximal dissections, or cardiac tamponade.

altered mental status (uncommon)

- Due to cerebral ischaemia.

paraplegia (uncommon)

- Due to compromise of intercostal vessels and subsequent spinal cord ischaemia.

hemiparesis/paraesthesia (uncommon)

- Due to cerebral or peripheral ischaemia.

abdominal pain (uncommon)

- Visceral ischaemia resulting from compromised organ perfusion.

limb pain/pallor (uncommon)

- Due to compromised limb perfusion.

left-sided decreased breath sounds/dullness (uncommon)

- Left pleural effusion.

Diagnostic tests

1st test to order

Test	Result
ECG • Important first-line test to look for evidence of myocardial ischaemia.	**ST segment depression may occur with acute dissection, but ST elevation occurs rarely**

Test	Result
CXR • Excludes other pulmonary causes of pain.	**may show widened mediastinum**
cardiac enzymes • Important to exclude MI; however, myocardial ischaemia and infarction can occur if the dissection extends to the coronary ostium.	**cardiac enzymes usually negative**
CT scan • Should be ordered as soon as diagnosis suspected. Should include chest, abdomen, and pelvis to visualise extent of the aneurysm. [Fig-5] [Fig-3] [Fig-2]	**intimal flap**
renal function tests • If renal perfusion compromised.	**elevated creatinine and BUN**
FBC • Anaemia may be present in the case of haemorrhage.	**reduced or normal**
type and cross • Surgical intervention/transfusion may be necessary in some cases.	**preparation for surgery**

Other tests to consider

Test	Result
D-dimer • Despite a high sensitivity, D-dimer is not recommended as the sole screening tool for acute aortic dissection (while negative D-dimer may be helpful to rule out aortic dissection in low-risk patients, positive D-dimer lacks specificity, thereby limiting its clinical role).[14] [13] However, it will be of value when considering the differential diagnosis (e.g., pulmonary embolus).[13] Ten per 100,000 Americans will have an acute aortic dissection, and missing the diagnosis in those with dissection may be catastrophic, hence the importance of further investigation.[13]	**positive**
trans-thoracic echocardiography (TTE) • Can be ordered as supplementary test, or in unstable patients when acute proximal dissection is suspected.	**intimal flap**
trans-oesophageal echocardiography • Can be done to confirm the diagnosis and better evaluate the aortic valve, or if CT unavailable. Sensitivity and specificity are higher than for TTE. [Fig-4]	**intimal flap**
MRI • Very accurate, but rarely used in the acute setting because difficult to obtain.	**intimal flap**

Test	Result
intravascular ultrasound • In the setting of type B dissections, if medical therapy fails and surgery required, helps define morphology of the dissection and assists in treatment plan.	**intimal flap**
smooth muscle myosin heavy chain protein • A protein released from damaged aortic medial smooth muscle.[15] [18]	**elevated**

Differential diagnosis

Condition	Differentiating signs / symptoms	Differentiating tests
Acute coronary syndrome	• Chest pain is typically pressing. • There may be a history of prior exertional chest pain.	• ECG and troponin T may indicate myocardial infarction or ischaemia. • ST segment depression may occur in acute dissection, but ST elevation rare.
Pericarditis	• Chest pain typically pleuritic.	• ECG typically shows diffuse ST elevation.
Aortic aneurysm	• Stable (non-dissecting and non-leaking) aneurysms are asymptomatic. Diagnosis is usually incidental to work-up for another entity.	• CT scan of the chest does not show dissection.
Musculoskeletal pain	• Pain may be reproducible on palpation of the affected area.	• CT scan of the chest does not show dissection.
Pulmonary embolus	• Dyspnoea, hypoxia, and pleuritic chest pain. • There may be evidence of deep vein thrombosis: for example, calf swelling or tenderness.	• CT scan of the chest shows pulmonary embolus.
Mediastinal tumour	• Possible cough or haemoptysis.	• CT scan of the chest shows evidence of tumour.

Step-by-step treatment approach

Appropriate treatment is determined by accurate diagnosis of aortic dissection according to the following criteria:

- Type A (ascending)
  [Fig-1]
- Uncomplicated type B (descending)
  [Fig-2]
- Type B (descending) with end-organ ischaemia.

Initial management

Local resuscitation protocols should be followed. Supplemental high-flow oxygen and haemodynamic support with IV fluid resuscitation and judicious use of inotropes is recommended in cases of incipient renal failure and hypovolaemic shock.

Initial management of both type A and B dissections involves intensive monitoring and anti-impulse therapy. IV beta blockade is used to achieve a heart rate less than 80 bpm and systolic BP less than 110 mmHg. If beta blockade alone fails, vasodilator therapy is added.

To decrease sympathetic tone and facilitate hemodynamic stability, pain should be controlled with intravenous opioids. Morphine causes vasodilation and reduces heart rate by increasing vagal tone.

Type A dissection

Type A dissections require urgent surgical replacement of the diseased aorta. Depending on the extent of retrograde extension, the aortic valve may or may not need to be repaired or replaced in order to prevent cardiac tamponade or fatal exsanguination from aortic rupture.

Type B dissection

Surgical or endovascular intervention is required if the patient's course is complicated by rupture, visceral or extremity ischaemia, expansion, or persistent pain. Although both open and endovascular therapies are acceptable options, the endovascular approach is gaining preference over the open technique for patients presenting with complications.[19]

For uncomplicated type B aortic dissections, surgical repair has no proven superiority over medical treatment in stable patients. However, updated analysis of data from the International Registry of Aortic Dissection suggests that there may be some benefit to endovascular intervention in patients with refractory pain and hypertension.[20] Additionally, the recently published results of the INSTEAD-XL trial have demonstrated that the use of endovascular stent-grafting in uncomplicated subacute patients results in improved survival and aortic remodelling compared with optimal medical therapy at 5 years.[21]

Continued treatment

Blood pressure control is continued after discharge from hospital.[22] Beta-blockers and ACE inhibitors are usually required, with additional antihypertensives such as diuretics or calcium-channel blockers used if necessary. At least 40% of patients will require combination treatment to control blood pressure.

Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

Presumptive (summary)

Patient group	Tx line	Treatment
haemodynamically unstable: suspected aortic dissection	1st	haemodynamic support + O2 + ALS

Acute (summary)

Patient group	Tx line	Treatment
confirmed aortic dissection	1st	beta blockade
▪ confirmed aortic dissection	plus	opioid analgesia
▪ beta blockade insufficient	plus	vasodilators
▪ type A; or type B with complications (rupture, visceral/extremity ischaemia, expansion, or persistent pain)	plus	open surgery or endovascular stent-graft repair

Ongoing (summary)

Patient group	Tx line	Treatment
after hospital discharge	1st	antihypertensives

Treatment options

Presumptive

Patient group	Tx line	Treatment
haemodynamically unstable: suspected aortic dissection	**1st**	**haemodynamic support + O2 + ALS** » Local resuscitation protocols should be followed. » Supplemental high-flow oxygen and haemodynamic support with IV fluid resuscitation and judicious use of inotropes is recommended in cases of incipient renal failure and hypovolaemic shock. **Primary options** » noradrenaline: 0.5 to 1 microgram/min intravenously initially, adjust according to response, usual dose range 2-12 micrograms/min, maximum 30 micrograms/min **--AND/OR--** » dobutamine: 0.5 to 1 microgram/kg/min intravenously initially, adjust according to response, usual dose range 2-20 micrograms/kg/min, maximum 40 micrograms/kg/min

Acute

Patient group	Tx line	Treatment
confirmed aortic dissection	**1st**	**beta blockade** » Intravenous beta blockade is essential to reduce the continued pulsatile force (dP/dt) on the already-thinned walls of the false channel. Beta blockade may prevent further propagation of the dissection and reduces the risk of acute rupture. The risk of therapy is low. **Primary options** » labetalol: 1-5 mg/min intravenous infusion Goal heart rate is 60-75 bpm and systolic BP 90-120 mmHg. **OR** **Primary options**

Acute

Patient group	Tx line	Treatment
		» esmolol: 500 micrograms/kg intravenously initially, followed by 50 micrograms/kg/min for 4 min, may repeat loading dose and increase infusion up to 200 micrograms/kg/min if necessary Adjust maintenance infusion to achieve goal blood pressure.
		OR
		Primary options
		» metoprolol: 5 mg intravenously every 5-10 minutes, maximum 15 mg/total dose
confirmed aortic dissection	**plus**	**opioid analgesia**
		» Pain control is an important first-line therapy to reduce sympathetic tone and facilitate haemodynamic stability.
		Primary options
		» morphine: 2-5 mg intravenously every 5-30 minutes as needed
beta blockade insufficient	**plus**	**vasodilators**
		» Intravenous antihypertensive vasodilator therapy includes sodium nitroprusside and calcium-channel blockers. This therapy is used if beta blockade is insufficient for control of HTN. It will further reduce systolic blood pressure to 100 to 120 mmHg if beta blockade is inadequate. Risk of therapy is minimal.
		Primary options
		» nitroprusside: 0.3 to 0.5 micrograms/kg/min intravenously initially increase by 0.5 micrograms/kg/min increments; maximum 10 micrograms/kg/min
		OR
		Secondary options
		» diltiazem: 0.25 mg/kg intravenous bolus initially, followed by 5-10 mg/hour infusion; maximum 15 mg/hour
type A; or type B with complications (rupture, visceral/extremity ischaemia, expansion, or persistent pain)	**plus**	**open surgery or endovascular stent-graft repair**
		» Type A dissection involves the ascending aorta with or without involvement of the arch and descending aorta. Open surgery for type A dissection, with replacement of the ascending aorta, is performed immediately upon diagnosis. Depending on the extent of retrograde extension,

Acute

Patient group	Tx line	Treatment
		the aortic valve may or may not need to be repaired or replaced. » Type B dissection involves only the descending thoracic aorta (distal to the left subclavian artery) and/or abdominal aorta. For the complicated type B dissections, the goal of open surgery is to resect/cover the entry tear and re-establish flow into compromised branch vessels. Although both open and endovascular therapies are acceptable options, the endovascular approach with endovascular stent-grafting is gaining preference over the open technique for patients presenting with complications.[19] For uncomplicated type B aortic dissections, surgical repair has no proven superiority over medical treatment in stable patients. However, updated analysis of data from the International Registry of Aortic Dissection suggests that there may be some benefit to endovascular intervention in patients with refractory pain and hypertension.[20] Additionally, the recently published results of the INSTEAD-XL trial have demonstrated that the use of endovascular stent-grafting in uncomplicated subacute patients results in improved survival and aortic remodelling compared with optimal medical therapy at 5 years.[21] » There is a growing body of experience with endovascular interventions for the treatment of complicated type B dissections, including fenestration and stenting.[23] [24] [25] Several series have demonstrated high technical success rates for endovascular stenting to seal proximal entry tears. This promotes false lumen thrombosis and aortic remodelling. Static or dynamic side-branch obstruction can be relieved with additional endovascular stents. Compromised branches can be treated with ostial bare stents or stent grafts that enlarge the compressed true lumen. Survival rates and neurological complications with endovascular treatment of type B dissections are favourable compared with those of open surgery. Long-term outcomes will need to be evaluated. In the US, there is currently no approved device with this specific indication. However, this approach is rapidly becoming the treatment of choice for complicated type B dissection, because of good outcomes compared with historical open surgical controls and reduced invasiveness compared with conventional surgery.[23] [25] [26] [Fig-1]

Acute

Patient group	Tx line	Treatment
		[Fig-2]

Ongoing

Patient group	Tx line	Treatment
after hospital discharge	**1st**	**antihypertensives** » No patient is considered cured. Blood pressure control is continued after discharge from hospital. Beta-blockers and ACE inhibitors are usually required, with additional antihypertensives such as diuretics or calcium-channel blockers used if necessary. At least 40% of patients will require combination treatment to control blood pressure. **Primary options** » metoprolol: 100-450 mg/day orally (immediate-release) given in 2-3 divided doses; 25-100 mg orally (modified-release) once daily **-and/or-** » enalapril: 5-40 mg orally once daily or given in 2 divided doses **OR** **Secondary options** » metoprolol: 100-450 mg/day orally (immediate-release) given in 2-3 divided doses; 25-100 mg orally (modified-release) once daily **-and-** » enalapril: 5-40 mg orally once daily or given in 2 divided doses **--AND--** » hydrochlorothiazide: 12.5 to 50 mg orally once daily **-and/or-** » nifedipine: 30-60 mg orally (extended-release) once daily

Emerging

Endovascular stent-grafting of uncomplicated type B dissections

For uncomplicated type B dissection, there is currently no evidence to support the routine use of thoracic stent grafts. In fact, the INSTEAD randomised trial comparing best medical therapy to early stent-grafting had a trend towards better outcomes in the medical arm, but an appreciable number of patients crossed over to require stent-grafting.[27] More recent evidence suggests that there may be a subset of patients at high risk for early complications predictable by features on CT.[24] [28] Anticipated results of the ADSORB trial may shed more light on the appropriate use of stent-grafting in uncomplicated patients.[29]

Endovascular stent-grafting of chronic type B dissections with aneurysm

Although there is a growing experience with many case series describing the safety of placing stent grafts into the true lumen of patients with chronic aortic dissection, there are no long-term data to support this treatment as a method to reduce the risk of aneurysmal rupture. The controversy surrounds the mechanism of repair because the patients are likely to have persistent pressurisation of the false channel due to distal fenestrations. Nonetheless, for high-risk patients with this chronic disease, some have advocated the use of endovascular treatments and point to evidence that thrombosis occurs in the false lumen within the treated segment of the aorta in a vast majority of patients.[30]

Hybrid surgical repair/frozen elephant trunk operation

For type A dissection, there is a late risk of aortic dilation and other complications of up to 50%. New surgical approaches combining open repair of the proximal aorta under deep hypothermic circulatory arrest combined with open placement of thoracic stent grafts into the distal aortic arch and upper descending aorta have been shown to increase the chances for late thrombosis and remodelling of the aortic false lumen. Longer term follow-up is necessary, but these techniques have the promise to redefine treatment of acute aortic dissection.[31] [32]

Endovascular stent-grafting of the ascending aorta

Although several case reports have demonstrated feasibility of stent-grafting the ascending aorta, there have been no larger series to confirm the safety of these techniques and it is very much investigational at this time.[33]

Recommendations

Monitoring

Imaging of the aorta should be performed before discharge. Subsequently, regularly scheduled imaging of the aorta should be performed at 6 months, then yearly, to follow aneurysmal degeneration that may require later intervention.[16] [38]

Patient instructions

Heavy lifting of more than 30 lb to 50 lb should be avoided. Aerobic exercise is acceptable after discussion with the physician. Patients should follow up for imaging and blood pressure control. Smoking cessation and control of hyperlipidaemia is important for overall vascular health. It is recommended that patients maintain a heart-healthy diet.

Complications

Complications	Timeframe	Likelihood
pericardial tamponade	**short term**	**high**
Occurs when an ascending aortic dissection extends proximally with rupture into the pericardial space. Emergency surgical repair of the aortic dissection is required. The team may wish to prepare the patient's chest for incision while the patient is awake in the event that the patient sustains cardiac arrest during the induction of anaesthesia.		
aortic incompetence	**short term**	**high**
Occurs when dissection propagates proximally, leading to loss of commissural support for the valve leaflets. Urgent surgical repair of the dissected aorta with aortic valve repair or replacement is required.		
myocardial infarction	**short term**	**low**
Occurs when the dissection propagates proximally and coronary ostial occlusion occurs. Urgent surgical repair of the aortic dissection with coronary reconstruction and/or coronary bypass grafting is required.		
aneurysmal degeneration/rupture	**variable**	**medium**
Complications from distal aortic dissection occur in 20% to 50% of patients. This occurs due to continued pulsatile force (dP/dt) on already-thinned walls of the false channel or new dissection. A multi-drug antihypertensive regimen including beta blockade to maintain systolic pressure less than 120 mmHg may alter the natural history of chronic dissection by diminishing the rate of aneurysmal dilation.[1] [35]		
regional ischaemia	**variable**	**low**

FOLLOW UP

Complications	Timeframe	Likelihood
Cerebral, renal, visceral, spinal cord, or lower-extremity ischaemia occur when the dissection propagates distally and true lumen occlusion occurs. Emergency surgical or endovascular repair of the dissected aorta with or without additional revascularisation of compromised branch vessels is required.		
left arm ischaemia/vertebral steal	**variable**	**low**
Occurs when the left subclavian artery is covered after endovascular aortic repair in approximately 15% of patients. The Society for Vascular Surgeons Practice Guidelines recommends routine preoperative revascularisation of the left subclavian artery for patients who need elective stent-graft repair where achievement of a proximal seal necessitates coverage of the left subclavian artery, and selective revascularisation in urgent indications.[36] [37]		

Prognosis

Syncope at presentation is usually associated with worse outcomes. A deadly triad of hypotension/shock (not syncope), lack of chest or back pain (presumably related to delay in diagnosis), and branch vessel involvement is also described.[34]

Left untreated, the natural history of proximal acute aortic dissection is of false channel rupture with fatal exsanguination in 50% to 60% of patients within 24 hours.[29]

Late degeneration of the dissected aorta into a false lumen aneurysm occurs in 30% to 50% of patients.[7] Following treatment, patients remain at risk for further aneurysmal degeneration of remaining diseased aorta. The 10-year survival after surgery of ascending aortic dissection is 52%. Freedom from re-operation at both 5 and 10 years ranges from 59% to 95%.[24]
[Fig-6]

Diagnostic guidelines

Europe

2014 ESC Guidelines on the diagnosis and treatment of aortic diseases

Published by: European Society of Cardiology **Last published:** 2014

Summary: Clinical practice guidelines covering acute and chronic aortic diseases of the thoracic and abdominal aorta in adults. Includes guidelines on diagnosis of aortic dissection.

Treatment guidelines

Europe

2014 ESC Guidelines on the diagnosis and treatment of aortic diseases

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Summary: Clinical practice guidelines covering acute and chronic aortic diseases of the thoracic and abdominal aorta in adults. Includes recommendations on treatment of aortic dissection.

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Key articles

- Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease. Circulation. 2010;121:e266-e369. Full text Abstract
- DeBakey ME, McCollum CH, Crawford ES, et al. Dissection and dissecting aneurysms of the aorta: twenty-year follow-up of five hundred twenty-seven patients treated surgically. Surgery. 1982;92:1118-1134. Abstract
- Svensson LG, Kouchoukos NT, Miller DC, et al. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. Ann Thorac Surg. 2008;85(suppl 1):S1-S41. Abstract

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Images

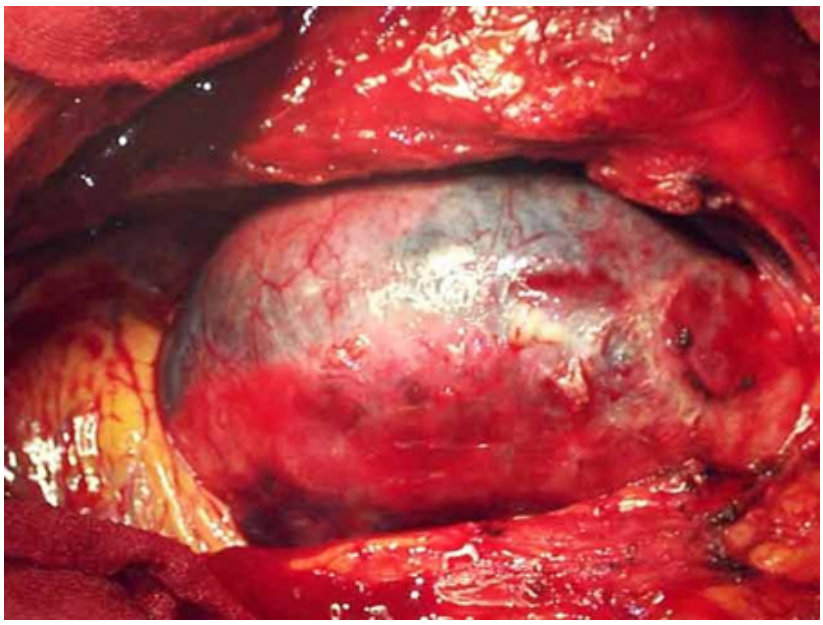

Figure 1: Proximal dissection

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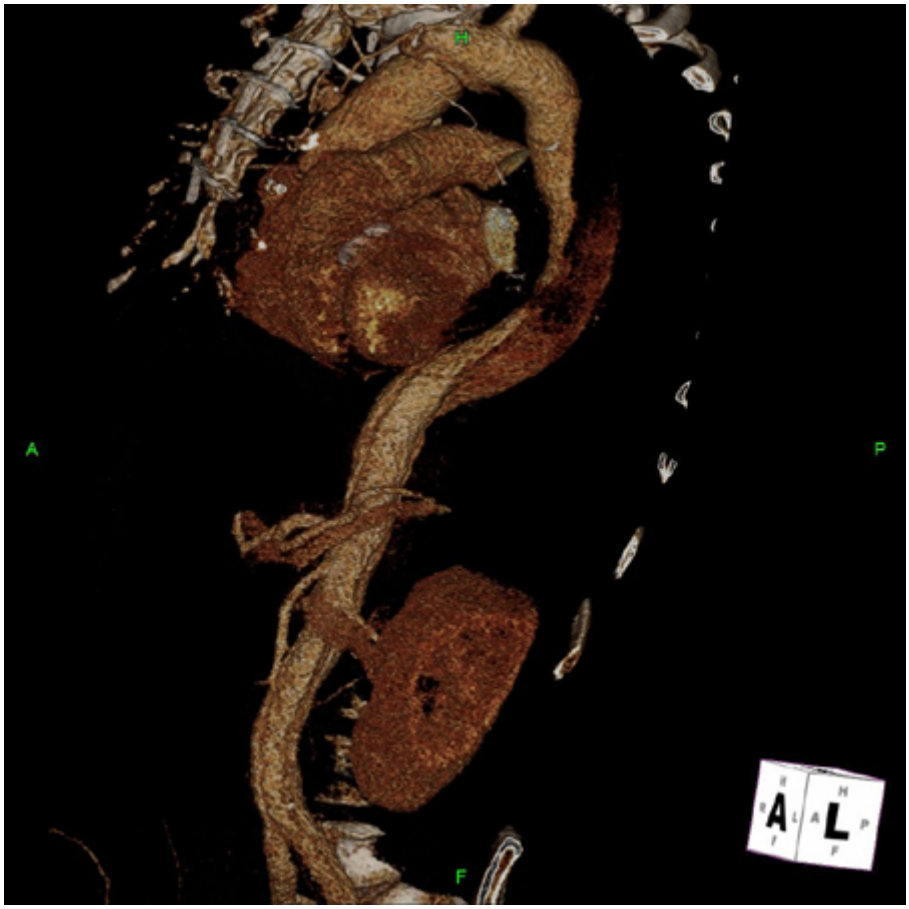

Figure 2: 3D CT, distal dissection

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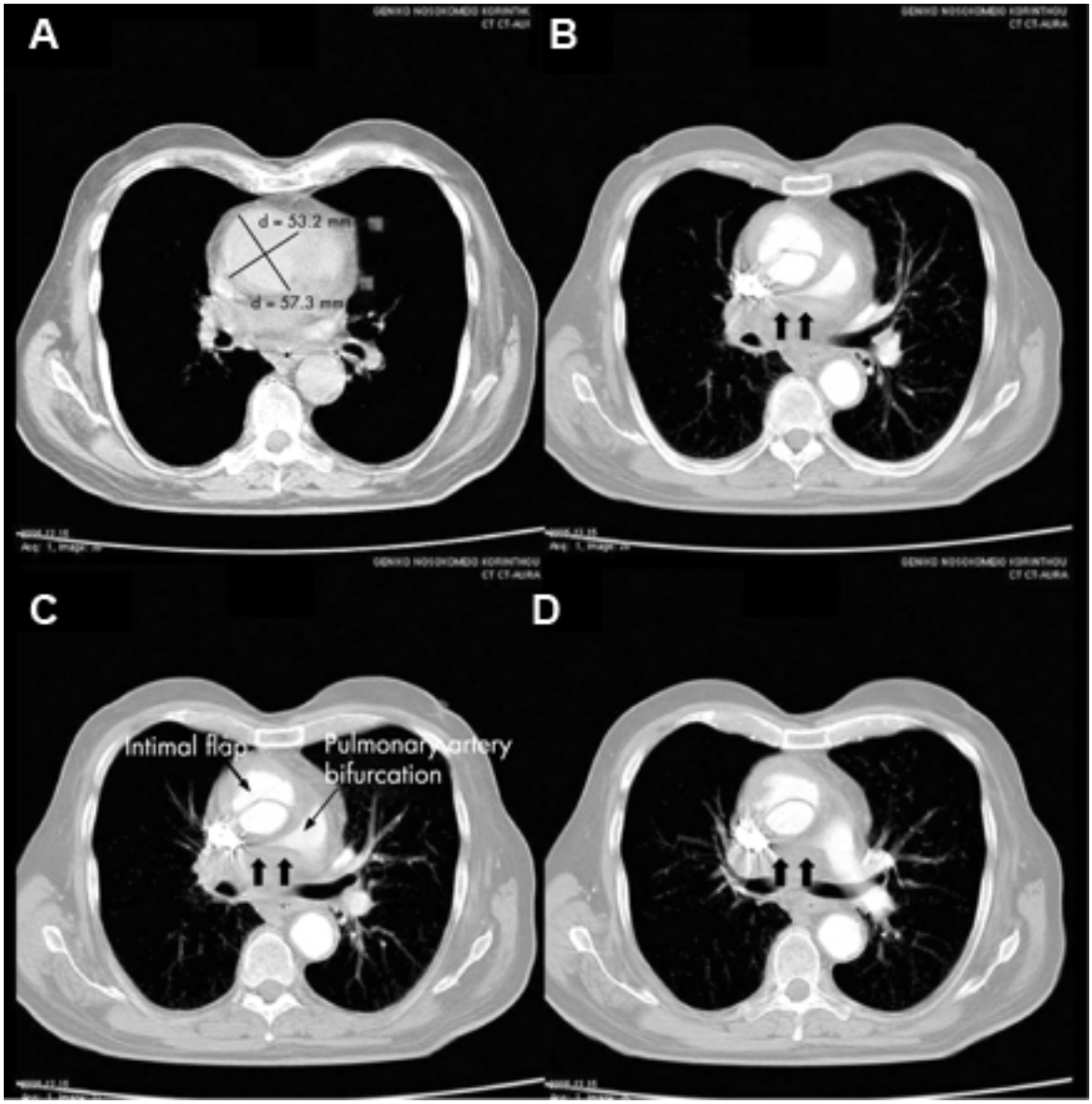

Figure 3: CT of a 71-year-old man showing type II dissecting aneurysm of the ascending aorta. Haematoma around the proximal segment of the ascending aorta (panels A-D) compressed the right pulmonary artery, almost occluding its patency and limiting the perfusion of the reciprocal lung

Stougiannos PN, Mytas DZ, Pyrgakis VN. The changing faces of aortic dissection: an unusual presentation mimicking pulmonary embolism. BMJ Case Reports 2009; doi:10.1136/bcr.2006.104414

Figure 4: Trans-oesophageal echocardiography (transverse aortic section) showing a circumferential dissection of the ascending aorta in a 30-year-old patient with features of Marfan's syndrome

Bouzas-Mosquera A, Solla-Buceta M, Fojón-Polanco S. Circumferential aortic dissection. BMJ Case Reports 2009; doi:10.1136/bcr.2007.049908

IMAGES

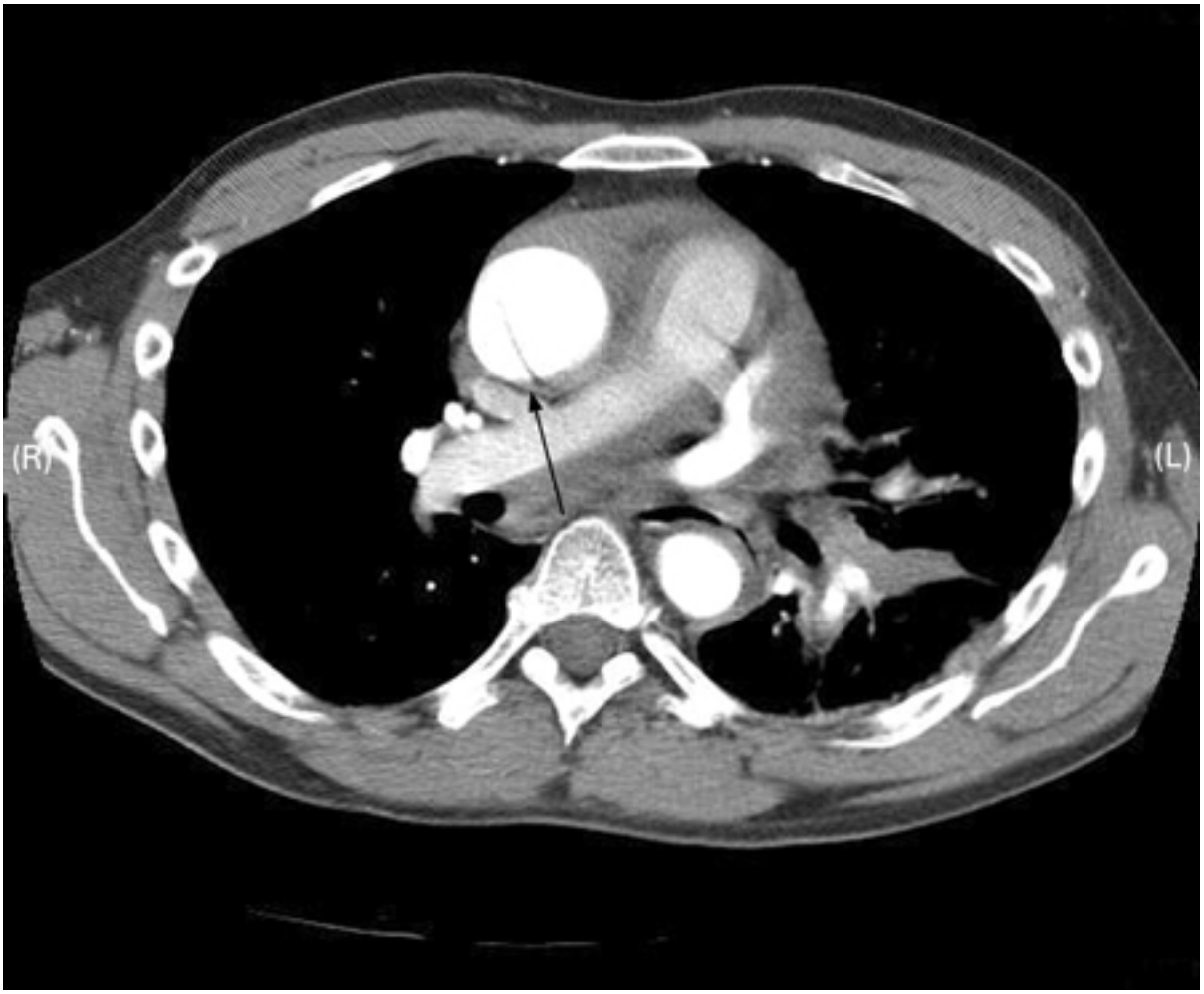

Figure 5: CT scan showing dissecting aneurysm in a 45-year-old patient with Marfan's syndrome experiencing chest pain

Sanyal K, Sabanathan K. Chest pain in Marfan syndrome. BMJ Case Reports 2009; doi:10.1136/bcr.07.2008.0431

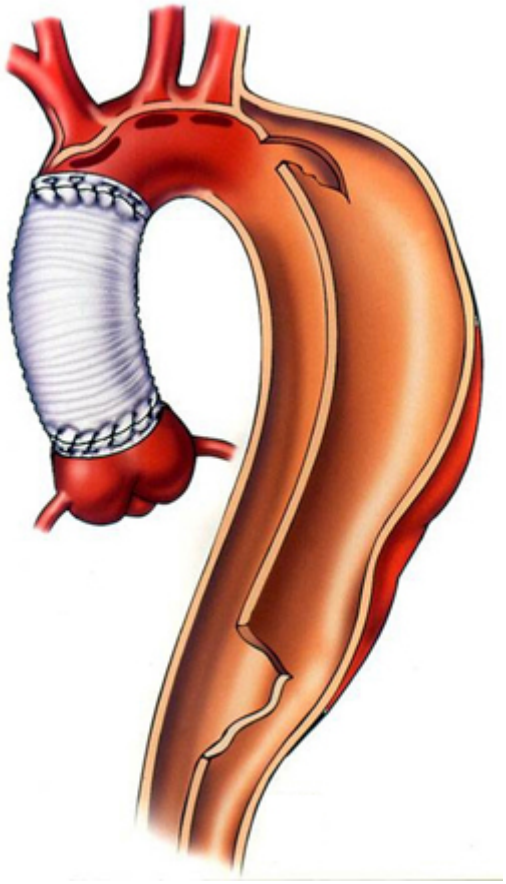

Figure 6: Dissection status post-proximal repair with late distal aneurysm

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