

IRISH ASSOCIATION FOR EMERGENCY MEDICINE



IAEM Clinical Guideline

Acute Thoracic Aortic Dissection Emergency Medicine Guideline

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DISCLAIMER

IAEM recognises that patients, their situations, Emergency Departments and staff all vary. These guidelines cannot cover all clinical scenarios. The ultimate responsibility for the interpretation and application of these guidelines, the use of current information and a patient's overall care and wellbeing resides with the treating clinician.

Revision History

Date	Version	Section	Summary of changes	Author
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GLOSSARY OF TERMS

AD	Aortic dissection
CT	Computed tomography
ECG	Electrocardiogram
mmHg	Millimetre of mercury
N/A	Not applicable
NR	Not recorded
MRI	Magnetic resonance imaging
SBP	Systolic blood pressure
SIRS	Systemic inflammatory response syndrome
TEVAR	Thoracic endovascular aortic repair
TOE	Trans-oesophageal echocardiogram

Acute Thoracic Aortic Dissection Emergency Medicine Guideline

INTRODUCTION

Thoracic aortic dissection (AD) is a rare but potentially fatal event resulting in separation of the layers of the tunica media by ingress of blood, producing a false lumen with variable proximal and distal extension.

AD can be challenging to diagnose. A high index of suspicion is required due to the high mortality rate. It is essential to get early senior help and radiology assistance.

PARAMETERS

Target audience: This guideline is intended for all Emergency Department (ED) staff-managing adult patients with a possible/ confirmed acute thoracic aortic dissection.

Patient population: The target patient population is adult patients presenting to the ED with possible/ confirmed acute thoracic aortic dissection.

AIMS

To provide an evidence-based guideline for the assessment and management of adult patients presenting to the ED with a possible/ confirmed acute thoracic aortic dissection.

EPIDEMIOLOGY

The incidence of AD is estimated at 3.5-6 per hundred thousand persons per year. This incidence is higher in men than in women and increases with age. The peak incidence is between 48-67 years of age. The prognosis is poorer in women, as a result of atypical presentation and delayed diagnosis.

The most common risk factor associated with AD is hypertension. Other risk factors include pre-existing aortic diseases or aortic valve disease, family history of aortic diseases, history of cardiac surgery, cigarette smoking, direct blunt chest trauma, pregnancy and use of illicit drugs (e.g. cocaine and amphetamines).

CLASSIFICATION

Classical anatomical aortic dissection is classified according to DeBakey or Stanford.

Table 1: DeBakey classification

Type I	Involves ascending aorta, aortic arch, and descending aorta
Type II	Confined to ascending aorta only
Type III	Confined to descending aorta distal to the left subclavian artery only
Type IIIa	Extends up to diaphragm
Type IIIb	Extends beyond the diaphragm

Table 2: Stanford classification

Type A	Involves the ascending aorta but may extend into the arch and descending aorta
Type B	Involves the descending aorta only

CLINICAL PRESENTATION AND COMPLICATIONS

Chest pain is the most frequent symptom of acute AD. Abrupt onset of severe chest and/or back pain is the most typical feature. The pain may be sharp, ripping, tearing, knife-like, and typically different from other causes of chest pain. Back pain and abdominal pain are more common in type B dissection. The main clinical presentations and complications of patients with acute AD are outlined in Table 3.

Table 3: Main clinical presentations and complications of patients with acute aortic dissection (Adopted from Erbel et al 2014).

	Type A	Type B
Chest pain	80%	70%
Back pain	40%	70%
Abrupt onset of pain	85%	85%
Migrating pain	<15%	20%
Aortic regurgitation	40-75%	N/A
Cardiac tamponade	<20%	N/A
Myocardial ischaemia or infarction	10-15%	10%
Heart failure	<10%	<5%
Pleural effusion	15%	20%
Syncope	15%	<5%
Major neurological deficit (coma/stroke)	<10%	<5%
Spinal cord injury	<1%	NR
Mesenteric ischaemia	<5%	NR
Acute renal failure	<20%	10%
Lower limb ischaemia	<10%	<10%

NR= Not recorded

Presence of predisposing factors (hypertension, Marfan syndrome, Ehlers-Danlos, pregnancy etc) should be included when taking the past medical history.

The ACC/American Heart Association (AHA) guidelines proposed a risk assessment tool based on three groups of information—predisposing conditions, pain features, and clinical examination (Table 4).

Table 4: Clinical data useful to assess the a priori probability of acute aortic syndrome (Adapted from Hiratzka et al. 2010)

High-risk conditions	High-risk pain features	High-risk examination features
<ul style="list-style-type: none"> • Marfan syndrome (or other connective tissue diseases) • Family history of aortic disease • Known aortic valve disease • Known thoracic aortic aneurysm • Previous aortic manipulation (including cardiac surgery) 	<ul style="list-style-type: none"> • Chest, back or abdominal pain described as any of the following: <ul style="list-style-type: none"> ○ Abrupt onset ○ Severe intensity ○ Ripping or tearing 	<ul style="list-style-type: none"> • Evidence of perfusion deficit <ul style="list-style-type: none"> ○ Pulse deficit ○ Systolic blood pressure difference ○ Focal neurological deficit (in conjunction with pain) • Aortic diastolic murmur (new and with pain) • Hypotension or shock

FEATURES IN THE EXAMINATION SUGGESTIVE OF AD

- Tachycardia
- Asymmetric (>15mm Hg difference between SBP in both arms) or absent blood pressure and pulses in the limbs
- Severe hypertension/hypotension
- New aortic regurgitation

- Features of cardiac tamponade (Beck’s triad (hypotension, muffled heart sounds, distended neck veins) and pulsus paradoxus)
- Neurological deficit, for example, hemiplegia from carotid artery occlusion

All patients presenting with acute neurological complaints should be questioned about the presence of chest, back, and/or abdominal pain and checked for peripheral pulse deficits as patients with dissection related neurologic pathology are less likely to report thoracic pain.

DIFFERENTIAL DIAGNOSES

Note, whilst there are several differential diagnoses (Table 5) for high-risk pain or examination features, out ruling aortic dissection is (time) critical due to its associated mortality and morbidity.

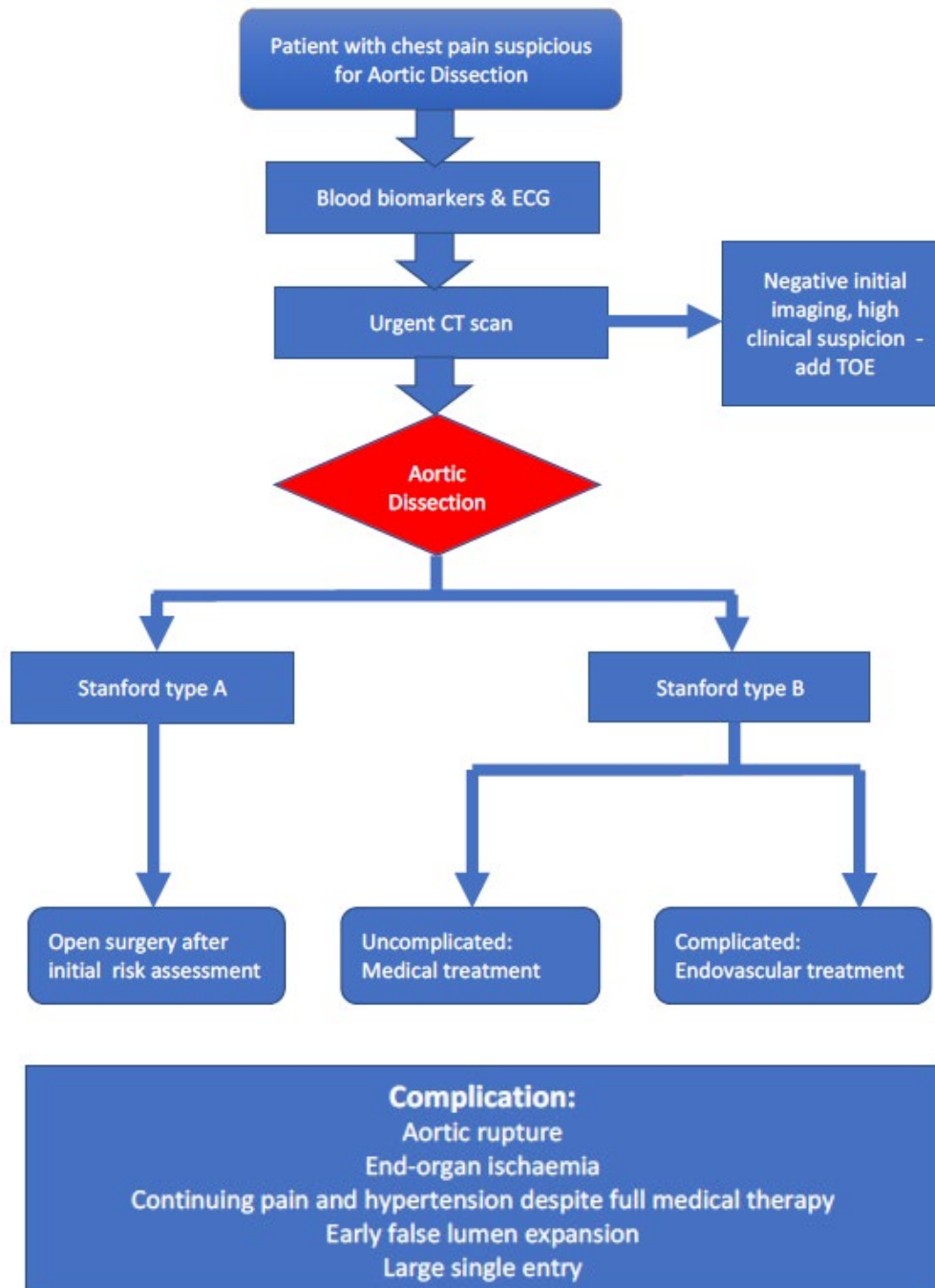
Table 5: Differential diagnoses for high risk pain or examination features (adapted from Hiratzka et al. 2010)

Chest pain	<ul style="list-style-type: none"> ○ Acute myocardial infarction ○ Pulmonary embolism ○ Spontaneous pneumothorax ○ Oesophageal rupture
Abdominal pain	<ul style="list-style-type: none"> ○ Renal/biliary colic ○ Bowel obstruction/ perforation ○ Non-dissection-related mesenteric ischaemia
Back pain	<ul style="list-style-type: none"> ○ Renal colic ○ Musculoskeletal pain ○ Intervertebral disk herniation
Pulse deficit	<ul style="list-style-type: none"> ○ Non-dissection-related embolic phenomena ○ Non-dissection-related arterial occlusion
Focal neurologic deficit	<ul style="list-style-type: none"> ○ Primary ischaemic stroke ○ Cauda equine syndrome

INVESTIGATIONS

The suggested diagnostic/management flow chart for AD is depicted in Figure 1.

Figure 1: Management flow chart for patient with chest pain suspicious for aortic dissection



(Adapted from Nienaber CA, Clough RE. 2015.)

Electrocardiogram (ECG)

An immediate ECG must be done to exclude acute myocardial infarction for which the treatment is very different and may involve transfer to the cath lab. About 20% of patients with type A dissection have ischaemic changes on ECG due to extension of the dissection into a coronary ostium.

Laboratory testing

In patients presenting with chest pain and suspicion of AD, the following laboratory tests are required for differential diagnosis or detection of complications (Table 6).

Table 6: Laboratory tests required for patients presenting with chest pain and suspicious for AD (Adapted from Erbel et al 2014)

Laboratory tests	To detect signs of:
Red blood cell count	Blood loss, bleeding anaemia
White blood cell count	Infection, inflammation (SIRS)
C-reactive protein	Inflammatory response
Creatine Kinase	Reperfusion injury, rhabdomyolysis
Troponin	Myocardial ischaemia, myocardial infarction
D-dimer	Aortic dissection, pulmonary embolism, thrombosis
Creatinine	Renal failure (existing or developing)
Liver function tests	Liver ischaemia, liver disease
Lactate	Bowel ischaemia, liver disease
Glucose	Diabetes mellitus
Blood gases	Metabolic disorder, oxygenation

A group and cross match sample should also be sent. D-dimer has a sensitivity of 51.7% to 100.0% and specificity of 32.8% to 89.2% for AD.

Diagnostic imaging in acute aortic dissection

Chest X-Ray findings which suggest AD include:

- Widened mediastinum (20% to 28% of dissections lack this finding)
- Small left pleural effusion
- Double shadow on aortic knuckle
- Intimal calcification separated more than 6 mm from the edge
- Cardiomegaly (pericardial effusion)

Echocardiogram findings (particularly transoesophageal echocardiogram [TOE]), which suggest AD, include:

- Dilated aorta
- Aortic regurgitation
- Aortic dissection flap
- Pericardial effusion

NB: The utility of electrocardiography and chest x-ray in suspected AD is limited to ruling out other pathologies that present with chest pain.

Note, ECG and Chest X-ray are often equivocal and CT or MRI should not be delayed if AD is suspected.

The main purpose of imaging in AD is the comprehensive assessment of the entire aorta ([Table 7](#)). Computed tomography (CT) is the most commonly used imaging technique for evaluation for AD in particular, because of its speed, widespread availability, and excellent sensitivity.

However, it noted that CT, Magnetic Resonance Imaging (MRI), and TOE are equally reliable for confirming or excluding the diagnosis of acute AD. However, CT and MRI are considered to be superior to TOE for the assessment of acute AD extension and branch involvement.

Table 7: Details required from imaging in acute aortic dissection (Adapted from Erbel et al 2014).

Aortic dissection
Visualisation of intimal flap
Extent of disease according to the aortic anatomic segmentation
Identification of the false and true lumens (if present)
Localisation of entry and re-entry tears (if present)
Identification of antegrade and/or retrograde aortic dissection
Identification grading, mechanism of aortic valve regurgitation
Involvement of side branches
Detection of malperfusion (low flow or no flow)
Detection of organ ischaemia (brain, myocardium, bowels, kidneys, etc)
Detection of pericardial effusion and its severity
Detection and extent of pleural effusion
Detection of peri-aortic bleeding
Signs of mediastinal bleeding
Co-existence of other aortic lesions: aneurysms, plaques, signs of inflammatory disease etc.

TREATMENT

NB: Urgent referral to local cardiothoracic surgery service in all confirmed cases.

General management

- Move patient to critical care area for close monitoring
- Careful intravenous fluid infusion
- Pain relief (morphine sulphate)
- BP titration to about 110–120 mmHg systolic with beta blockers as a first line

Definitive management

The European Society of Cardiology recommendations for treatment of AD are summarised in Table 8.

Table 8: ESC Recommendations for treatment of aortic dissection (Adapted from Erbel et al. 2014)

Recommendations	Class of recommendation	Level of Evidence
In all patients with AD, medical therapy including pain relief and blood pressure control is recommended.	I	C
In patients with Type A AD, urgent surgery is recommended.	I	B
In patients with acute Type A AD and organ malperfusion, a hybrid approach (i.e. ascending aorta and/or arch preplacement associated with any percutaneous aortic or branch artery procedure) should be considered.	IIa	B
In uncomplicated Type B AD, medical therapy should always be recommended.	I	C
In uncomplicated Type B AD, TEVAR should be considered.	IIa	B
In complicated Type B AD, TEVAR is recommended.	I	C
In complicated Type B AD, surgery may be considered.	IIb	C

Type A aortic dissection

Surgery is the treatment of choice. Acute Type A AD has a mortality of 50% within the first 48 hours if not operated on.

Type B aortic dissection

Uncomplicated Type B AD

Patients with uncomplicated Type B AD receive medical therapy to control pain, heart rate, and blood pressure, with close surveillance to identify signs of disease progression and/or malperfusion. Repetitive imaging is necessary, preferably with MRI or CT.

Thoracic endovascular aortic repair (TEVAR) should also be considered in uncomplicated Type B AD. Thoracic endovascular aortic repair (TEVAR) aims at stabilization of the dissected aorta, to prevent late complications by inducing aortic re-modelling processes.

Complicated Type B AD

In complicated Type B AD, TEVAR is recommended and surgery may be considered.

OUTCOME

In-hospital mortality for Type A has shown to be 22% and 14% for type B. A study looking at trends from the international registry of acute aortic dissection found that endovascular management increased from 7% to 31%.

LONG-TERM FOLLOW-UP

The 10-year actuarial survival rate among patients with AD who survive initial hospitalization ranges from 30% to 60%.

Late complications include progressive aortic insufficiency, recurrent dissection or progression of dissection. Regular assessment of the entire aorta should be done after discharge from endovascular or open treatment. Patients may require management of hypertension and lipid-lowering therapy. They require ongoing imaging surveillance. They should be counseled to stop smoking and about risk factor modification for atherosclerotic disease, avoiding cocaine and other stimulating drugs and to exercise. Patients may need genetic counseling.

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