Section 1 Cardiovascular

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Cardiovascular Conditions

Cardiovascular Presentations

1 Acute Coronary Syndromes

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AREAS OF CLINICAL PRACTICE

A condition in: Acute and emergency, and Cardiovascular.

INTRODUCTION

The term 'acute coronary syndromes' (ACS) includes a range of conditions including unstable angina, non-ST-segment-elevation myocardial infarction (NSTEMI) and ST-segment-elevation myocardial infarction (STEMI), caused by a sudden reduction of myocardial perfusion. Unstable angina and STEMI/NSTEMI will be covered in other topics, as will stable angina.

EPIDEMIOLOGY AND PATHOLOGY

Acute coronary syndrome is a significant cause of morbidity and mortality worldwide. In 2015, heart disease was the leading cause of death in men and the second most common cause of death in women in England. In 2015–16, more than 58,000 people were admitted to hospital in England with a myocardial infarction.

The common mechanism to all ACS is rupture or erosion of atherosclerotic plaque within a coronary artery with resultant ischaemia and eventually infarction due to platelet-rich clot formation and vasoconstriction. While ACS is often a complication of progressive angina, it can also be seen in previously asymptomatic patients due to rupture of a soft and vulnerable plaque that was previously non-occlusive.

The distinction between different variants of acute coronary syndrome is made from the clinical history, electrocardiographic features, and biomarkers of cardiac injury (most commonly troponin) (Table 1.1).

HISTORY AND COMMUNICATION

Obtaining a complete clinical history is of paramount importance as it helps distinguish between types of ACS and therefore helps with risk stratification and ongoing management. Bear in mind, however, that patients may present acutely unwell or in cardiogenic shock and some details may need to be established once immediate management has been instituted and the patient is stabilised.

- Characteristics of chest pain – often central, tight or crushing in nature, radiating to neck, jaw or arms, worse on exertion, may be improved or even resolve at rest. Time of onset is often acute but in unstable angina can be gradual and progressive. If chest pain has been ongoing for several days, it is important to clarify the exact timing of the maximal severity of pain. Clarify response to nitrates, aspirin or morphine. Be aware that women and patients with diabetes may not present with typical features.
- Associated features – shortness of breath, sweating, nausea, dizziness, syncope, palpitations, vomiting, 'feeling of impending doom'.
- Risk factors – history of pre-existing angina, myocardial infarction, previous percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG), hypertension, hypercholesterolaemia, diabetes, peripheral vascular disease, cerebrovascular disease (stroke/TIA), smoking history, family history of premature coronary artery disease, lifestyle factors (carbohydrate- / saturated fat-rich diet, lack of exercise, excessive alcohol intake, obesity).

	Unstable angina	Non-ST-elevation MI	ST-elevation MI
Clinical history	• Progressively worsening chest pain over a short period of time (days–weeks). This is otherwise known as 'crescendo angina' • Reduced exercise tolerance or chest pain at rest • Reduced response to nitrates or rest	• Acute onset of chest pain at rest or on exertion • Possibly, but not necessarily, if background of previous angina • Limited response to nitrates or rest	• Acute onset of chest pain at rest or on exertion • Possibly, but not necessarily, if background of previous angina • Limited or no response to nitrates or rest • Can present with syncope or cardiac arrest secondary to ventricular arrhythmias
ECG	• Can be normal • Transient or persistent T wave inversion or ST depression • Transient ST elevation	• Can be normal • Transient or persistent T wave inversion or ST depression • Transient ST elevation	• Persistent ST elevation • Can have reciprocal ST depression in electrically opposite leads
Cardiac biomarkers	• Not elevated	• Elevated	• Significantly elevated

Table 1.1 Classification of acute coronary syndromes

CLINICAL EXAMINATION

The presentation of patients with ACS can vary significantly depending on the clinical variant. It is important to assess patients thoroughly, but in a timely manner to be able to institute suitable management strategies promptly. In an emergency, an ABCDE assessment is the most appropriate.

A – Airway
- Assess airway patency and need for airway adjuncts.

B – Breathing
- Assess respiratory rate and oxygen saturations. Is there evidence of respiratory distress? Supplemental oxygen targeted to patient's saturations may be required.
- Auscultate lungs – is there evidence of bilateral crepitations suggestive of pulmonary oedema? Intravenous furosemide or nitrate infusion may be helpful.

C – Circulation
- Palpate peripheral and central pulse, paying attention to rate, rhythm and volume.
- Assess blood pressure and heart rate. Are there features of cardiogenic shock (hypotension, tachycardia, patient feels cold and clammy to touch, reduced capillary refill time)?
- Auscultate the heart – are there any murmurs (e.g. pansystolic murmur in acute mitral regurgitation or ventricular septal defect)?
- In acutely unwell patients, continuous cardiac monitoring is advised.

D – Disability
- Assess patient's alertness using Glasgow Coma Scale.
- Assess blood glucose levels.

E – Environment
- Are there any scars suggestive of previous cardiac surgery?

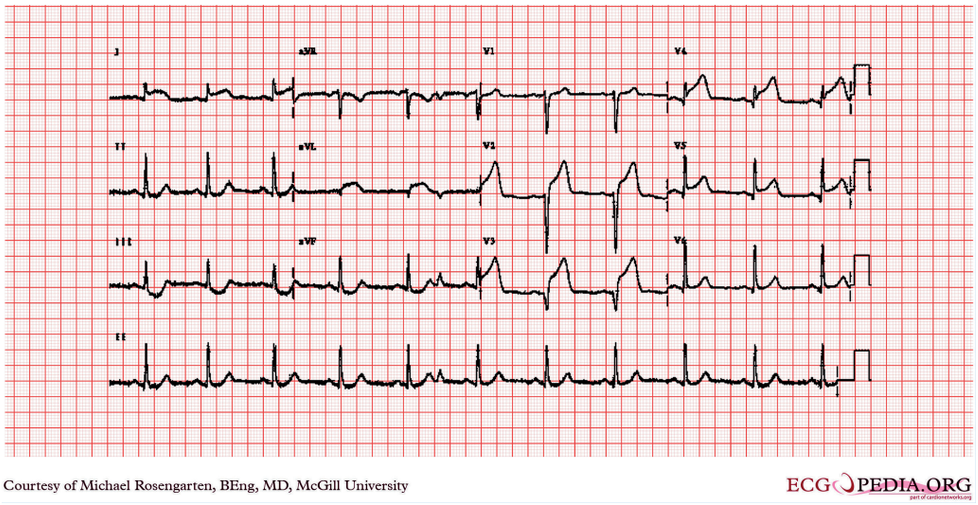

Figure 1.1 Acute anterior ST-elevation myocardial infarction – ST elevation in the anterior leads V1–V6, I and aVL reciprocal ST depression in the inferior leads. *Source:* E304 (CardioNetworks ECGpedia) © Michael Rosengarten BEng, MD.McGill (https://upload.wikimedia.org/wikipedia/commons/2/2e/E304_%28CardioNetworks_ECGpedia%29.jpg). CC BY-SA 3.0 DEED.

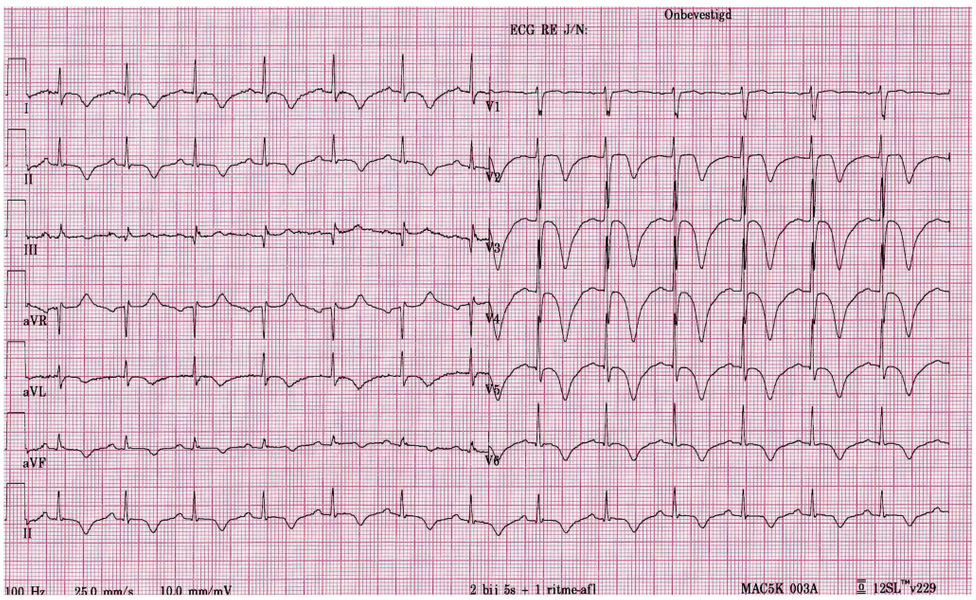

Figure 1.2 Anterior non-ST-elevation myocardial infarction – deep T wave inversion in V2–V6 and I, II, aVL and aVF. This is a high-risk ECG suggestive of a critical lesion in the LAD territory. *Source:* DVA2013 (CardioNetworks ECGpedia) © W.G. de Voogt, MD, PhD, SLAZ, The Netherlands (https://upload.wikimedia.org/wikipedia/commons/7/70/DVA2013_%28CardioNetworks_ECGpedia%29.jpg). CC BY-SA 3.0 DEED.

DIFFERENTIAL DIAGNOSIS

- Pulmonary embolism – thromboembolism risk factors (history of cancer, previous PE/DVT, pregnancy, exogenous oestrogen use, immobility, recent abdominal/lower limb surgery), pleuritic chest pain worse on inspiration, haemoptysis, syncope
- Aortic dissection – sudden, tearing chest pain radiating through to the back, history of hypertension, family history of sudden death, history of connective tissue disease or Marfan syndrome, known aortic dilatation

- Pneumothorax – sudden-onset unilateral pleuritic chest pain, history of smoking, history of previous recurrent episodes
- Pericarditis – central chest pain alleviated by sitting forward, worse on inspiration, associated with systemic symptoms (fever, fatigue), history of recent viral illness

DIAGNOSTIC TESTS/INVESTIGATIONS

ECG: baseline and repeat if patient is complaining of further chest discomfort

Blood tests:

Baseline troponin and further repeat troponin at either 3 or 6 hours depending on type of assay used in the department

Full blood count – to exclude anaemia contributing to presentation

Renal function tests – to guide decision making regarding use of IV contrast in coronary angiography

Lipid profile – to assess for hyperlipidaemia or familial conditions. Be aware that in the acute phase cholesterol may be falsely low

HbA1c – to rule out diabetes or assess diabetic control

Chest Radiograph: to rule out alternative diagnoses and assess pulmonary oedema

Echocardiogram: to assess ventricular systolic function following myocardial infarction, and valvular function

Continuous Cardiac Monitoring: to capture and respond to post-infarct arrhythmias promptly

MANAGEMENT

Management of acute coronary syndromes depends on the clinical variant and risk stratification. ST-elevation myocardial infarction requires immediate revascularisation, usually by percutaneous coronary intervention (primary PCI). For unstable angina and NSTEMI, there are several risk assessment scores in use. One example is the TIMI score (Table 1.2), which can predict the subsequent risk of STEMI and death. Patients at high or intermediate risk are advised to have early coronary angiography with a view to revascularisation to reduce mortality risk.

Risk factor	Score
Age > 65	1
More than 3 coronary artery disease risk factors (hypertension, hyperlipidaemia, diabetes, family history, smoking)	1
Known coronary artery disease (stenosis > 50% on previous angiography)	1
Aspirin use in the last 7 days	1
At least 2 episodes of rest pain in the last 24 hours	1
ST deviation on admission ECG	1
Elevated cardiac injury markers	1

Low risk: 0–2; intermediate risk: 3–4; high risk > 4.

Table 1.2 TIMI risk score

The management of acute coronary syndromes is outlined below:

1. Promptly load with dual anti-platelet therapy (DAPT) (usually consisting of 300 mg of aspirin and 600 mg of clopidogrel, or 180 mg ticagrelor, or 60 mg prasugrel).
2. Analgesia – titrate morphine to response, starting at 2.5 mg IV.
3. Nitrates – GTN spray sublingually or a GTN infusion if ongoing pain.
4. Oxygen titrated to patient's target O_2 saturations.
5. If ECG is suggestive of ST-elevation myocardial infarction, the patient should be transferred to a primary PCI centre immediately for early revascularisation.
6. If ECG and cardiac markers are suggestive of Unstable Angina / NSTEMI, commence low molecular weight heparin (e.g. fondaparinux 2.5 mg SC) and refer the patient for a cardiology opinion. A TIMI risk score can be helpful in determining ongoing pathway (inpatient versus outpatient).
7. Secondary prevention therapies should be commenced. These include:
   a. High intensity statin therapy, e.g. Atorvastatin 40 mg ON (for plaque stabilisation)
   b. ACE inhibitor, e.g. ramipril 2.5 mg ON (supports LV remodelling and improves long-term outcomes)
   c. Beta-blocker, e.g. bisoprolol 2.5 mg OD (antianginal; reduces risk of post-infarct arrhythmias; improves long-term outcomes)
   d. Ongoing DAPT – aspirin 75 mg OD and second anti-platelet therapy dependent on cardiologist choice (either clopidogrel 75 mg OD, ticagrelor 90 mg BD or prasugrel 10 mg OD) to prevent further events or stent thrombosis
   e. All of the above therapies are continued indefinitely, with the exception of the second anti-platelet drug which is usually discontinued 12–18 months after the event
   f. In addition, a proton pump inhibitor may be advised to reduce GI bleeding risk due to DAPT
8. Cardiac rehabilitation referral – advice regarding smoking cessation, lifestyle factors, exercise, return to work, driving.

COMPLICATIONS

Acute coronary syndrome complications relate to the degree and area of myocardial infarction:

- Heart failure secondary to ischaemic cardiomyopathy
- Ventricular arrhythmias (early, secondary to reperfusion, or late, relating to heart failure and myocardial fibrosis)
- Atrial arrhythmias, most commonly transient atrial fibrillation
- Conduction deficits (e.g. complete heart block)
- LV thrombus formation, in the case of extensive myocardial infarction
- Post-infarct ventricular septal defect
- Acute mitral regurgitation, secondary to infarction of papillary muscles / ruptured chordae
- Pericarditis, possibly complicated by pericardial effusion (Dressler's syndrome)
- Cardiogenic shock
- Death

2 Aneurysms, Ischaemic Limb and Occlusions

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AREAS OF CLINICAL PRACTICE

Conditions in: Cardiovascular, and Clinical imaging.

INTRODUCTION

Popliteal aneurysms are the most common peripheral artery aneurysm and these can present electively or as an emergency. Acute thrombosis of these aneurysms can present with an acutely ischaemic leg or with trashing of distal tissues.

EPIDEMIOLOGY AND CLINICAL PRESENTATIONS

Popliteal aneurysms are the most common peripheral artery aneurysm, occurring in 1% of men aged 65–80 years but much less frequent in women. They are more common in patients who have aneurysmal disease elsewhere (40% of patients with popliteal aneurysms will have abdominal aortic aneurysms) and other risk factors include smoking, hypertension, being male, advancing age and family history. Although diabetes increases the risk of aneurysmal disease, it is also associated with slower growth rates.

The clinical presentation of these aneurysms ranges from incidental discovery to acute limb ischaemia; 30–40% of patients are asymptomatic at the time of first discovery of the popliteal aneurysm, which can be either as an incidental radiological finding or with clinical detection during knee examinations.

Acute or chronic limb ischaemia with pain or limb swelling are all possible symptomatic presentations of popliteal aneurysms. If the aneurysm is more than 2 cm it is possible for the patient to experience a fullness sensation behind the knee due to the aneurysm's mass effect. This can sometimes progress to compression on the sciatic, tibial or peroneal nerves, which causes motor or sensory deficits. If the aneurysm is compressing the popliteal vein, then deep venous thrombosis and subsequent leg swelling can occur and could be the first presenting sign of a previously undiagnosed popliteal aneurysm.

Rupture occurs in less than 5% of patients with popliteal aneurysms, and the haemorrhage is usually confined to the popliteal fossa. Acute presentations of popliteal aneurysms are much more likely due to acute thrombosis (causing acute limb ischaemia) or distal trashing (distal embolisation of thrombus within the aneurysm).

Progressive luminal narrowing of the popliteal artery can cause vascular claudication symptoms (see 'Peripheral Vascular Disease') which may improve as collateral circulation develops. Repeated episodes of distal embolisation from the aneurysm can impair distal run-off as well as presenting with trashing of distal tissues. Eventual thrombosis of the artery will likely be associated with a deterioration of the claudication symptoms. For more detail of Arterial Thrombosis, see separate topic. Abrupt thrombosis of the aneurysm will present as acute limb ischaemia, which is the presentation in two-thirds of thrombosed popliteal aneurysms.

COMPLICATIONS / RISK FACTORS

Complications of popliteal aneurysms are claudication, chronic ischaemia, acute ischaemia and rupture.

HISTORY

Questions	Rationale
History of pain, numbness, cold sensation, reduced movement or appearance of pallor?	To determine whether presentation is acute ischaemia
Vascular claudication history including calf cramping on walking, exercise tolerance or pain at night and history of tissue loss?	For chronic ischaemia presentations
Sudden increase in swelling?	Rupture presents in less than 5% of popliteal aneurysms and the haemorrhage is often contained to the popliteal fossa

CLINICAL EXAMINATION

Perform a routine vascular examination.

Popliteal aneurysms can sometimes be felt as a mass in the popliteal fossa which can be distinguished from other differentials such as Baker's cyst, lymphadenopathy or malignancy by pulsatile expansion. More distal examination should include dorsalis pedis and posterior tibial pulses (with handheld doppler if not palpable) and examination of the tissues for evidence of trashing. Formal ankle brachial pressure index (ABPI) is not required for a diagnosis of popliteal aneurysms but can be used to document severity of ischaemia.

The presence of the 6 'P's' of acute ischaemia (Pain, Pallor, Paralysis, Paraesthesia, Pulselessness and Perishing cold) must necessitate emergency clinical review and escalation.

DIAGNOSTIC TESTS/INVESTIGATIONS

The first-line investigation for popliteal aneurysms in the elective setting is duplex ultrasonography which can confirm the size and patency of an aneurysm. For operative planning a CT angiography is useful for more specific visualisation of the aneurysm. Endovascular approaches to repair can be considered but are restricted due to the need for specific anatomy for stent placement and there is a risk that they can occlude if placed across the knee crease. CT angiography is the most appropriate first-line investigation in the emergency environment for any acutely compromised limb.

MANAGEMENT PLAN

A systematic approach should be adopted in order to stabilise the patient. Analgesia will be required as well as gaining IV access for CT angiography.

Acute management could include IV heparin and then progression to intervention to revascularise the lower limb.

In the semi-elective setting there is a role for endovascular approach to popliteal aneurysms. Surgical approaches can consist of an exclusion bypass graft or an inline repair with a tube graft.

These patients will need discussion in the vascular MDT for consideration of optimal treatment.

3 Aortic Aneurysm

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AREAS OF CLINICAL PRACTICE

A condition in: Acute and emergency, Cardiovascular, and Surgery.

INTRODUCTION

An aortic aneurysm is a dilatation of the arterial wall which can lead to vessel weakness, turbulent blood flow and rupture. The morbidity and mortality of having an aneurysm that ruptures is very high, with an approximately 20% overall survival. Therefore, the aim of elective treatment is to prevent rupture by active surveillance and intervention at an appropriate point, where the procedure will benefit the patient, outweighing the risks of intervening.

Top Tips

- If you think someone has a ruptured aneurysm, speak immediately with your senior. While waiting for assistance, obtain venous access, send a cross-match and call the blood bank.
- Remember to optimise cardiovascular comorbidities and to emphasise lifestyle changes to patients with an aneurysm.
- Always palpate for an aneurysm during an abdominal examination.

PATHOLOGY AND EPIDEMIOLOGY

Anything that can weaken an arterial wall can cause an aneurysm – causes include hypertension, abnormal blood flow and atherosclerosis. There are additional factors that also contribute to this pathology, such as structural changes within the arterial wall. These include loss of elastin, collagen or smooth muscle.

Aneurysms can occur in any artery in the body. The most common location for an aneurysm is the aorta, of which the infra-renal segment is most often affected. See Figure 3.1.

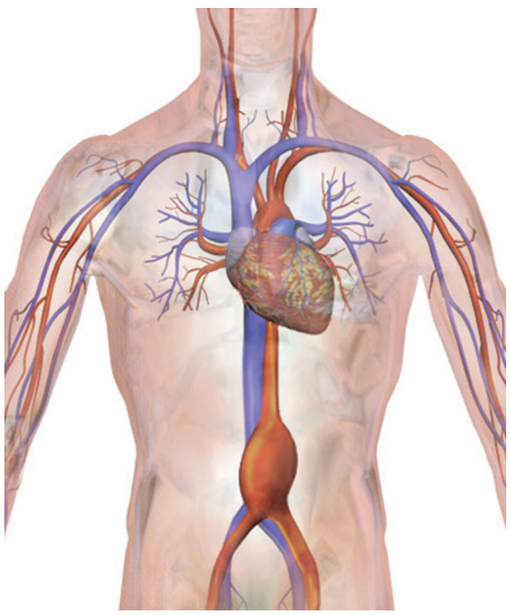

Figure 3.1 Abdominal aortic aneurysm. *Source:* Blausen.com staff, 'Medical gallery of Blausen Medical 2014', *WikiJournal of Medicine* 2014, 1 (2): doi.org/10.15347/wjm.2014.010. ISSN 2002-4436.

Aneurysms are uncommon, with small aneurysms found in 4–8% of screening studies. They are rarely seen under the age of 55 and affect males significantly more than females.

RISK FACTORS

These can be divided into modifiable and non-modifiable risk factors, as shown in Table 3.1.

Modifiable	Non-modifiable
Smoking	Male sex
Hypertension	Family history
Sedentary lifestyle	Age
The same risk factors as for atherosclerosis	Connective tissue disorders (Marfan, Ehlers-Danlos)
	Infective pathology (mycotic aneurysms)

Table 3.1 Risk factors for abdominal aortic aneurysm

HISTORY AND COMMUNICATION

Most abdominal aortic aneurysms (AAA) are asymptomatic and are identified as a pulsatile mass or incidentally on imaging. Infrequently, patients can feel abdominal, back or loin tenderness that arises from their aneurysm. Diagnosis is late and for this reason there is a UK national screening programme for men over the age of 65. Therefore, it is important to ask questions relating to the modifiable risk factors of cardiovascular disease:

- Do they smoke?
- Do they have high blood pressure?
- Do they have high cholesterol?
- Is their diet healthy?
- Do they exercise?

It is common for patients to find it daunting knowing that they have a disease process that can rupture and kill them at any time. Communication and a thorough clarification are therefore paramount. Patients with aneurysms are counselled by specialists to ensure understanding of pathology and to offer explanation of why the aneurysm is not fixed immediately after diagnosis, with emphasis on risk versus benefit of intervention.

Depending on the licence the patient holds, the DVLA must be updated by them when their AAA reaches 6 cm, as driving is banned with an AAA over 6.5 cm.

CLINICAL EXAMINATION

Alongside a detailed cardiovascular examination, you must examine for AAA. This is defined as a pulsatile and expansile mass. Place the patient supine and ensure the abdominal muscles are relaxed. The aorta is located above the umbilicus and slightly to the left. Palpate the aorta by placing both hands down into the abdomen, either side of the vessel. Try to assess the width of the aorta as you do this – intensity of the pulsation is not related to presence or absence of an aneurysm, nor is the examination associated with increasing the chance of rupture.

It is important to examine the peripheral vascular system as there is an association with peripheral aneurysms when an AAA is present.

Top Tips

If the patient presents with a ruptured AAA, the clinical signs and symptoms may include:

- Sudden and severe abdominal, flank or back pain
- Sudden loss of consciousness
- Hypotension, sweating, pallor, shock (etc.) – all due to rapid blood loss
- A pulsatile mass is often harder to examine as the abdomen is rigid

DIAGNOSTIC TESTS/INVESTIGATIONS

Ultrasound: duplex ultrasound scan (USS) is a useful non-invasive test for AAA surveillance. There is a screening programme (Table 3.2) for men over the age of 65 which assesses presence and size of any AAA.

Size	Screening frequency	Further action
< 3 cm (normal aorta)	No further screening	None required
3 – 4.4 cm (small)	Scan annually	Refer to vascular services within 12 weeks – to discuss options with the patient, explain results, discuss future management
4.5 – 5.4 cm (medium)	Scan every 3 months	
> 5.5 cm (large)	Urgent referral to vascular services, to be seen within 2 weeks	

Table 3.2 Summary of screening programme within the UK

When ruptured, it is vital to confirm the diagnosis in order to guide management plan. This is done with a CT angiogram (duplex USS is not specific enough for ruptured AAA). This uses contrast to show the anatomy of the aorta, the location and extent of the rupture and the shape of the vessel. In the UK, it is unusual for a patient to be so critically unwell that there is no time for a diagnostic scan.

MANAGEMENT PLAN

In the elective setting, alongside the screening programme to monitor aortic size changes, there is focus on reducing the risk of rupture. This is done by treating the risk factors of atherosclerotic disease and hypertension:

- Smoking cessation advice and treatment
- Optimisation of hypertension
  - Lifestyle changes – increasing exercise, decreasing alcohol intake
  - Medical management – antihypertensives

- Optimisation of atherosclerosis
  - High-dose statins (there is some evidence that statins may slow the growth of AAA)
  - Anti-platelets (clopidogrel or aspirin)

Once aneurysms reach a certain threshold, their risk of rupture is greater to the patient than the risk of intervention. Consideration for repair is made with the following criteria and these patients should be referred through to a vascular consultant:

- > 5.5 cm Anterior–Posterior diameter in asymptomatic patients
- > 4 cm Anterior–Posterior diameter and growing more than 1 cm per year
- Any symptomatic patients

Aneurysms can be repaired with surgery or endovascularly (radiologically). There are advantages and disadvantages to both which can be seen in Table 3.3.

Endovascular aneurysm repair	Surgery
Reduced early morbidity and mortality compared to open surgery	Mortality is greater than endovascularly
May require reintervention	Unlikely to require reintervention (i.e. a 'one-off' procedure)
Requires lifelong surveillance	Increased risk of perioperative complications (e.g. MI, respiratory)
No long-term survival benefits to surgery	More expensive in the short term
More expensive over longer time frame	

Table 3.3 Advantages and disadvantages of the two methods of repair

The choice between these options is made at a multidisciplinary meeting with consideration to patient comorbidities, preference, and suitability of the aneurysm. In both instances, a stent is placed within the aneurysm. The stent allows the blood to pass through it, rather than passing through the dilated and weakened aneurysm wall, which restores anatomy and vessel pressures.

When ruptured, management differs and actions are performed in a time-critical manner:

- Resuscitate
  - ABCDE management
  - Consider activating the major haemorrhage protocol – ensure urgent cross-match of blood as multiple blood products will likely be required; plan ahead and plan quickly
  - Seek immediate senior support
  - Permissive hypotension is allowing the body to maintain a lower than desired blood pressure. Trying to reach normotension can worsen the rupture
  - Opiate-based analgesia
  - Early discussion with a vascular consultant
- Diagnose – CT angiogram to confirm
- Decision – made by vascular team
  - Is the patient stable enough for a transfer to a vascular centre?
  - Is this patient suitable for intervention?
  - If not, mortality from a free rupture is inevitable, palliative care is vital

COMPLICATIONS

Complications of a ruptured AAA include ischaemia of all parts of the body due to the rapid exsanguination of blood (e.g. myocardial infarction, spinal ischaemia leading to paralysis, renal ischaemia leading to kidney failure, bowel ischaemia leading to necrosis and then perforation, lower limb ischaemia, etc.).

If a patient undergoes a successful emergency repair, there remains a long road to recovery and some patients will not reach the same level of function and independence they had prior.

OTHER TYPES OF ANEURYSMS

- This topic has focused on AAA as they are the most common type of aortic aneurysm (> 90%).
- Thoraco-abdominal aortic aneurysms are managed differently and have a much higher rate of mortality when intervention is planned electively.
- Beware of the hypotensive patient with acute abdominal pain who is not known to have an AAA – ruptured iliac aneurysms can be misdiagnosed as an acute abdomen.

4 Aortic Dissection

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AREAS OF CLINICAL PRACTICE

A condition in: Cardiovascular, and Surgery.

INTRODUCTION

An aortic dissection is a potentially life- or limb-threatening condition where a tear in the innermost layer (tunica intima) of the aorta causes blood to accumulate between the tunica intima and the middle (tunica media) layers of the aorta. This accumulation of blood in the wall of the aorta is known as the false lumen. Along with intra-mural haematoma (IMH) and penetrating aortic ulcer (PAU), acute aortic dissection forms the spectrum known as acute aortic syndromes.

Aortic dissections can be classified by their time of onset or location in relation to the aortic arch. Onset less than 14 days ago is termed 'acute', 14–90 days is termed subacute, and beyond 90 days is termed chronic. The commonest classification criterion used for aortic dissection is the Stanford (others include the DeBakey) and describes the dissection in relation to the origin (Figure 4.1).

Stanford Classification

Type A	Dissection arises in the ascending aorta
Type B	Dissection arises in the descending aorta, distal to the left subclavian artery

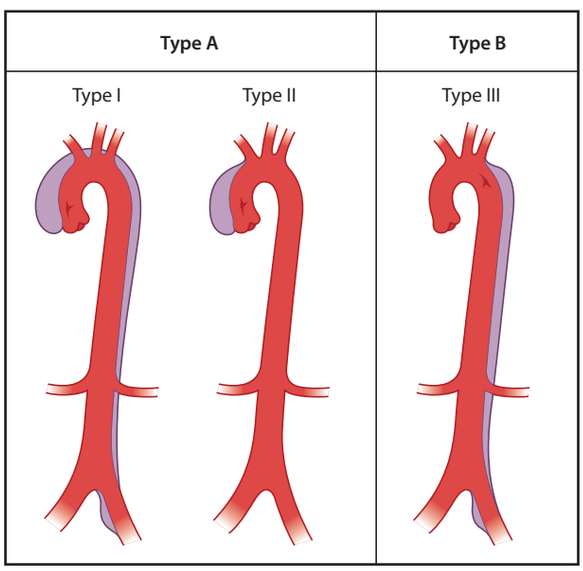

Figure 4.1 Diagram demonstrating the anatomical classification of aortic dissection.

EPIDEMIOLOGY AND RISK FACTORS

Aortic dissection has an incidence of about 6 per 100,000 persons per year. It is associated with several risk factors, with hypertension being the most important (Table 4.1). Individuals with connective tissue disorder or other genetic predispositions to aortic dissection are more likely to present at younger age and without hypertension.

Hypertension
Aortic aneurysm
Risk factors for atherosclerosis (smoking, diabetes, hyperlipidaemia, increasing age, male gender)
Cocaine use
Connective tissue disorder (e.g. Marfan syndrome)
Polycystic kidney disease
Bicuspid aortic valve
Turner's syndrome
Aortic surgery or manipulation through procedures (e.g. CABG or PCI)
Pregnancy
History of aortitis

Table 4.1 Risk factors associated with aortic dissection

HISTORY AND COMMUNICATION

Aortic dissections are often challenging to diagnose, and one must have a high index of suspicion. The most common presenting symptom in aortic dissection is chest pain. It is typically sudden in onset, severe, sharp or tearing in nature. Type A dissections present with chest pain, but Type B dissections may present with interscapular or back pain instead. Pain may also be described to be migratory, reflecting extension of the false lumen. Patients can also present with signs of distal ischaemia (i.e. abdominal pain, acute limb ischaemia). Patients may describe a precipitant to an acute rise in blood pressure – e.g. cocaine use, an emotional stressor or intense physical activity such as weightlifting.

Syncope is an important symptom that can occur with acute aortic dissection and may be sign of more serious sequelae, such as tamponade, rupture or limitation of cerebrovascular blood flow.

Aside from pain, aortic dissection can cause symptoms of malperfusion when the false lumen obstructs blood flow in branches of the aorta. This could include coronary arteries, cerebral or spinal vasculature, mesenteric arteries, renal arteries or arteries supplying the limbs. Symptoms here will be wide ranging and correspond to the occluded vessel. For example, occlusion of cerebral vessels can cause neurological symptoms in keeping with stroke, or occlusion of coronary ostia (typically the right) can cause the appearance of concomitant STEMI.

CLINICAL EXAMINATION

	Clinical features
General inspection	• Pallor / clammy/cool extremities / confusion – these are features of shock. Acute aortic rupture, decompensated acute aortic regurgitation and tamponade can cause a patient with aortic dissection to present in shock • Syndromic features – can give clues to underlying aetiology (e.g. height or pes excavatum in Marfan syndrome or evidence of webbed neck in Turner's syndrome)
Hands	• Tar staining – smoking is a risk factor for aortic dissection
Peripheral pulses	• Radio-radial delay – a loss of synchronicity between each arm may be associated with aortic dissection • Lower limb pulses – aortic dissection can cause malperfusion in lower limbs • Tachycardia may be present – this could be part of a sympathetic response to pain or an attempt by the heart to compensate
Blood pressure	• Hypertension is common • Hypotension can be seen (more commonly with Type A dissection) and may represent shock secondary to any of aortic rupture, tamponade, acute aortic regurgitation • Wide pulse pressure: > 100 mmHg difference between systolic and diastolic blood pressure (aortic regurgitation) • > 20 mmHg difference in blood pressure between left and right arm may suggest aortic dissection
Neck	• Distended neck veins – could be a sign of tamponade
Eyes	• Pupil dilation – can be secondary to cocaine use
Auscultation	• Aortic regurgitation – early diastolic murmur (usually quiet and difficult to hear). Ask the patient to sit forward and hold their breath in expiration, and listen at the left lower parasternal edge • Muffled heart sounds (one of the signs of pericardial effusion/ tamponade)

Table 4.2 Important clinical features to look for on examination

DIAGNOSTIC TESTS/INVESTIGATIONS

Electrocardiogram: in aortic dissection, an ECG may take on a range of appearances. It may appear normal, have evidence of ischaemic changes / STEMI (due to occlusion of the coronary ostia by the false lumen) or may have small complexes representing large pericardial effusions. There may be evidence of chronic hypertensive changes, such as left ventricular hypertrophy and lateral T wave inversion. Tachycardia is a common finding.

Blood Tests: a routine panel including a full blood count, urea and electrolytes, CRP, troponin, D-dimer. It is useful to do cross-matching if planned for theatre. An amylase can be useful in ruling out pancreatitis (may present similarly to Type B dissection). A D-dimer < 0.5 μg/ml makes the diagnosis of aortic dissection very unlikely.

Chest X-ray (CXR): this helps to assess some of the key differential diagnosis such as pneumothorax or perforated viscus. Subtle signs of aortic dissection may also be present, such as a widened mediastinum, or new pleural effusion. A normal CXR does not rule out aortic dissection.

CT Angiogram Aorta: this is the gold standard investigation for aortic dissection. It should be requested if there is a high index of suspicion from clinical symptoms or any of the above tests.

Echocardiography: transoesophageal echocardiography is useful in unstable patients to identity dissections but may not always be readily available. Transthoracic echo can quickly identify some of the key complications of dissection in an unstable patient (tamponade, aortic regurgitation, inferior ischaemia).

MANAGEMENT PLAN

Aortic dissection carries a high mortality rate, particularly with Type A dissection. Patients with an aortic dissection have the potential to be very unwell, and you should assess them in A → E approach. The immediate priority is resuscitation (if required) and stabilising the patient for imaging. Once the diagnosis is established, management depends on the classification.

Type A

Immediate referral to regional cardiothoracic centre for emergency surgery to repair damaged aorta +/– aortic valve. Blood pressure monitoring and analgesia as for Type B in the interim, but these should not delay the transfer to theatre.

Type B

If evidence of malperfusion, refer to vascular surgery. Otherwise, Type B dissection can be managed medically in a high dependency unit / coronary care unit. It involves strict blood pressure control (aiming for systolic BP < 120) potentially utilising IV labetalol (verapamil is an alternative if beta-blocker intolerant) and invasive blood pressure monitoring, analgesia and urine output monitoring. Other acute aortic syndromes (APU and IMH) are often managed the same way.

Follow-up imaging is required acutely (if pain symptoms persist) or in the medium to long term, as there is a risk of aneurysm formation due to weakening of the vessel.