Abdominal Aortic Aneurysm (AAA) — Risk Factors and Prognosis

Aortic aneurysm is a common and serious condition that can be fatal upon rupture. The aneurysm can be detected months or years before a rupture occurs but commonly goes undetected, due to its asymptomatic nature in its early stages. Ideally, early detection prevents complications and reduces the mortality rate of this condition. Knowledge of Aortic aneurysm is essential for physicians, especially as a differential diagnosis for abdominal, back, chest and leg pain. It needs to be excluded early even if there is a small possibility of its existence due to its high mortality rate. Unpredictable sudden rupture of aortic aneurysms can quickly lead to hypovolemic shock or death.

Definition and Background of Aortic Aneurysm

An aortic aneurysm is a permanent and irreversible widening of the aorta to more than 1.5 times its normal diameter. It commonly goes undiagnosed until it ruptures,
as it is otherwise asymptomatic. Aneurysms are commonly located in the abdomen but can occur at any location along the aorta, including the thoracic aorta. They are associated with a high mortality if they progress to rupture. Patients at the greatest risk are those who are over the age of 65 with multiple cardiovascular and genetic risk factors or have existing peripheral vascular diseases.

Epidemiology of Aortic Aneurysm

Asymptomatic aortic aneurysms occur at a rate of between 5—10 % and are more common in males than in females. Most cases occur in men who are over the age of 50 and in women who are over the age of 60. Within this age bracket, there is a sharp increase in the number of cases. Abdominal aortic aneurysms are more common in white Caucasians than in African American, Asian, and Hispanic ethnicities. Ruptured abdominal aortic aneurysm causes 15,000 deaths a year in the United States alone, making it the 13th most common cause of death. In 2013, 152,000 deaths were caused by aortic aneurysms, an increase from 100,000 in 1990.
Etiology of Aortic Aneurysm

Aortic aneurysms are caused by **degradation of the aortic walls**, which causes them to become weak. This can occur for many reasons:

- **Atherosclerosis**: aortic wall atheromatous disease
- Genetic disease; affects the structure and function of connective tissues / proteins (e.g. collagen and elastin) in the walls of the aorta – Marfan’s syndrome, Ehler-Danlos, Collagen vascular-diseases (more likely to cause **thoracic aortic aneurysms**)
- Infection/Mycotic aneurysm – local invasion of the intima and media by an infective pathogen (most commonly gram-positive organisms: *Escherichia coli*, *Salmonella*) leads to abscess formation and dilatation
- Trauma
- **Aortitis** (usually syphilitic — TAA > AAA)
- **Hypertension**
- Alcohol

Risk factors

<table>
<thead>
<tr>
<th>Modifiable Risk Factors</th>
<th>Unmodifiable Risk Factors</th>
</tr>
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<tbody>
<tr>
<td>Atherosclerotic disease (can be genetically more susceptible – unmodifiable)</td>
<td>Age: &gt; 55</td>
</tr>
<tr>
<td>Cardiovascular and peripheral vascular disease risk factors</td>
<td>Sex: Male</td>
</tr>
<tr>
<td>Smoking</td>
<td>Genetic Conditions (e.g. Marfan’s, Ehlers-Danlos)</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>Family History</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Peripheral vascular disease</td>
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Classification of Aortic Aneurysm

Aneurysms are classified as follows:

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>True aneurysm</strong></td>
<td>Involves all three layers of the wall of the vessel — intima, media, and adventitia.</td>
<td>Atherosclerosis, infection (syphilitic), ventricular and congenital</td>
</tr>
<tr>
<td><strong>False aneurysm (pseudoaneurysm)</strong></td>
<td>Blood that has leaked out of artery/vein but is held around the vessel by the surrounding tissue – this cavity can clot or rupture eventually</td>
<td>Trauma, surgery (percutaneous), artery injection</td>
</tr>
</tbody>
</table>

Aortic aneurysm classification occurs based on the location of an aneurysm:

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aortic root aneurysm</strong></td>
<td>Rare</td>
</tr>
<tr>
<td><strong>Thoracic aortic aneurysms (TAA)</strong></td>
<td>Ascending, aortic arch and descending</td>
</tr>
<tr>
<td><strong>Abdominal aortic aneurysms (AAA/Triple-A)</strong></td>
<td>Most common</td>
</tr>
</tbody>
</table>

Morphology

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
<th>Occurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fusiform</strong></td>
<td>spindle-shaped, affects the entire circumference of the vessel. Variable length and diameter</td>
<td>Most common – aorta and sometimes iliac arteries are affected</td>
</tr>
<tr>
<td><strong>Saccular</strong></td>
<td>spherical-shaped, only affecting part of the vessel</td>
<td>Uncommon – Usually filled with a thrombus</td>
</tr>
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</table>
Pathophysiology of Aortic Aneurysm

Aortic aneurysms are caused by the degradation of the aortic wall which results in a permanent and irreversible dilatation. The degradation consists of loss of the elastic lamellae and smooth muscle cells, increased proteolysis, and leucocytic infiltration. The dilatation becomes defined as an aneurysm once the diameter has reached 1.5 times the normal aorta’s diameter; abdominal → 3 cm/thoracic → 4.5 cm.

**Abdominal aortic aneurysm (AAA)**

This is the most common type of an aortic aneurysm. 90 % of cases occur below the L1-L2 vertebra; this correlates to the level of the renal arteries (infra renal). AAA’s are more common than TAA’s because the abdominal aortic walls contain less elastin. Elastin is a protein which allows for elasticity and enables a vessel to expand, contract and then return to its original position. The thoracic aorta is usually under higher pressure as it is closer to the heart and therefore contains more of this load-bearing protein. Having fewer elastin subjects the abdominal aorta to more strain and if it becomes diseased, it takes less time to degrade all the elastin present in the AAA compared to that in the TAA.

The main mechanism behind AAA’s development is the destruction of the tunica media by proteolysis which can occur for a number of reasons; increased metalloproteinase activity can lead to increased destruction of the media. The media contains the muscular, structural and elastic tissue which gives the walls their strength. The abdominal aorta also has fewer vasa vasorum compared to the TAA; therefore, as the cells within the abdominal aortic wall rely on diffusion, this makes them more prone to becoming damaged especially when the vessel is atherosclerotic (increased diffusion distance).

**Thoracic aortic aneurysm (TAA)**

These are aneurysms that are located in the thoracic aorta, above the diaphragm. They can be very fatal if they go undetected, as rupture can easily lead to death due to their close proximity to the heart. They are rarer than AAAs. TAAs are more likely to be
syphilitic, genetic, congenital or due to trauma. Descending aortic aneurysms are primarily atherosclerosis-related, whereas those of the aortic arch are also generally due to atherosclerosis or dissection.

Hypertension

If the patient is hypertensive, this can lead to a rapid progression of the disease as the weakened walls of the aorta are under more strain.

Atherosclerosis

Atherosclerosis occurs when atherosclerotic plaque builds up within the aorta and reduces the diffusion of blood to the media of the aortic wall by increasing the diffusion distance. This causes atrophy of the media, as there is reduced blood supply. The decrease in size and therefore the strengthening of this layer allows for larger amounts of irreversible dilatation to occur; this is because the media is made up of smooth muscle cells and elastic tissue which usually absorbs the blood pressure generated by the heart.

Genetic

Marfan’s syndrome is a connective tissue disorder which involves the misfolding of fibrillin-1. Fibrillin-1 is a protein that forms elastic tissue and has roles in signaling. One such role includes binding to the TGF-beta; in the case of Marfan’s syndrome, mutated fibrillin-1 fails to do this and causes an accumulation of TGF-beta in various tissues including the aorta. This results in formation of weakened tissue that have an abnormal structure and function.

Ehlers-Danlos syndrome is where a genetic problem causes incorrect production and processing of collagen, an essential protein involved in the structuring of tissues. This can lead to weakened vessel walls that can quickly become aneurysmal.

Pathology of Aortic Aneurysm

There are changes within the aorta wall:

- Tunica media and intima: foam cells, cholesterol, ulceration, thrombosis, ruptures – consistent with atherosclerosis.
- Adventitia – inflammatory infiltrate.
- Loss of smooth muscles cells and the elastic lamellae.

There may be further evidence of atherosclerosis; abnormal connective tissue structure in genetic conditions, infection and/or inflammation.

Symptoms of Aortic Aneurysm

Usually asymptomatic if not ruptured

- Most aneurysms are asymptomatic until rupture occurs and are usually found on routine health checks or through radiological investigations incidentally.

Over time as an aneurysm enlarges, the patient can present with:

- flank/back pain
- abdominal pain
- pulsatile mass, usually in the abdomen, which increases in size over months and years.

An aneurysm can compress surrounding structures such as organs and nerves depending on its size and location.

**Vocal hoarseness**

- Arch of the aorta aneurysms can irritate the left recurrent *laryngeal nerve* (branch of vagus which goes underneath the arch of the aorta before turning back upwards to supply blood to the laryngeal muscles that help produce voice)

**Urinary symptoms**

- Ureteral compression

**Thromboses/emboli**

- As a result of pressure on surrounding vessels

**Emboli symptoms**

- The plaque that usually lines atherosclerotic aortic walls can break off and as a result block a downstream vessel.

**Symptoms of systemic disease(s)**

- Patients may have established disease(s) related to an aortic aneurysm and could, therefore, have symptoms of peripheral vascular disease, infection, trauma, Marfan’s syndrome or Ehler-Danlos syndrome.

**Signs**

**Inspection**: patient appears comfortable at rest. A visible abdominal pulsation may be seen.

**Auscultation**: bruits heard in the central abdominal region

**Palpation**: pulsatile and expansile mass in the abdominal region can be felt when the hands are placed on either side of the aorta. Move hands gradually outwards to feel the borders of the aorta to determine if it is expansile.

**Signs of peripheral vascular disease may be present:**

- arterial insufficiency in the lower limbs, e.g. arterial ulcers, weak pulses, pallor, cold, loss of hair, gangrene, loss of sensation – paraesthesia, paralysis
- Patient in *shock*: cold, clammy, pale, *tachycardia*, tachypnoea.
- Pulses- carotid, brachial and femoral may be abnormal.
- Anxious, sense of impending doom.
- Marfan’s syndrome signs – e.g. high arched palate
- *Ehlers-Danlos syndrome* signs
- Infection signs – e.g. fever
- Trauma signs – e.g. *knife wound*
Complications

**Rupture** is the major complication of aortic aneurysms and is especially dangerous when the diameter of an aneurysm is more than 5 cm. It is a severe life-threatening emergency and the patient usually presents in shock if they survived the journey to the emergency department:

**Patient presents with a classic triad:**

- Flank pain – severe tearing pain that usually radiates towards the back
- Hypotension (from blood loss)
- Pulsatile abdominal mass

**The patient can have:**

- **tachycardia**
- altered mental status
- flank bruising if there is retroperitoneal bleeding
- skin of blue complexion
- loss of consciousness
- Hypotension and shock (hypovolemic) – eventually death by **exsanguination** (blood loss)
- Permanent disability from a stroke (**CVA**)
- Global **ischemia** -- e.g. mesenteric, bowel, renal, spinal cord, visceral ischemia/infarction
- Compression – esophagus, left recurrent laryngeal nerve (hoarseness and vocal cord paralysis).

**Diagnosis of Aortic Aneurysm**

The diagnosis of AAA needs to be fast and accurate. The following procedures may be used:

- **Physical examination** – pulsatile and expansile abdominal mass with bruits on auscultation; failure to detect this is **not** enough to rule out AAA.
- **Anamnesis**: In stable patients, a detailed history should be taken to assess risk factors, risk of rupture, symptom duration and onset and any family history or genetic conditions.

**Imaging** is used to diagnose and reveal the extent of the disease:

**Imaging methods**

The following imaging methods are used to detect aortic aneurysms:

**Ultrasound**

- Gold standard diagnostic test for an aortic aneurysm
- Used to monitor aortic aneurysms over time if the patients does not require immediate surgery
- Quickest and easiest option
- Can be done at the bedside
- Reveals size and extent of disease
**CT > (MRI) Scan**

- Used after ultrasound diagnosis to give more insight on size and anatomy for monitoring purposes and to assist surgeons
- CT scan has 99% sensitivity
- CT scan uses dye-contract to measure blood flow through the aorta

**X-Ray Scan**

- Abdominal/Kidney Ureter and Bladder (may be discovered after KUBXR for urinary symptoms caused by ureteral compression)
- Can reveal calcification of the aorta
Aortic angiography

- Catheterisation of the aorta (via the femoral artery) and injection of contrast whilst x-rays of the aorta are taken. Useful for determining the luminal size and examining any aortic dissections that may be present.

**Blood tests** – FBC, U+Es, ESR/CRP, Clotting screen, lipid profile

**Blood pressure tests**

**Differential Diagnoses**

<table>
<thead>
<tr>
<th>Kidney</th>
<th>Pyelonephritis, Nephrolithiasis</th>
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<tbody>
<tr>
<td><strong>GI system</strong></td>
<td>Acute gastritis, perforated peptic ulcer, bleed, ischaemic bowel, large bowel obstruction, pancreatitis, gallstones, diverticulitis, IBS, IBD, appendicitis</td>
</tr>
<tr>
<td><strong>Heart</strong></td>
<td>Myocardial infarction, aortic dissection</td>
</tr>
<tr>
<td><strong>Gynaecological</strong></td>
<td>Cystitis, ruptured ovarian cyst, ovarian torsion</td>
</tr>
</tbody>
</table>

**Treatment of Aortic Aneurysm**

**AAA**

<table>
<thead>
<tr>
<th>Conservative</th>
<th>Surgical Repair</th>
<th>Medication</th>
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</thead>
<tbody>
<tr>
<td>• Surveillance usually by ultrasound to monitor progression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Reduce modifiable risk factors</td>
<td>• Open aneurysm repair (OR)</td>
<td></td>
</tr>
<tr>
<td>• Endovascular aneurysm repair (EVAR)</td>
<td>• To monitor and treat risk factors</td>
<td></td>
</tr>
<tr>
<td>• Immediate surgery for rupture</td>
<td>• Lipids (statins)</td>
<td></td>
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<tr>
<td></td>
<td>• Strict blood pressure measurement (antihypertensives)</td>
<td></td>
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**TAA**

TAA’s are defined as aneurysmal after getting to a diameter > 4.5 cm, however, the treatment size involves a TAA with a diameter > 6 cm. They can be repaired via endovascular or open surgery.
**Prognosis**

The larger an aneurysm is, the more likely it is to rupture. However, it is important to note that smaller diameter aneurysms also rupture. The following table shows results from a 2003 study by the Society for Vascular Surgery, it shows the relationship between diameters of AAA’s and the risk of rupture annually (Brewster DC, 2003).

<table>
<thead>
<tr>
<th>AAA Size (cm)</th>
<th>Annual Rupture Risk (%)</th>
</tr>
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<tbody>
<tr>
<td>3—3.9</td>
<td>0</td>
</tr>
<tr>
<td>4—4.9</td>
<td>0.5—5</td>
</tr>
<tr>
<td>5—5.9</td>
<td>3—15</td>
</tr>
<tr>
<td>6—6.9</td>
<td>10—20</td>
</tr>
<tr>
<td>7 &gt;</td>
<td>20—50</td>
</tr>
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</table>

Ruptured AAAs have a significantly worse prognosis in comparison to unruptured AAAs. The postoperative mortality for a ruptured AAA is still > 40 % (Brown et al, 2002), compared to 1—6 % if the AAA is repaired before it gets ruptured (Greenhalgh et al, 2004).

**Screening**

Screening programs exist worldwide to detect aortic aneurysms early. The main criteria usually involves men over the age of 65 who may be at higher risk, due to having risk factors such as a history of smoking. It is not certain whether screening for women is useful.

There is increased screening for those with a family history and/or genetic diseases with known links to aortic aneurysms. Early detection is key in order to reduce the high mortality rate of aortic aneurysms.

**Prevention**

The risk of aortic aneurysms can be reduced by close monitoring of cholesterol and blood pressure. Reducing modifiable risk factors also decreases the chances of worsening or developing an aortic aneurysm - e.g. stop smoking, reduce alcohol intake and eat a healthy diet.

Regular health checks can help detect any developing aortic aneurysm early and prevent progression and rupture.

**Review Questions**

The correct answers are located below the references.

1. An aortic aneurysm is defined as an irreversible dilatation where the diameter of the aorta is increased by a factor of...?
   1. 5
   2. 1.5
   3. 7
   4. 
   5. 3

2. A patient is admitted to the hospital and has had syphilis, which has gone untreated for years. They get a CT scan and there is an incidental finding. The
radiologist’s report finds an aneurysm 5 cm in diameter. Where is the aneurysm most likely located?

1. Thoracic aorta
2. Abdominal aorta
3. Anterior cerebral artery
4. Left anterior descending coronary artery
5. Left internal iliac artery

3. Which one of the following is not a risk factor for aortic aneurysm?

1. Age over 60
2. Being of Caucasian ethnicity
3. Hypertension
4. Chronic liver disease
5. Smoking

References


Aneurysms: Aneurysms and Aortic Dissection at Merck Manual of Diagnosis and Therapy


Correct answers: 1B, 2A, 3D

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