

THORACIC AORTIC DISSECTION

The Essence of Aortic Dissection

Aortic dissection can be classified as acute if its onset has been less than 14 days or chronic if its onset has been more than 14 days.

Mortality approaches 1% per hour if it is untreated for the first 48 hours, and if there is no surgery, mortality is approximately 90% at 3 months. This decreases to a 56% to 87% survival within 5 years if surgery does occur.

The method by which dissection occurs is that there is a violation of the intima which allows blood to enter the media and dissect between the intimal and the adventitial layers.

Aortic dissection is about recognizing the symptoms and the signs that the patient presents with which are usually:

- Significant pain usually tearing or severe
- Neurology
- Cardiac symptoms including shock and aortic valve abnormalities

Aortic dissection is further about knowing how to properly investigate this patient. CT is really the newfound technique for investigating patients with suspecting dissection. The only other method available is transoesophageal echo if the patient is unstable.

In terms of treatment, there is either medical or surgical treatment. Medical treatment in the past has been reserved for those patients with a descending aorta dissection. However with new stenting procedures, surgical treatments will sometimes take place in this circumstance. Proximal dissections are usually surgically treated.

Initial medical management in the emergency department involves a decrease in the systolic blood pressure and the velocity with which blood is ejected from the left ventricle. This means the use of a beta blocker \pm a venodilator to decrease afterload.

Risk Factors

- Hypertension *** (the most common risk factor)
- Congenital cardiac disorders
- Aortic stenosis
- Marfan's syndrome
- Ehlers-Danlos syndrome

There may also be other causes such as iatrogenic causes and we certainly find that a large number of dissections incur in patients that have had previous cardiac surgery.

Cocaine, pregnancy, and giant cell arteritis can also cause dissection.

Sites Where Dissections Occurs

1. A few centimeters above the aortic valve.
2. Just beyond the insertion of the ligamentum arteriosum.

What Happens When a Dissection Extends?

Firstly, its propagation depends on the blood pressure and the gradient of arterial blood pressure waveform. If the dissection occurs proximally, it can dissect into the aortic valve causing aortic incompetence and it can also extend into the pericardial sac causing a pericardial effusion and tamponade.

The dissection can form a false lumen within the true lumen of the aorta.

The result can be:

1. It can obstruct the true lumen.
2. It can rupture into the true lumen.
3. It can rupture through the wall. This will cause exsanguination of the patient.

Systems of Classification

There are basically two systems of classification, the Stanford and the DeBakey. In both of these, we need to know where the ascending and descending aorta commences. In the Stanford classification, when the ascending aorta is affected it is a Type A Stanford classification and this accounts to up to 70% of cases. The significance of this is that Type A Stanford classifications can have a surgical treatment.

The descending aorta tends to be called the Type B Stanford classification. It occurs in up to about 35% of cases. It begins distal to the origin of the left subclavian artery.

How Do These Patients Present?

1. Pain.

Up to 95% of cases will have severe pain that is resistant to narcotic analgesia.

The pain is severe tearing and ripping and it is maximal at the time of onset.

The pain may reflect the extension of the dissection. This means that as the pain moves it means the dissection is moving. Pain in the anterior chest wall which occurs about 60% to 70% of cases indicates an ascending aorta dissection. Pain in the interscapular region indicates a descending thoracic dissection and pain in the lower back or abdomen may indicate a lower dissection.

2. Neurological symptoms.

As small vessels such as the carotid artery dissect, we can have the patient presenting with confusion or stroke or even coma which can occur in about 20% of cases.

Neurological symptoms may fluctuate and as dissection occurs into spinal vessels, the lower limbs can also be affected, resulting in paraesthesia or paraplegia.

3. Cardiac symptoms.

Pericardial tamponade can occur following dissection into the pericardial sac.

7% of dissections occur in the coronary arteries with the right coronary arteries affected more than the left coronary artery.

Syncope occurs in up to about 20% of cases, most being Type A dissections.

Symptoms can also occur due to local compression although this is uncommon. They can present with superior vena cava syndromes, dysphonia, dysphagia, or a Horner's syndrome.

Clinical Presentation

In up to 80% of patients, hypertension occurs (especially in Type B dissections).

Tachycardia can occur. This may be a result of the dissection or may be the result of pain.

Hypotension can also occur, although this tends to be secondary to rupture, or to cardiac tamponade.

Neurological signs suggest extension into carotid or vertebral circulation.

There may be a blood pressure or pulse deficit in the upper limbs.

Aortic incompetence occurs in approximately 70% of proximal dissections. This presents as a diastolic murmur.

Beck's triad which comprises hypertension, muffled heart sounds, and a raised JVP can also occur.

Pulsus paradoxus can occur.

A pericardial friction rub can be heard.

Frank hemoptysis or hematemesis may occur and indicate an aortic rupture.

Investigations

ECG: Only up to 2% of all ECG shown AMI, however a significant number may show some acute ischemic changes.

Chest x-ray: This may be used to increase our suspicion of a thoracic-aortic dissection, however we must remember that up to 40% of cases have no abnormality.

Chest x-ray findings:

1. Widened mediastinum up to 75%.
2. Aortic arch dilatation 31% to 47%.
3. Change of calibre between the ascending and descending aorta 36% to 67%.
4. Obliteration of the aortic knob.
5. Double density of the aorta; which may suggest a true and a false lumen.

CT scan: Has a very high sensitivity, and with new machines, this approaches 100%.

Echo: Transoesophageal echo can be up to 99% sensitive. It also gives information on the valve and the ventricles.

Aortography: Not performed.

MRI: Maybe 100% sensitive.

Biomarkers: Not really used at this point.

Treatment

Treatment is split into acute stabilization and then either medical or surgical treatment. The initial medical management will involve a negative inotrope.

Firstly, we need to analgesic the patient. These patients may require high levels of narcotic analgesia, which should not be withheld.

The aim is to decrease the systolic blood pressure and also the velocity with which blood is ejected from the ventricle. It is very important that we blunt the reflex tachycardia that might occur if we decrease afterload first. Therefore, we begin with a beta blocker.

Beta blockers that we can use may include Esmolol at a dose of 0.1 to 0.5 mg/kg as an initial load given over 1 minute. We can then use Esmolol as an infusion between 50 and 200 mcg/kg/min.

Metoprolol can also be used. This can be given intravenously at 5 mg increments up to 15 mg aiming at a heart rate of 60 to 80 beats per minute and a systolic blood pressure of 100 mmHg to 120 mmHg.

If we need further blood pressure decrease, a vasodilator could be used. Sodium nitroprusside decreases afterload and can be given at a dose of 0.5 to 10 mcg/kg/min.

GTN decreases both preload and afterload and is given at a dose of 5 mcg/min to 50 mcg/min.

Medical management was always the norm with Type B thoracic aortic dissections in the past and was associated with approximately 80% survival rate in this group. However with new endovascular repair techniques, more and more patients are being treated for Type B dissections.

Thoracic and Abdominal Aneurysms

An aneurysm is defined as being a dilatation of greater than 1.5 times the normal diameter of a vessel.

A fusiform aneurysm is one where the entire circumference of the vessel wall is dilated and involved whereas a saccular aneurysm is one where only part of the circumference is involved.

True Aneurysms

A true aneurysm contains all layers of the vessel wall. It includes connective tissue disease, familial history, smoking and hypertension as its major causes. This type of aneurysm may expand and rupture.

Pseudo Aneurysm

It consists partly of the vessel wall and partly of fibrous and connective tissue. It tends to develop at sites of arterial catheterisation or anastomoses. It may spontaneously thrombose if it is in a small vessel.

Micotic Aneurysm

It develops as a result of infection in the vessel wall.

Abdominal Aortic Aneurysm Characteristics

Most abdominal aneurysms are infrarenal. They account for 90% – 95% of all abdominal aneurysms. There is an increased risk of rupture when the aneurysm is >3cm. It will require surgical repair at ≥ 5 cm.

- Males greater than females
- Most are greater than 60 years old
- 5% – 10% are between 65 and 79 years old
- Familial tendency is about 18%, ie. 18% with AAA will have a first degree relative with the same
- The risk increases with the number of years of smoking and decreases with the number of years since quitting.

How do Patients with Abdominal Aortic Aneurysm Present?

Beware as AAA can mimic other diseases.

The patient with syncope, that is usually followed by a several abdominal pain or back pain needs to have abdominal aortic aneurysm excluded. The pain may be described as severe or abrupt in onset. 50% of patients describe the pain as being ripping or tearing. The pain may also be in different areas such as the flank or hip.

How good are we at picking up abdominal aneurysms on clinical examination?

Abdominal examination sensitivity is as follows;

- 29% for diameters up to 3.9cm
- 50% for diameters of 4cm – 4.9cm
- 76% for \geq 5cm diameter.

Remember that the lack of abdominal pain does not indicate an intact aorta. Some patients may in fact look very well, well enough to be discharged.

Acute Rupture of AAA can present in several ways:

- Periumbilical ecchymosis (Cullen's sign)
- Flank ecchymosis (Grey Turner sign)
- Scrotal or vulvar haematoma can occur
- Inguinal mass with blood dissection into the perineum
- The iliopsoas sign can occur if retro peritoneal blood irritates the psoas muscle
- Neuropathy can occur if blood compresses the femoral nerve

THE PRESENCE/RUPTURE OF AAA TYPICALLY DOES NOT ALTER FEMORAL ARTERY PULSATION.

In the patient that presents with the lower GI bleed we must suspect aortoenteric fistula.

Investigations

Ultrasound: this has a greater than 90% sensitivity for the presence of aneurysm and increasing diameter.

CT: IV contrast is useful to determine the anatomical layers of the aneurysm and also if there is any retro peritoneal haemorrhage.

Treatment

Surgery. 50% of patients with ruptured aneurysms who reach the operating room die.

A patient presenting with severe abdominal pain and hypotension, or syncope and significant abdominal pain does not need investigation but needs to be referred urgently to vascular surgery for treatment in the operating room.