AORTIC DISSECTION

LONGITUDINAL CLEAVAGE OF THE AORTIC MEDIA CREATED BY A DISSECTING COLUMN OF BLOOD

IN-HOSPITAL MORTALITY RATE IS 27%

EPIDEMIOLOGY:

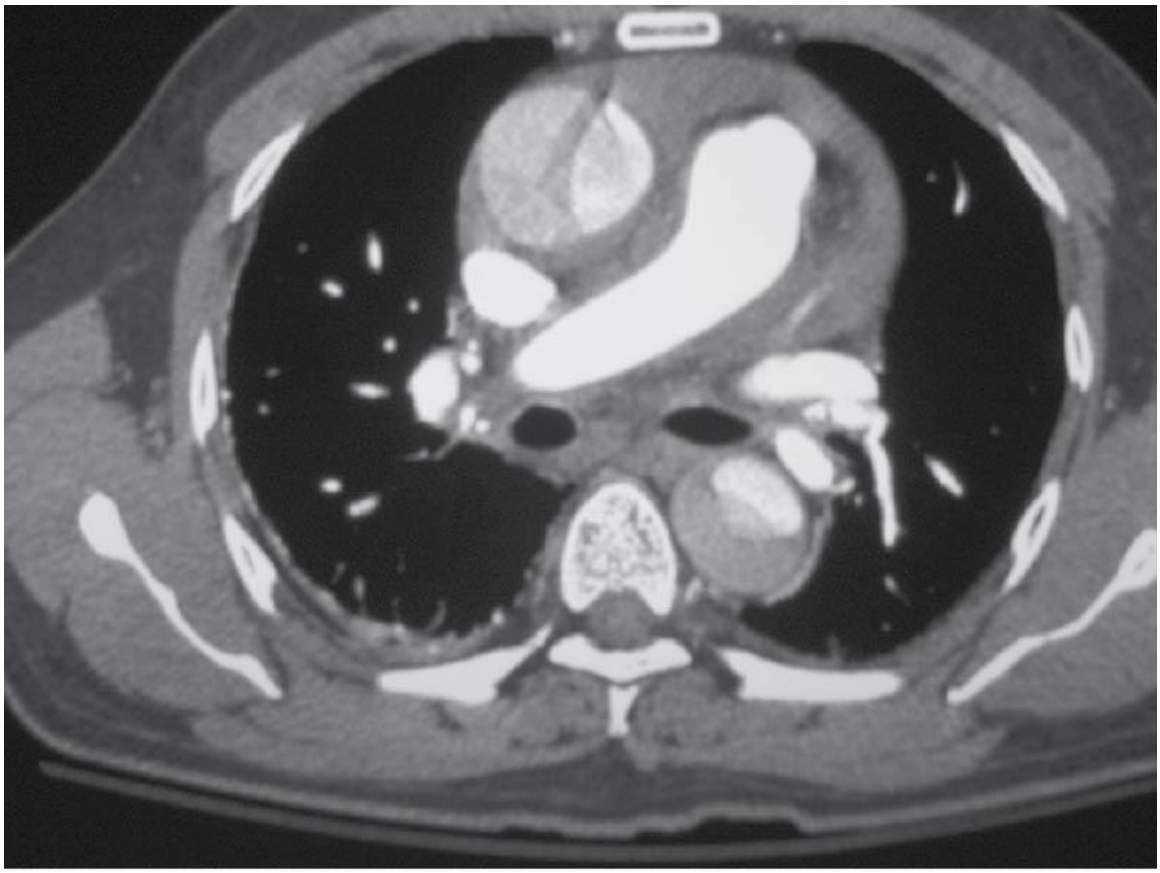
- Increases with age and is more frequent in men
- Mortality is 1-5/100,000 per year
- HYPERTENSION IS THE MOST COMMON RISK FACTOR
- History of cardiac surgery in ~18%
- Bicuspid aortic valve in 14% of all patients but more often in proximal dissections
- May have positive family history
- Uncommon before age 40 except in association with congenital heart disease, EHLERS-DANLOS, or MARFAN'S SYNDROME → account for 5% cases
    - Women with Marfans are at particular risk during pregnancy
- Also occurs with stimulant use, exertion and trauma
    - Can be seen in those who undergo cardiac surgery or IABP insertion

ANATOMY AND PHYSIOLOGY:

- The descending aorta flexes just distal to the left subclavian artery, where the mobile aorta is tethered
- Dissection occurs through a degeneration of the media characterised by loss of smooth muscle cells and elastic tissue, accompanied by scarring, fibrosis and hyaline-like changes (PREVIOUSLY CALLED CYSTIC MEDIAL NECROSIS → this term is misleading)

PATHOPHYSIOLOGY:

- Repetitive hydrodynamic forces contribute to weakening of the aortic intima and to medial degeneration → primarily affect the ascending aorta → sustained hypertension intensifies these forces and results in an increase in medial degeneration
- A bicuspid aortic valve may disrupt laminar flow and reorient flow of blood toward the aortic wall, causing local injury
- In Marfan's and Ehler's-Danlos → normal hydrodynamic forces act on an already weakened aortic wall
- As a result of medial degeneration and repeated flexion of the aorta, hydrodynamic stress tears the aortic intima and a column of blood gains access into the aortic media
    - An alternative theory suggests that these forces damage the VASO-VASORUM, which rupture into media
- Once a dissecting haematoma is established in the media, it migrates in antegrade or retrograde fashion, OR BOTH → forming a false lumen
    - Can rupture back into the TRUE LUMEN → spontaneous cure

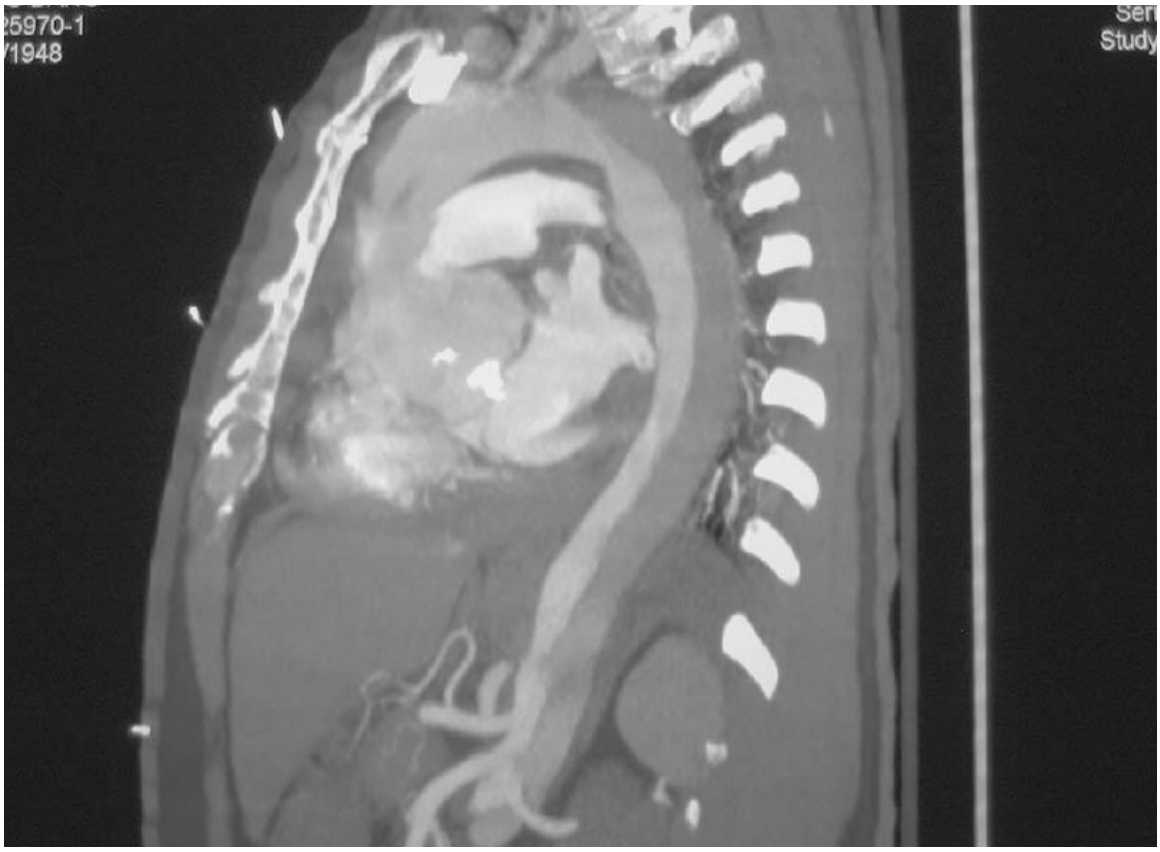


SHOWS TRUE AND FALSE LUMEN

  - Can rupture through the adventitia into the pericardial sac or pleural cavity → tamponade and haemothorax results respectively → because outer wall is thin, rupture out is more common
- The most important factors favouring continued dissection of the aorta are:
  - Degree of elevation of blood pressure
  - Steepness of pulse wave (dp/dt)

CLASSIFICATION:

- STANFORD CLASSIFICATION:
  - Based on involvement of THE ASCENDING AORTA
  - Type A → involve ascending aorta, type B do NOT
  - In the International Registry of Acute Aortic Dissection (IRAD) → 62% are TYPE A, 38% type B
    - Patients with type B tend to be older, heavy smokers with chronic lung disease and generalised atherosclerosis/HT



TYPE-B DISSECTION DISTAL TO SUBCLAVIAN ARTERY

- Two other closely related groups to dissection:
  - INTRAMURAL HAEMORRHAGE → haematoma contained within the aortic wall and occurs in about 10% of dissection → rupture of vasa vasorum
  - PENETRATING ATHEROSCLEROTIC ULCERS → occur in older hypertensive patients
- ACUTE → less than two weeks duration, CHRONIC → more than 2 weeks

CLINICAL FEATURES:

- HISTORY:
  - PAIN → by far the most common complaint (if painless → chronic)
    - Pain is usually excruciating, occurs abruptly, worst at onset, typically described as SHARP, rather than tearing or ripping
    - Migration of pain consistent with propagation suggests dissection but occurs in ONLY 17%
    - Often have associated features of visceral pain → nausea, vomiting, diaphoresis, apprehension
  - SYNCOPE → occurs in 9% cases of dissection. Heralds dissection into the pericardium causing tamponade, but can occur with transient disturbance to cerebral blood flow
  - NEUROLOGICAL SYMPTOMS → 17% cases
- PHYSICAL EXAMINATION:

- Presentation varies greatly, depending on location and extent of dissection
- Generally, the patient appears APPREHENSIVE
- Can have PSEUDOHYPOTENSION → due to disruption of flow to subclavian arteries
- AORTIC REGURGITATION → more common with type A dissections
- Look for signs of TAMPONADE → ↑JVP, muffled heart sounds, tachycardia, hypotension
- When integrity of one of the branches of the aorta is compromised, EXPECT ISCHAEMIC FINDINGS → intimal flap may cover true lumen of branch vessel or dissecting haematoma may compression adjacent true lumen:
  - Proximal dissections can cause strokes or coma
  - Anterior spinal artery → ischaemic paraperesis, ischaemic peripheral neuropathy
  - Mesenteric vessels → bowel ischaemia
  - Renal vessels → renal failure
  - Iliac vessels → lower limb ischaemia
  - In 3% cases, proximal dissection can dissect into OSTIUM OF CORONARY ARTERY → most frequently the RIGHT CORONARY causing inferior/posterior STEMI → occurs in about 0.1-0.2% of MI and can lead to catastrophic consequences if fibrinolysis administered
- Pulse deficits/discrepancies in BP can be helpful BUT OCCUR IN ONLY 30% CASES

Table 83-1 Characteristics of Aortic Dissection from the International Registry of Acute Aortic Dissection[4]

	CHEST PAIN (%)	SYNCOPE (%)	AORTIC INSUFFICIENCY MURMUR (%)	PULSE DEFICIT (%)	NORMAL CXR (%)	WIDENED MEDIASTINUM ON CXR (%)	NORMAL ECG (%)	ISCHEMIA (%)	LEFT VENTRICULAR HYPERTROPHY (%)
All ($n = 464$)	73	9	32	15	12	62	31	15	26
Type A ($n = 289$)	79	13	44	19	11	63	31	17	25
Type B ($n = 175$)	63	4	12	9	16	56	32	13	32

DIAGNOSTIC STRATEGIES:

- Routine lab tests are OF LITTLE VALUE
- With the advent of TOE and CT angiogram, formal angiography has been supplanted in diagnosis
- With a mortality rate in excess of 1% per hour after onset of aortic dissection, a diagnostic study should be performed AS SOON AS POSSIBLE

Table 83-2 Sensitivities and Specificities of Imaging Modalities for Diagnosing Aortic Dissection

TEST	TEE	HELICAL CT	MRI
Sensitivity (%)	98	100	98
Specificity (%)	95	98	98

- **ECG:**
  - Useful in EXCLUDING AMI, but 15% patients with dissection may have ECG abnormalities suggesting ischaemia
  - If dissection into RCA → inferior MI and constellation of symptoms (chest pain, nausea/vomiting, diaphoresis) difficult to differentiate
  - LVH common due to long-standing HT
- CXR:
  - Abnormal in 80-90% cases BUT ABNORMALITIES ARE NON-SPECIFIC
  - Mediastinal widening occurs in majority but may be difficult to differentiate from aortic tortuosity
  - Up to 12% may have normal CXR
  - Other findings:
    - DOUBLE-DENSITY → suggests true and false lumen
    - Obliteration of aortic knob
    - Displacement of trachea to right
- ECHOCARDIOGRAPHY:
  - TRANSTHORACIC → insensitive due to poor visualisation of the aorta, but can pick up pericardial effusion or aortic regurgitation
  - TRANSOESOPHAGEAL:
    - HIGHLY SENSITIVE
    - NOT READILY AVAILABLE, but is procedure of choice in unstable patients
- COMPUTED TOMOGRAPHY:
  - CT AORTOGRAPHY is a reliable test for diagnosing aortic dissection and is test of choice in most places
- MAGNETIC RESONANCE IMAGING:
  - **Appealing as it can detect low-grade dissection in stable patients and sensitivity/specificity is EXCELLENT**
  - Requires no contrast, radiation and is non-invasive
  - AVAILABILITY AND TIME TAKEN TO SCAN ARE PROBLEMS

DIFFERENTIAL CONSIDERATIONS:

- Signs and symptoms vary
- Often patients are thought to have AMI, PE, CHF

- Several clinical syndromes are PARTICULARLY SUGGESTIVE OF AORTIC DISSECTION:
    - Sudden-onset chest pain that is worst at onset
    - Migratory pain
    - Chest pain with concomitant neurologic deficits or syncope
    - Chest pain with pulse deficits
    - Pain above and below the diaphragm
- Think about dissection in patients with:
    - Pain or dysfunction in extremity
    - Peripheral neurologic diagnoses
    - Stroke syndromes
    - New diagnosis of pericardial effusion, tamponade
    - New aortic insufficiency

MANAGEMENT:

- EMERGENCY DEPARTMENT:
    - Early therapy IS CRITICAL and should be instituted while diagnostic tests are being performed
    - Opioids for pain relief and to decrease sympathetic tone
    - TWO GOALS OF MEDICAL MANAGEMENT:
        - Reduce blood pressure → 100-120mmHg with vasodilator (after beta blockade) → use nitroprusside, GTN
        - Decrease the rate of rise of the arterial pulse to diminish shear stress → BETA BLOCKERS FIRST TO LOWER DP/DT (consider esmolol, labetalol or metoprolol IV titrating to HR of ~60)
    - In patients with electromechanical dissociation or profound hypotension → pericardiocentesis may raise the BP while awaiting definitive surgery
- SURGERY:
    - Type A aortic dissections require prompt surgical treatment with graft replacement of ascending aorta to redirect blood into the true lumen
        - If AI present, it can be corrected with AVR
        - Patients with type A dissection have in-hospital mortality of 27% when treated surgically vs 56% when treated medically
- Definitive treatment of TYPE-B acute aortic dissections is LESS CLEAR:
    - These patients tend to be worse surgical candidates
    - Uncomplicated distal dissections are generally treated with blood pressure control with in-hospital mortality of 10%
    - Surgery indicated in type B dissections if:
        - Uncontrolled hypertension
        - Occlusion of major arterial trunk
        - Frank aortic leaking or rupture
        - Development of a localised aneurysm
            - These patients have 30-day mortality rate of 32%

- Interventional stent-graft and fenestration techniques are replacing surgery for complicated type-B dissections, especially for patients with renal and mesenteric ischaemia