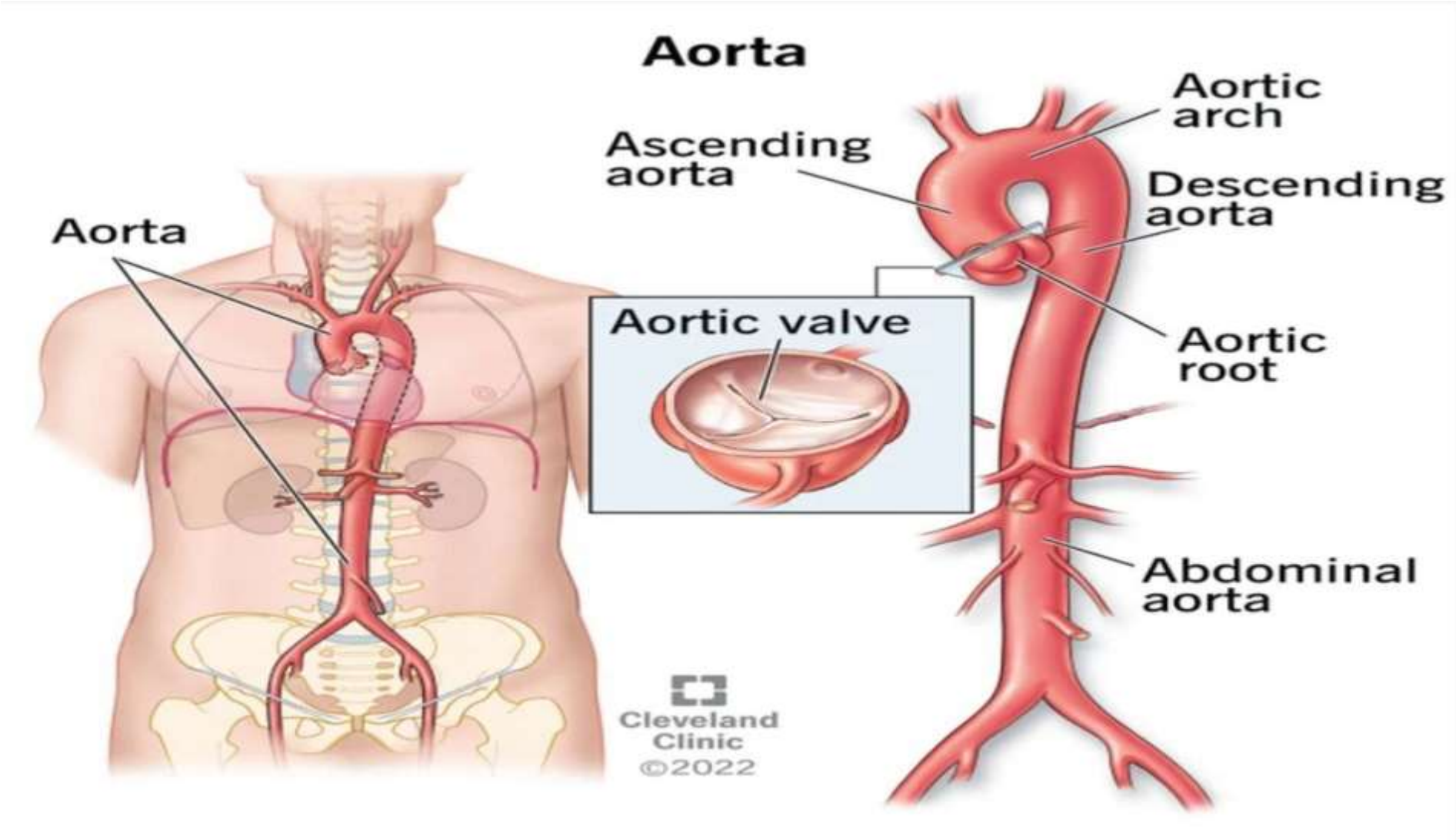
Aortic Dissection


Aorta
Aortic arch
Ascending aorta
Descending aorta
Aorta
Aortic valve
Aortic root
Abdominal aorta
Cleveland Clinic
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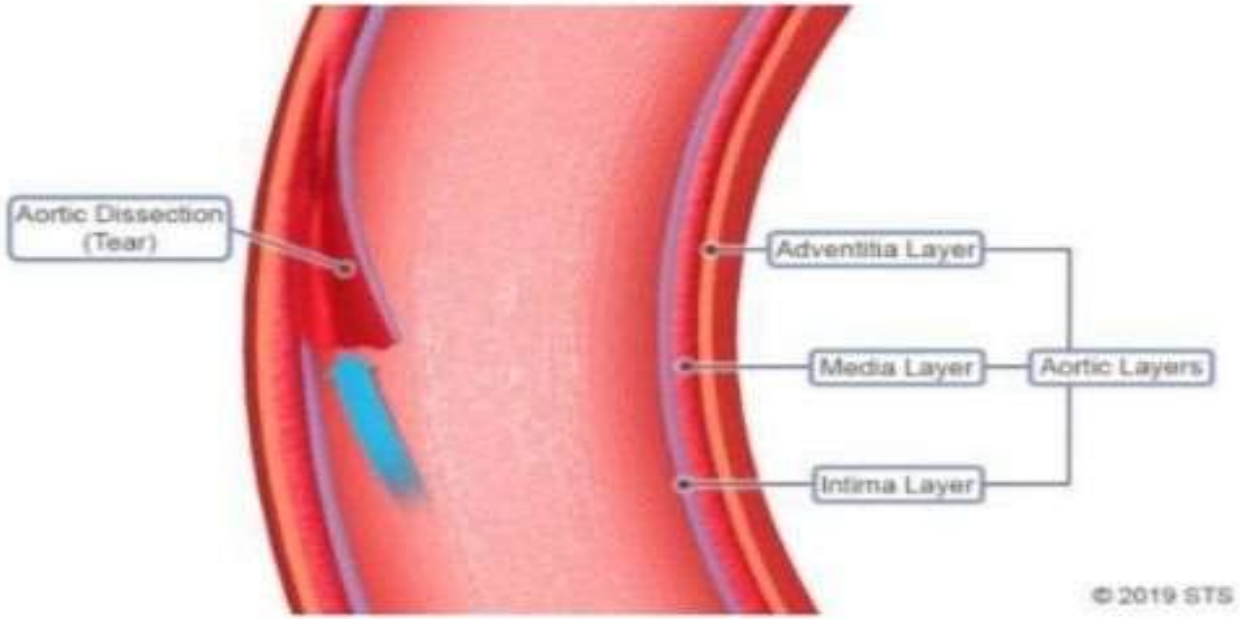
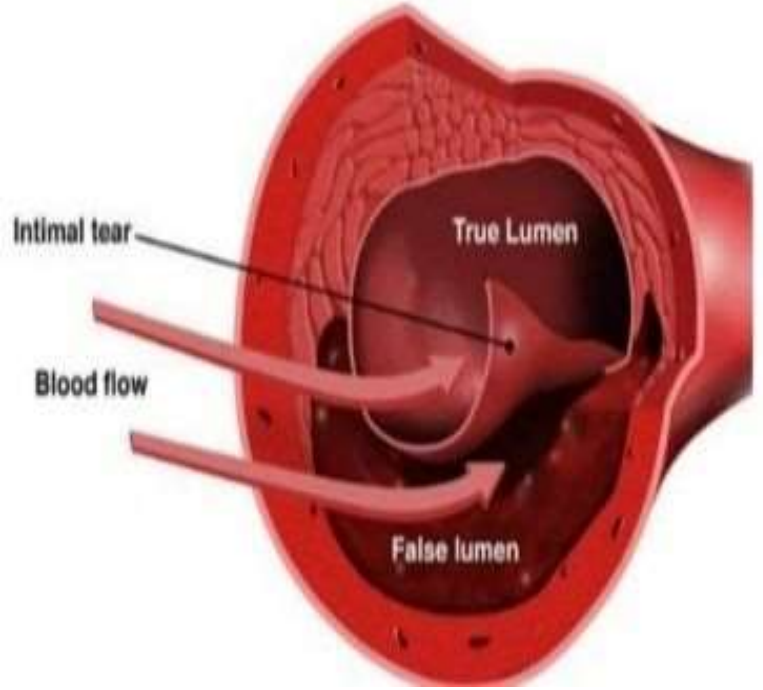
- Aortic dissection occurs when a breach in the integrity of the aortic wall (tearing of intima),allows arterial blood to enter the media, which is then split into two layers(intima and media)creating a false lumen alongside the existing or true lumen.

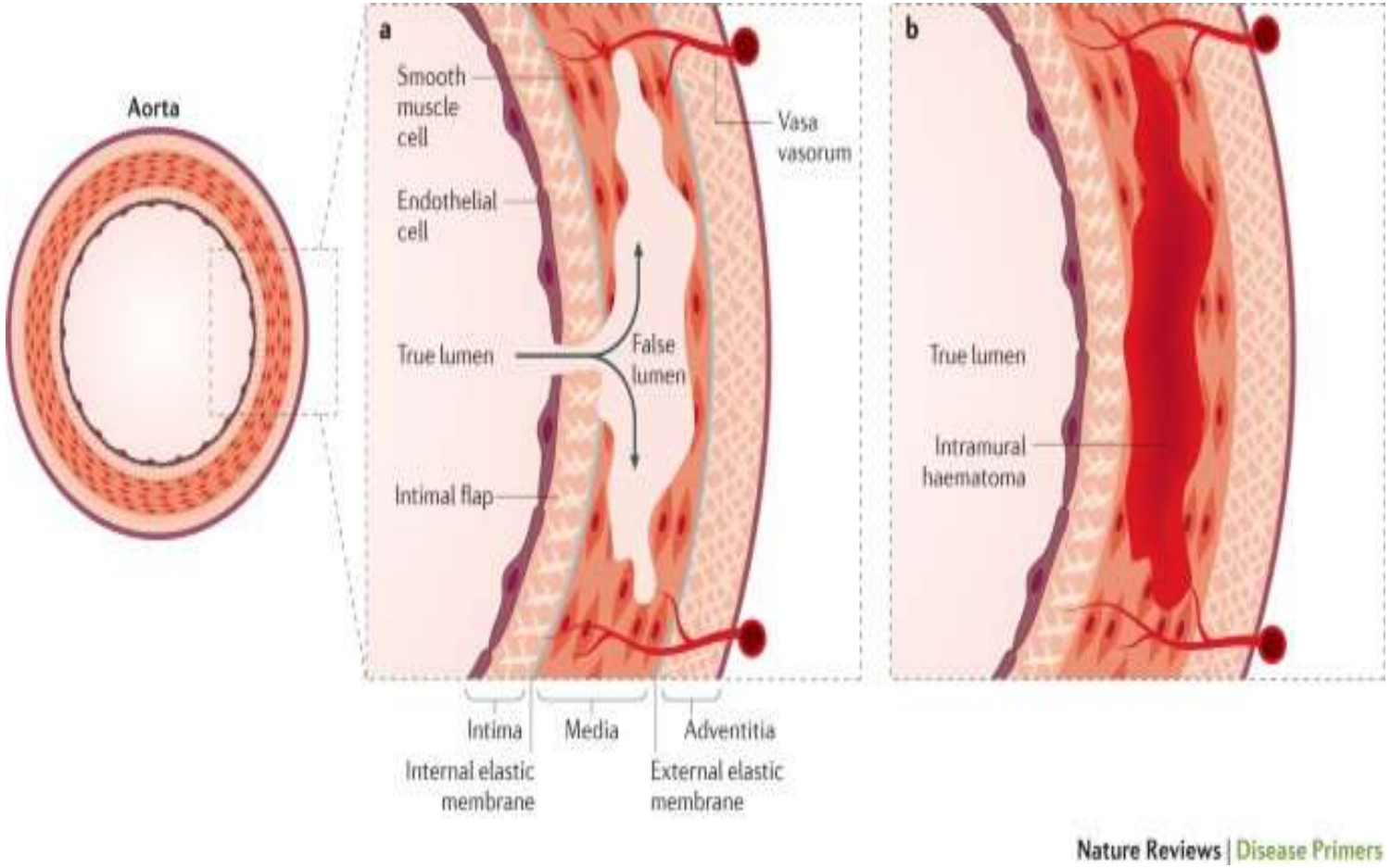
- The aortic valve may be damaged and the branches of the aorta maybe compromised. Typically, the false lumen eventually re-enters the true lumen, creating a double-barrelled aorta, but it may also rupture into the left pleural space or pericardium with fatal consequences.





Pathogenesis:

The primary event is often a spontaneous or iatrogenic tear in the intima of the aorta; multiple tears or entry points are common. Other dissections are triggered by primary hemorrhage in the media of the aorta,which then ruptures through the intima into the true lumen. This form of spontaneous bleeding from the vasa vasorum is sometimes confined to the aortic wall, when it may present as a painful intramural haematoma.



Classification :

❑ Stanford classification :

1. Type A (proximal): involves the ascending aorta (includes retrograde extension from descending aorta).

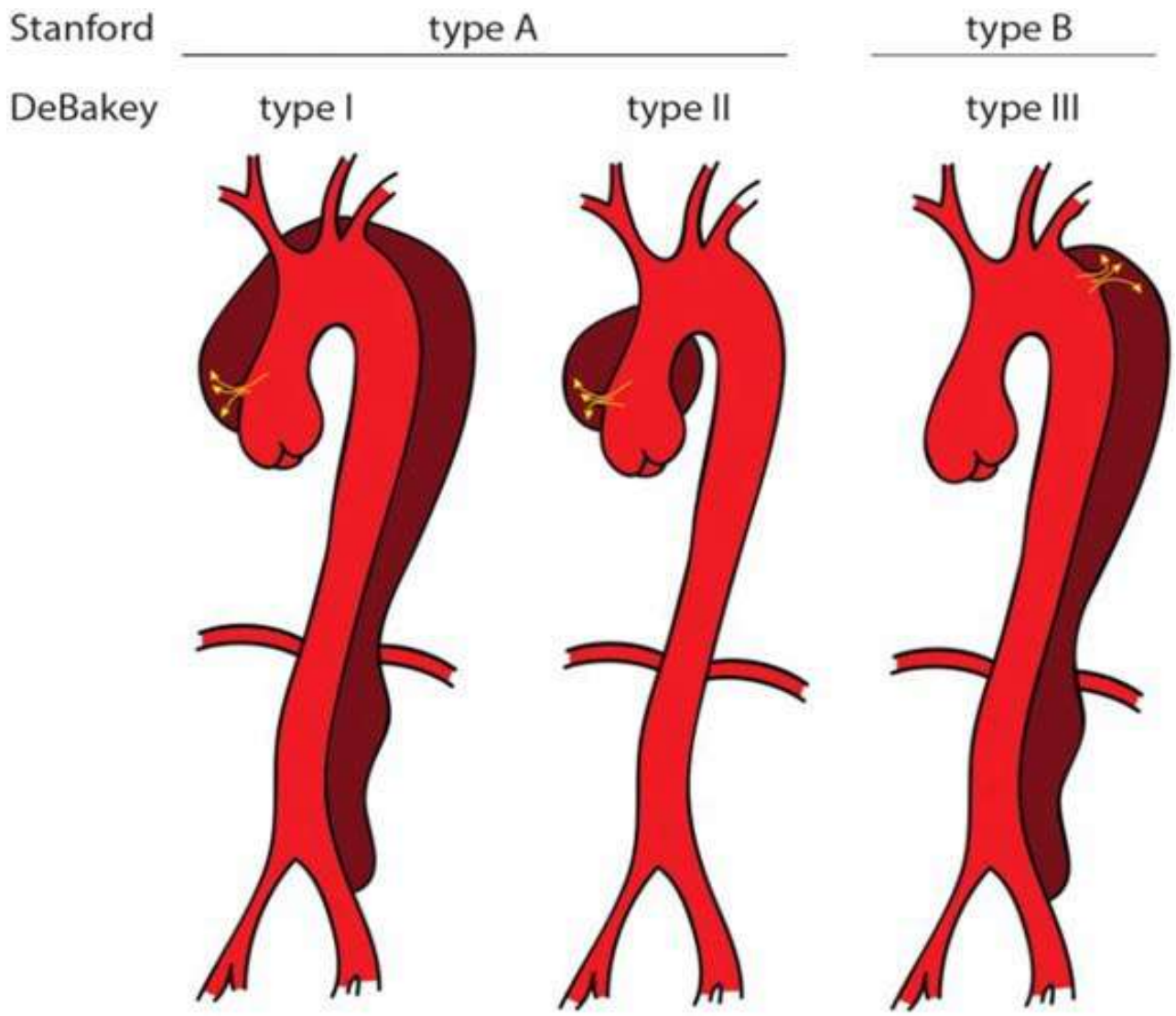
2. Type B (distal) : is limited to the descending aorta (distal to the take off of the subclavian artery).

❑ Debakey classification :

1) Type 1: originates in the ascending aorta and to at least the aortic arch.it is most often seen in patient less than 65 years of age and is the most lethal form of the disease

2) Type 2: originates in and is limited to the ascending aorta.

3) Type 3: begins in the descending aorta and extends distally. Classically in elderly patients with atherosclerotic disease and hypertension



Risk factors:

a. Long-standing systemic HTN (present in 70% of patients)

b. Cocaine use (may be remote)

c. Trauma

d. Connective tissue diseases, such as Marfan and Ehlers–Danlos syndrome

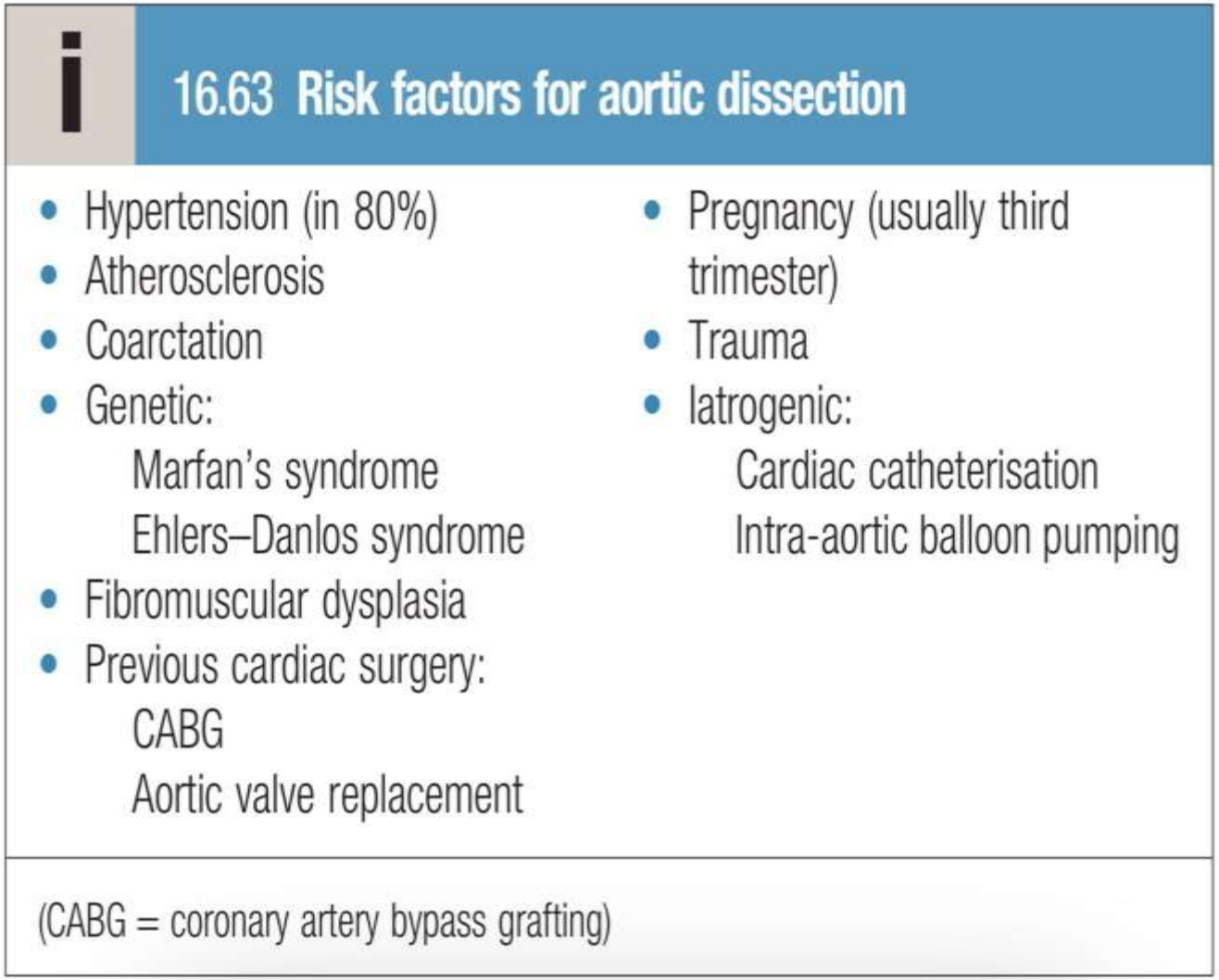
e. Bicuspid aortic valve

f. Coarctation of the aorta

g. Third trimester of pregnancy

h. Pre-existing aortic aneurysm

i. Intimal ulcer due to atherosclerosis



16.63 Risk factors for aortic dissection

- Hypertension (in 80%)
- Atherosclerosis
- Coarctation
- Genetic:
  - Marfan's syndrome
  - Ehlers–Danlos syndrome
- Fibromuscular dysplasia
- Previous cardiac surgery:
  - CABG
  - Aortic valve replacement
- Pregnancy (usually third trimester)
- Trauma
- Iatrogenic:
  - Cardiac catheterisation
  - Intra-aortic balloon pumping

(CABG = coronary artery bypass grafting)

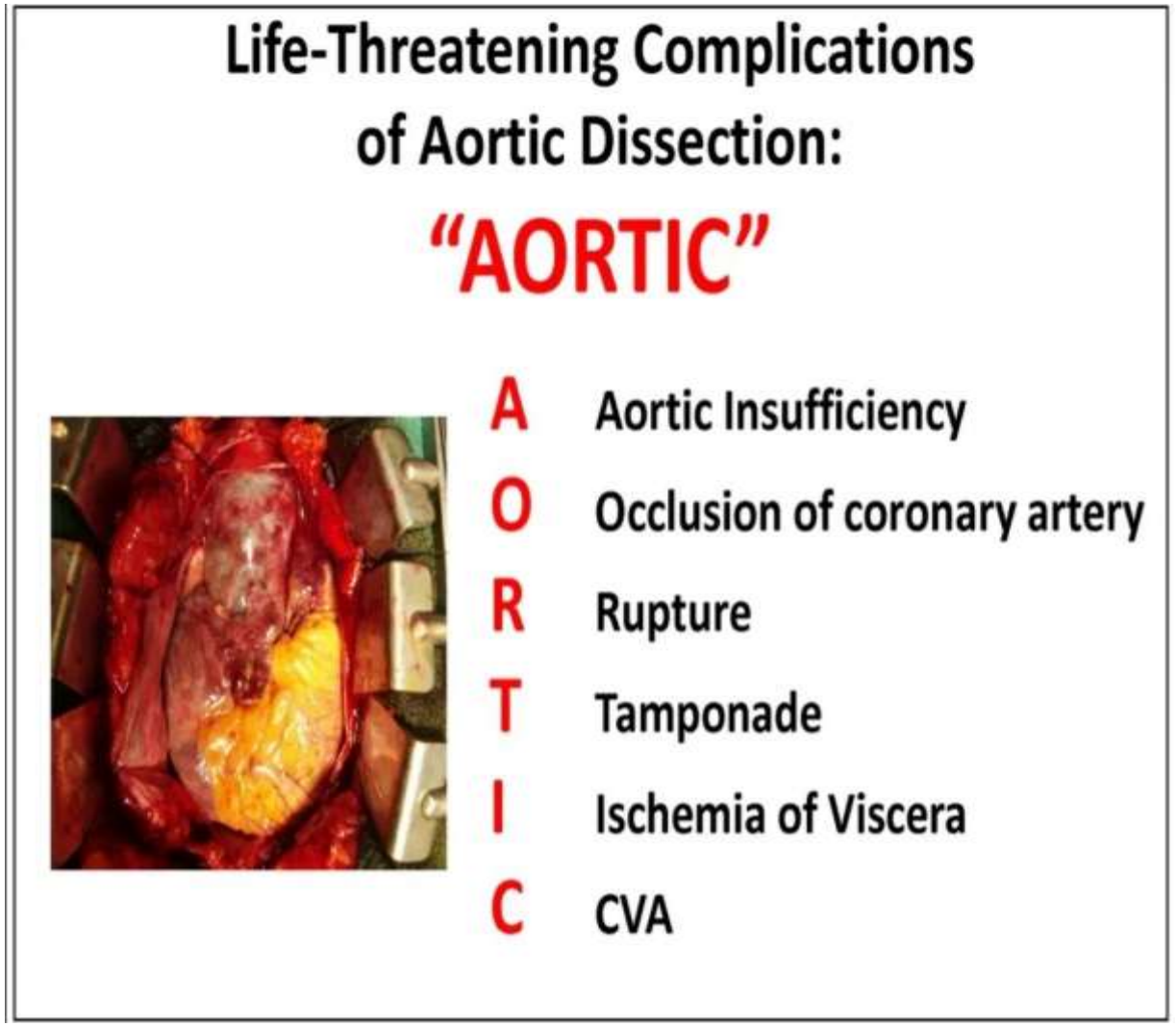
Complications :AORTIC

1. blood could continue in false tunnel until reach a branch of aorta compressing it: renal artery : decreased renal blood flow Subclavian artery: decreased flow to arms Carotid artery: cause neurological manifestations (hemiplegia,hemianesthesia) Coronary arteries: MI(Type A dissection)

2. aortic regurgitation: one half to two thirds of cases, which is mainly due to dilatation of the aortic root and thus valves leaflets pulled apart (diastolic decrescendo murmur heard best at left 3rd intercostal space accentuated by expiration and leaning forward.

3. cardiac tamponade : blood could back up from aorta to heart and enter pericardium(Type A dissection)

4. blood in false lumen could puncture a hole in tunica media and externa and blood leak in mediastinum (fatal due to blood loss and shock)



Clinical features :

A. Chest pain : severe stabbing pain

B. Diaphoresis.

C. Neurological manifestations (hemiplegia and hemianesthesia) due to obstruction of carotid artery.

- The most common cause of death is rupture of the dissection into the pericardial , pleural or peritoneal cavity

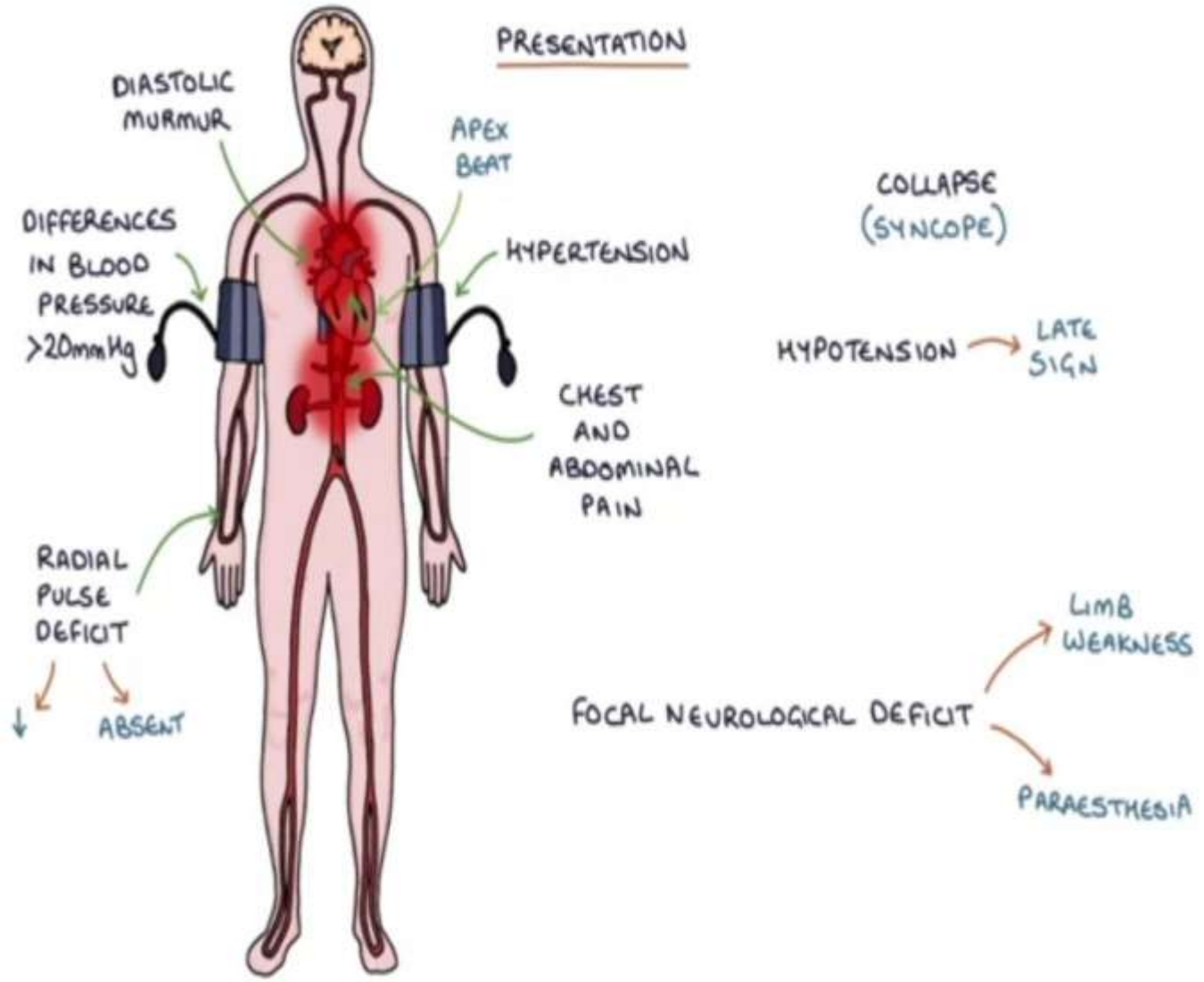
Site	Interscapular/retrosternal
Onset	Very sudden
Character	Tearing or ripping
Radiation	Back,between shoulders
Associated symptoms	Sweating,Syncope,Focal neurological signs,Signs of limb ischemia,Mesenteric ischemia
Timing	Acute presentation, Prolonged duration
Exacerbating/relieving factors	Spontaneous No maneuver relieves pain
Severity	Very sever

Physical examination findings :

Pulse or BP asymmetry between limbs

Diastolic murmur (aortic regurgitation)

Distant heart sound



Investigations:

1) ECG findings in aortic dissection are usually non-specific. Possible findings include:

A. Non-specific ST-segment changes

B. Atrioventricular block (second degree)

C. Sinus bradycardia

2) Lab tests: there is no particular laboratory findings for the diagnosis of aortic dissection. Possible laboratory findings associated with aortic dissection may include:

i. D-dimer elevation: has highest diagnostic value in the first hour of symptom presentation(aortic dissection leads to immediate increase in d-dimer

ii. Troponin elevation maybe observed in approximately 25% of patients with aortic dissection type A. It may be the result of hemodynamic stress and is not associated with poorer prognosis.

iii. Hematuria

3)CXR shows:

a) Widened mediastinum (>8 mm on AP view

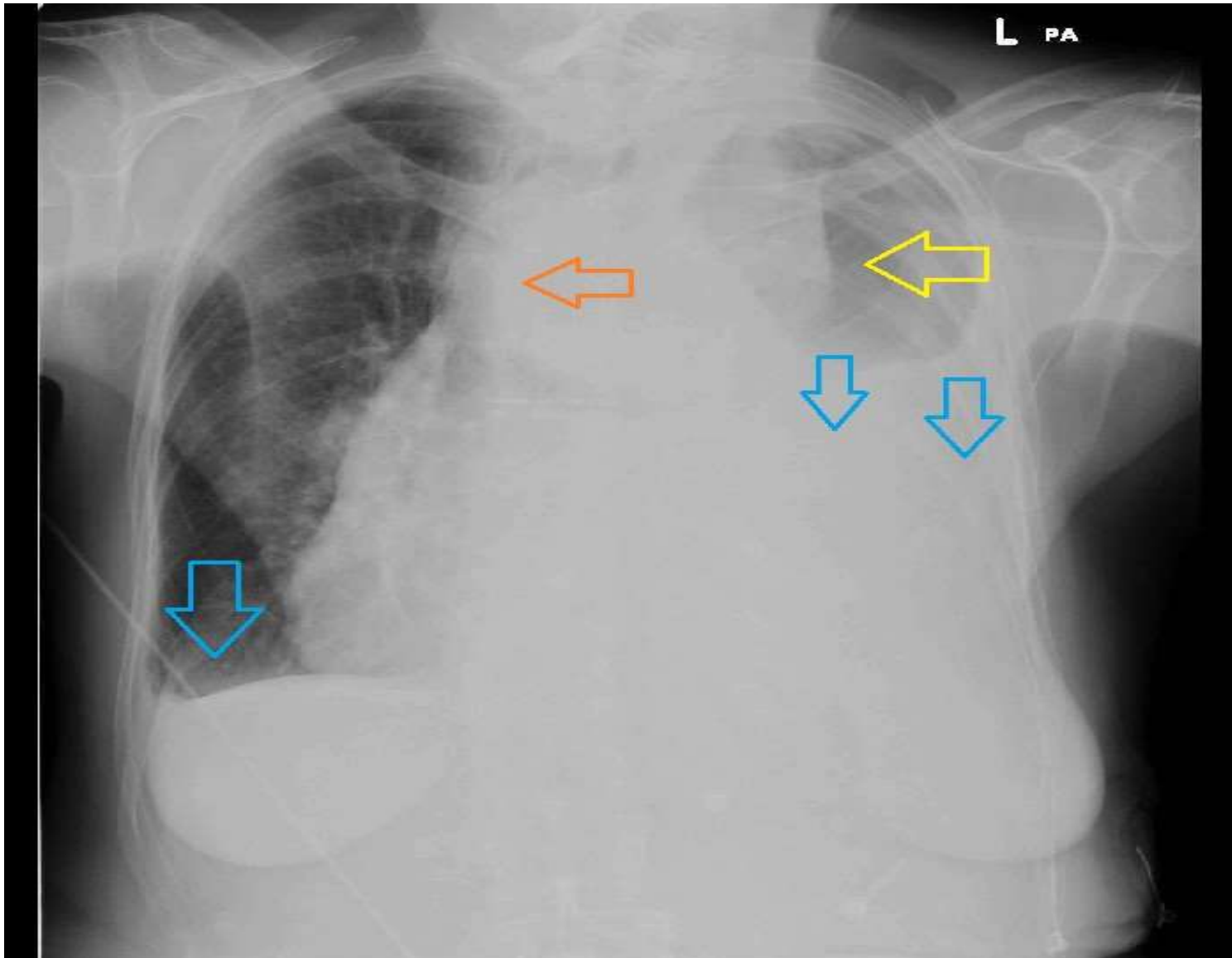
b) Wide aortic contour

c) Tracheal deviation

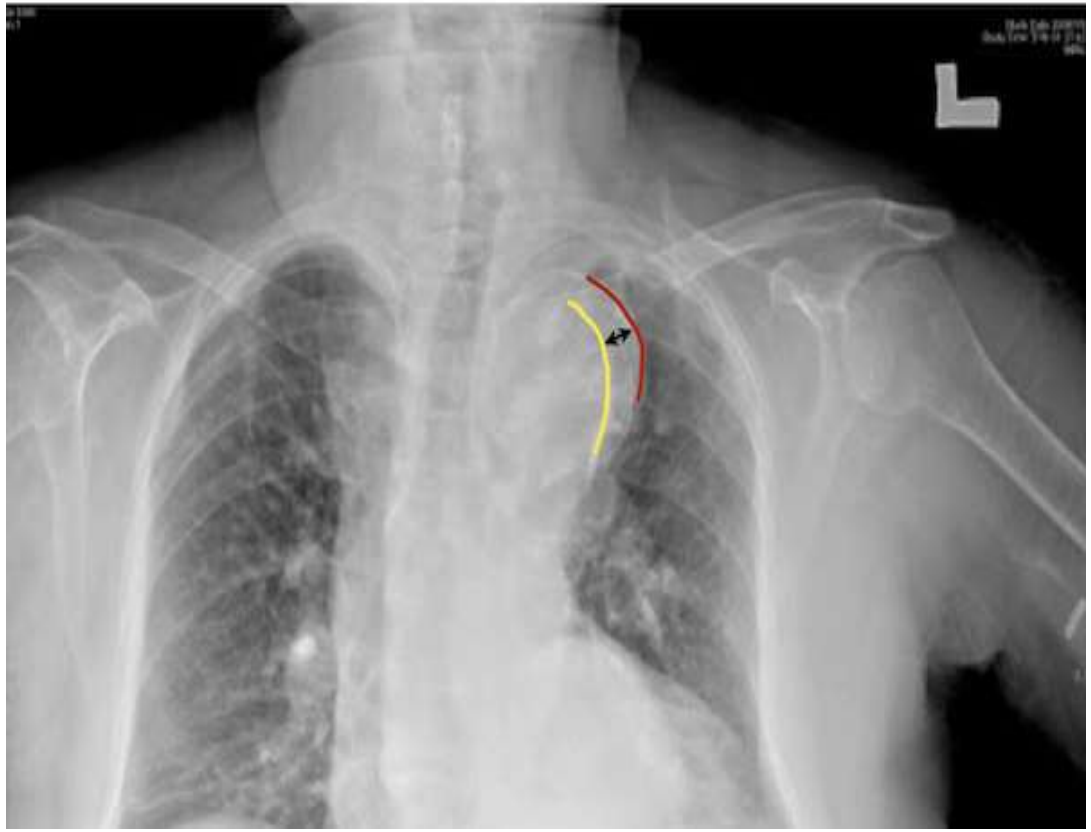
d) Loss of aortic knob

e) The calcium sign :a separation of two layers of the displaced intimal calcification from the outer aortic wall by at least 10 mm

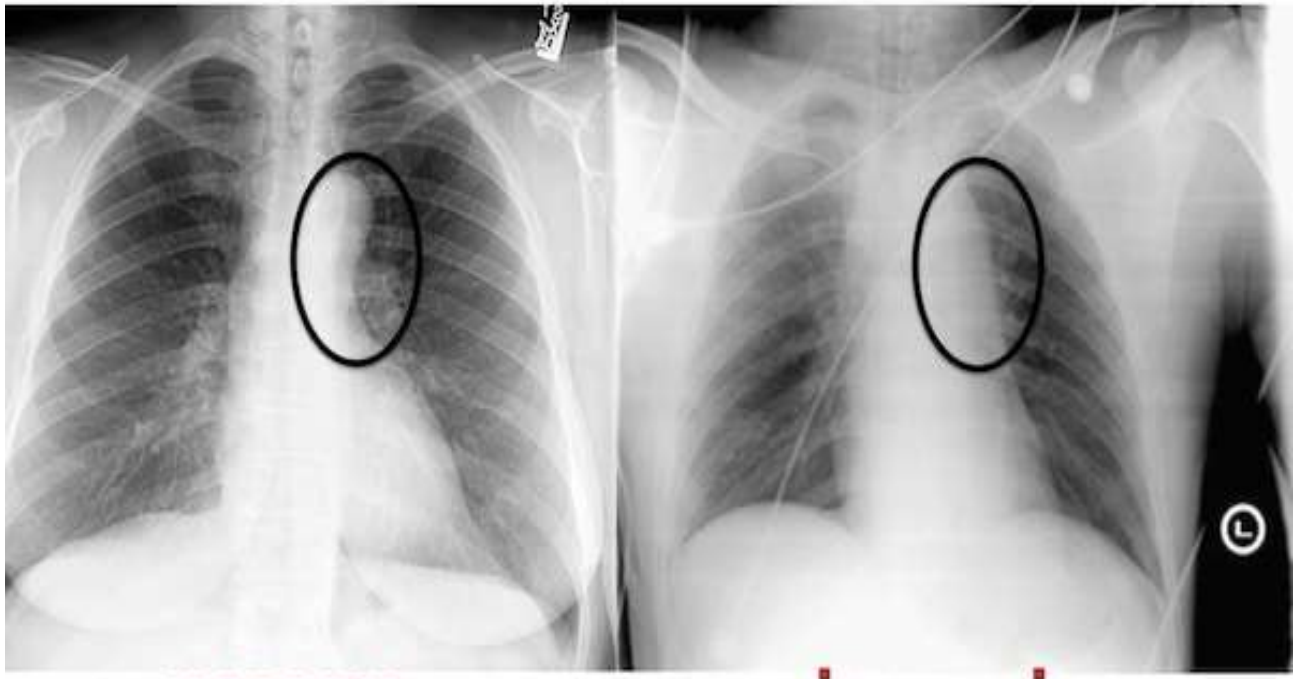
f) Pleural effusions may be noted especially left sided pleural effusion



CALCIUM SIGN >1CM



LOSS OF AORTIC KNOB

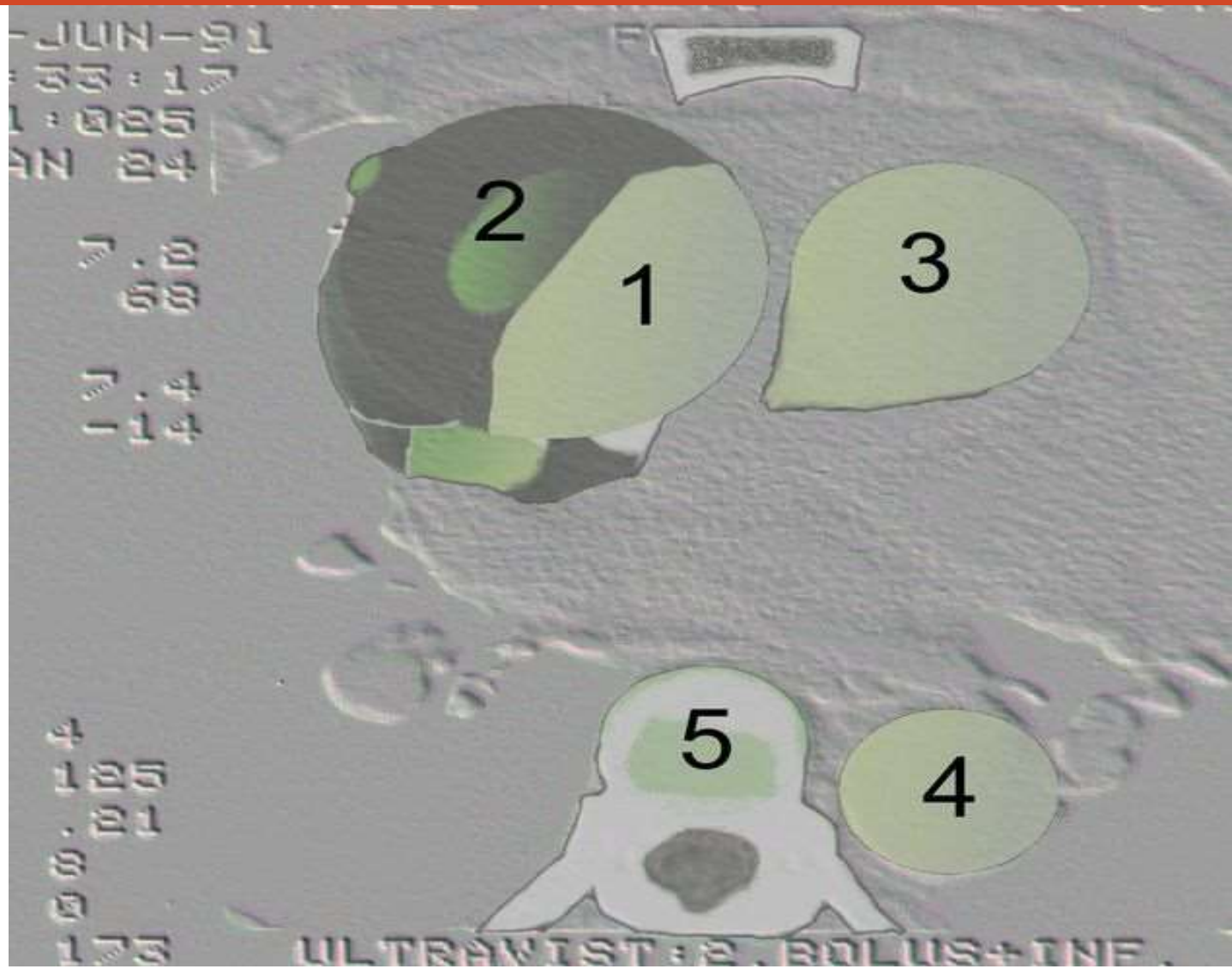


NORMAL

DISSECTION

4) CT angiogram (CTA) :fast non-invasive test that will give an accurate three-dimensional view of the aorta.

- ❑ The false lumen of the dissection plane appears larger on CT and MRI.

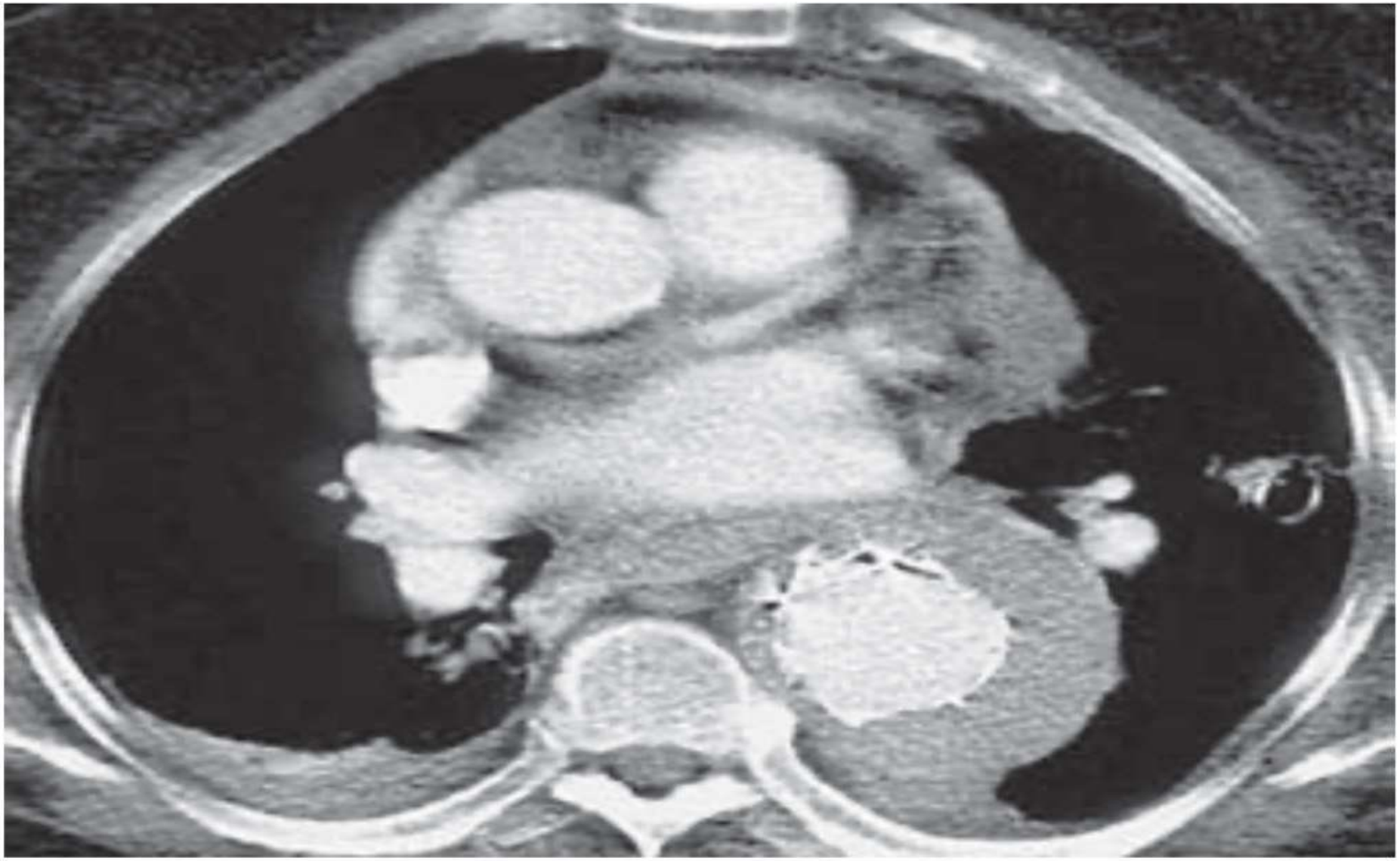


Type A dissection

Type B dissection

CT scan showing an intimal flap in the descending aorta and a large pleural effusion

CT scan after endoluminal repair



5)MRIs currently the gold standard test for the detection and assessment of aortic dissection, with a sensitivity of 98% and a specificity of 98%.

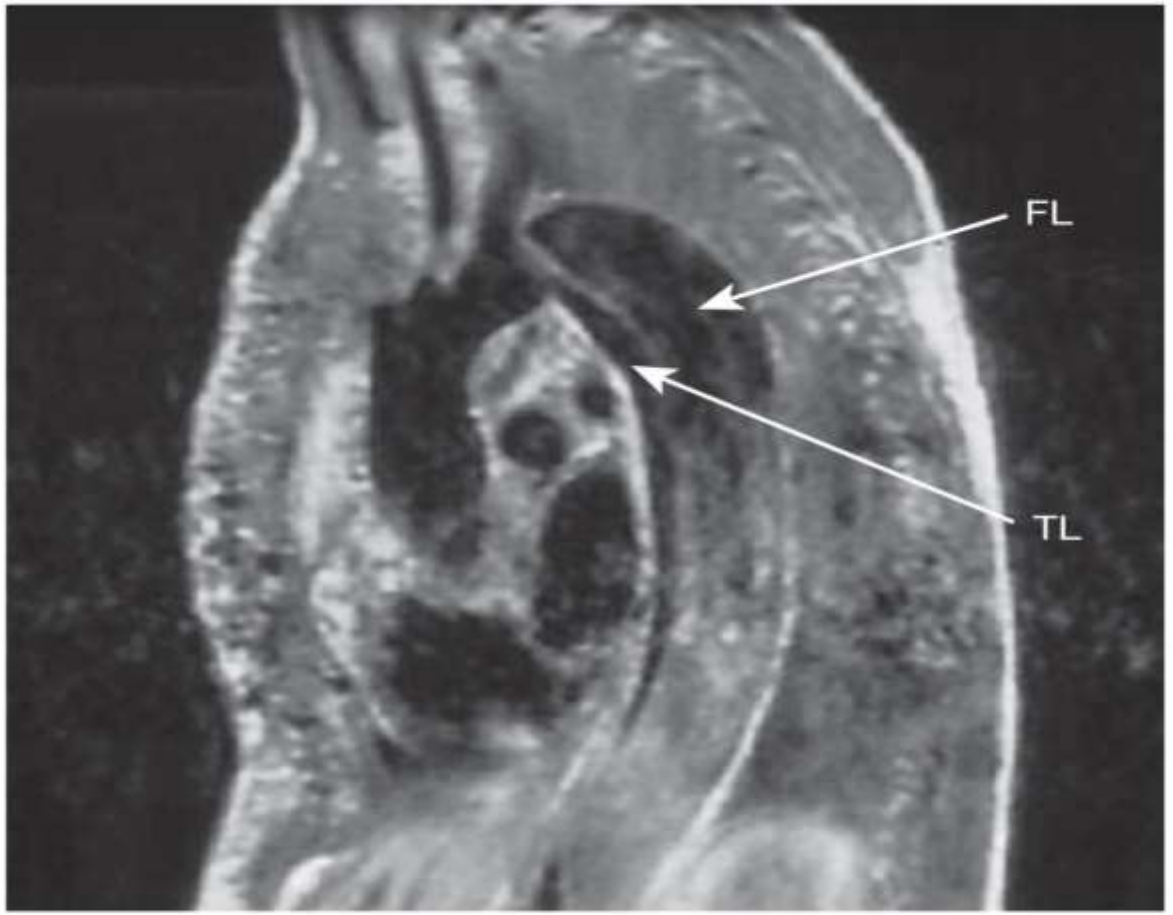
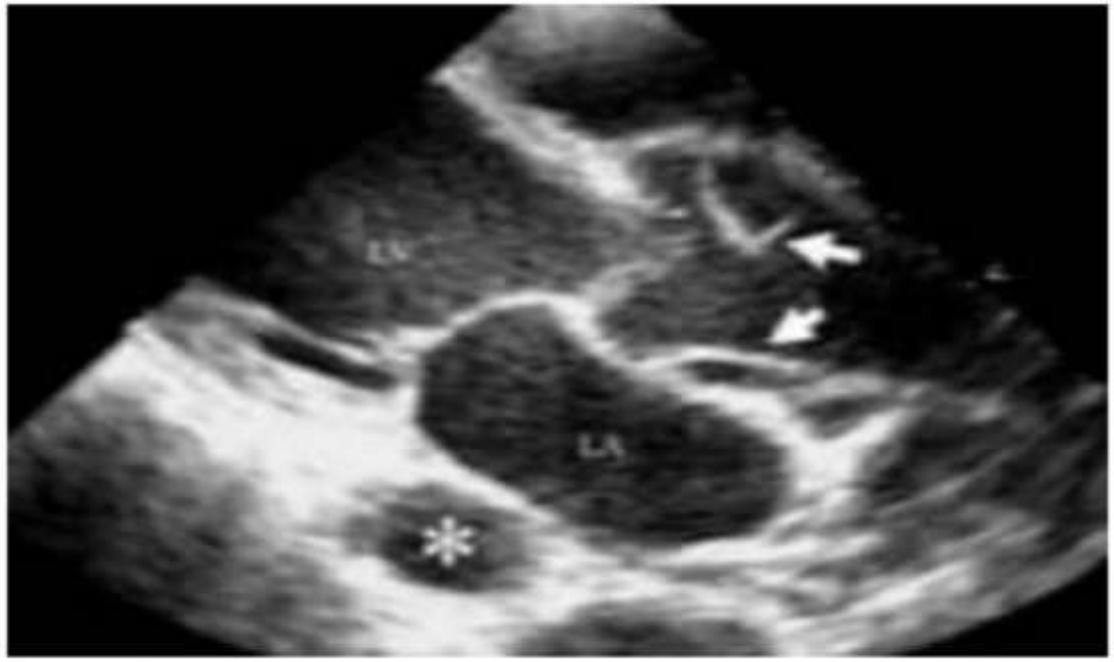


Fig. 16.73 Sagittal view of an MRI scan from a patient with long-standing aortic dissection, illustrating a biluminal aorta. There is sluggish flow in the false lumen (FL), accounting for its grey appearance. (TL = true lumen)

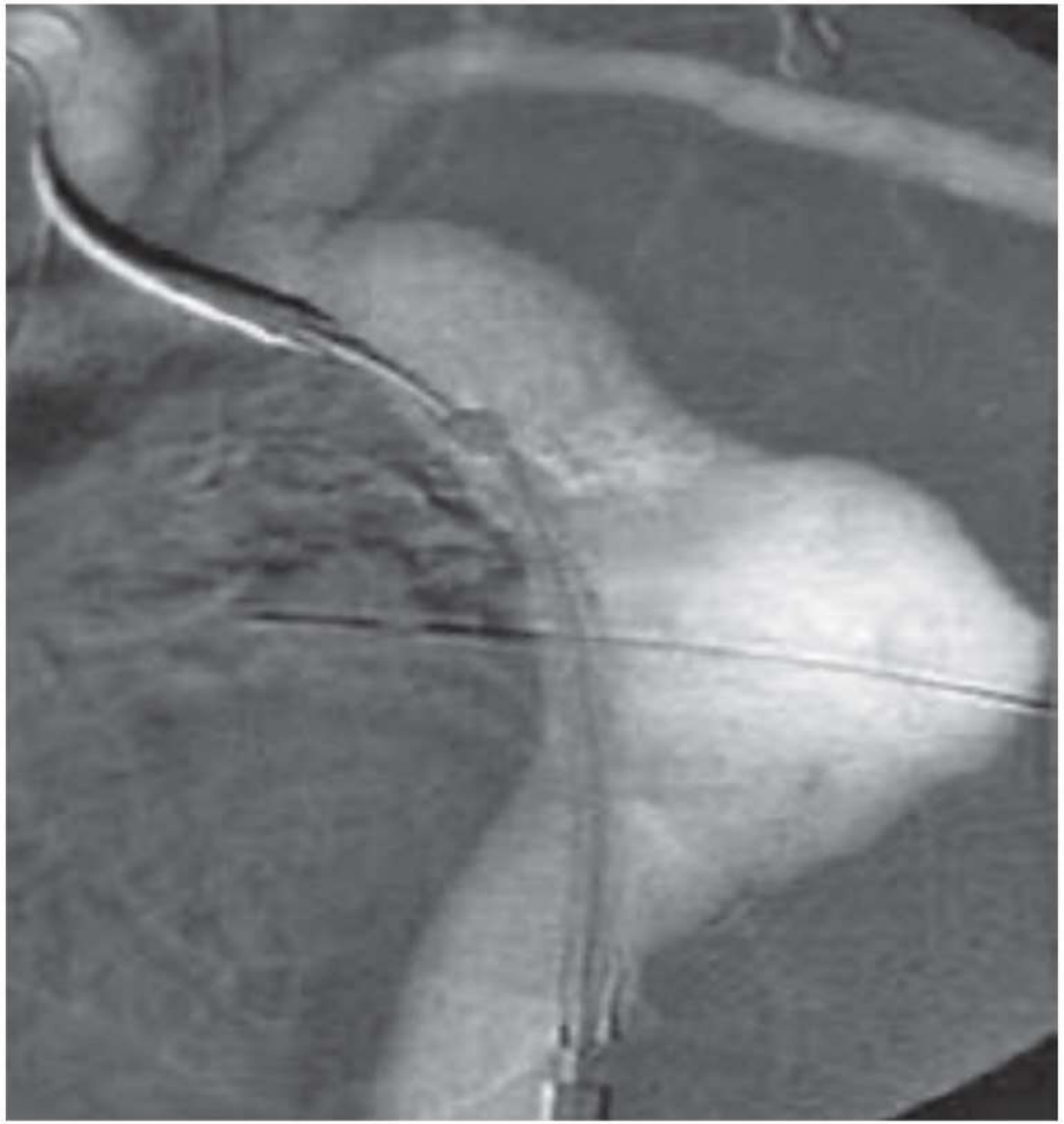
6) TEE (Transesophageal echocardiography) is a very high sensitivity and specificity; it is noninvasive and can be performed at the bedside.

-Prolapse of intimal flap through the aortic valve seen in transesophageal echocardiography is diagnostic of aortic dissection complicated by aortic regurgitation



Transthoracic parasternal long-axis view demonstrates an acute aortic dissection involving the proximal ascending aorta. The image shows intimal flaps (arrow) in the dilated ascending aorta, and not dilated, unaffected descending aorta (*). LV: left ventricle, LA: left atrium.

7) Aortic angiography was once the gold standard, and has largely been replaced by CTA. It can be used for patients in whom noninvasive imaging is nondiagnostic, or during endovascular intervention for dissection.



Treatment :

1) Initiate medical therapy immediately

a. IV β-blockers (labetalol, esmolol) to lower heart rate and diminish the force of left ventricular ejection, thus reducing shear stress on intima

b. Goal systolic BP 100 to 120 mm Hg, to be achieved as soon as possible to minimize shear stress. Can be accomplished with β-blockers, or can add sodium nitroprusside

2) For type A dissections—surgical management

a. Most cases of type A dissections should be treated as surgical emergencies to prevent complications such as MI, aortic regurgitation, or cardiac tamponade

b. Open surgery is still the standard of care

c. High risk of mortality, 1% to 2% per hour after symptom onset without intervention

3) For type B dissections—medical management for hypertensive patient:

a. Lower blood pressure as quickly as possible. First-line drugs include IV βblockers such as labetalol, esmolol

b. Pain control with morphine or hydromorphone

c. Unrelenting symptoms may require surgical treatment—both open and endovascular surgical options exist

Management of patient that become hypotensive due to these complications (aortic regurgitation ,rupture,cardiac tamponade) we give him :

I. IV fluids to increase blood volume

II. Alpha 1 agonist like norepinephrine

III. Sometimes blood also to increase blood volume

IV. Goal MAP>70 mm Hg

A 64-year-old female presents to the ED with sudden onset of severe chest pain that occasionally radiates to her back. She has a history of angina and takes nitroglycerin, but this time the pain is much worse and is not relieved with nitroglycerin. PMH is significant for HTN. She is afebrile, and HR = 105, BP =160/105, RR = 17. Cardiac enzymes are negative. CXR shows a widened mediastinum. What is the likely diagnosis?

A. Myocardial infarction

B. Aortic dissection

C. Stroke

D. Acute aortic regurgitation

E. Hypertensive emergency

1)pathogenesis,classification,risk factors and complication presented by Rana Emran

2)clinical features and physical examination and treatment of A dissection presented by Dana Alreqeb

3)investigations and treatment of B dissection presented by Salwa AlSakarneh

Thank you