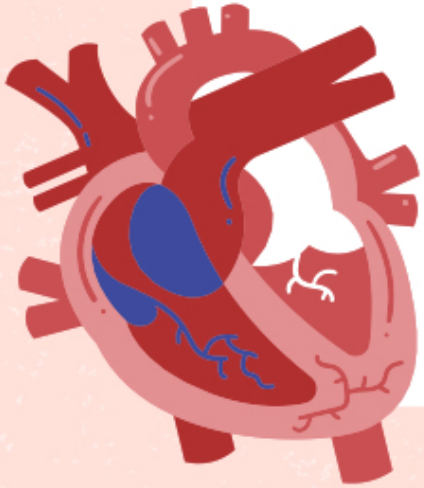
THROMBOEMBOLIS



Presented by:
Toqa alsalamat
Heba alamairh
Rama Jehad

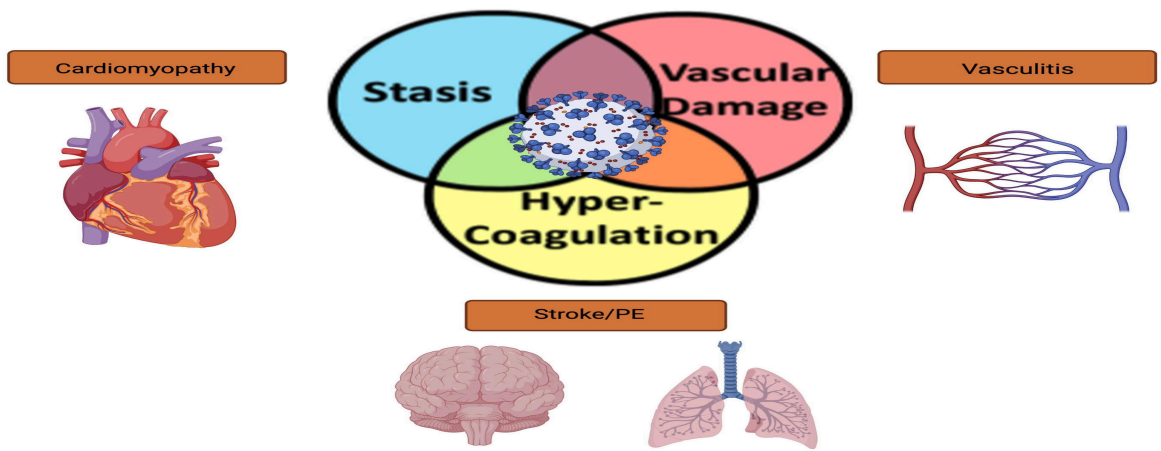
THROMBOEMBOLISM: THE FORMATION AND/OR MIGRATION OF BLOOD CLOTS IN DIFFERENT LOCATIONS
OF THE VENOUS OR ARTERIAL VASCULATURE THAT CAN OCCLUDE OR IMPAIR THE PULMONARY OR SYSTEMIC
CIRCULATION.

Virchow triad : A combination of pathophysiological factors that promote thrombus

formation, including endothelial damage (e.g., inflammation, trauma), venous stasis

(e.g., varicosis, immobilization), and hypercoagulability (e.g., increased platelet adhesion,

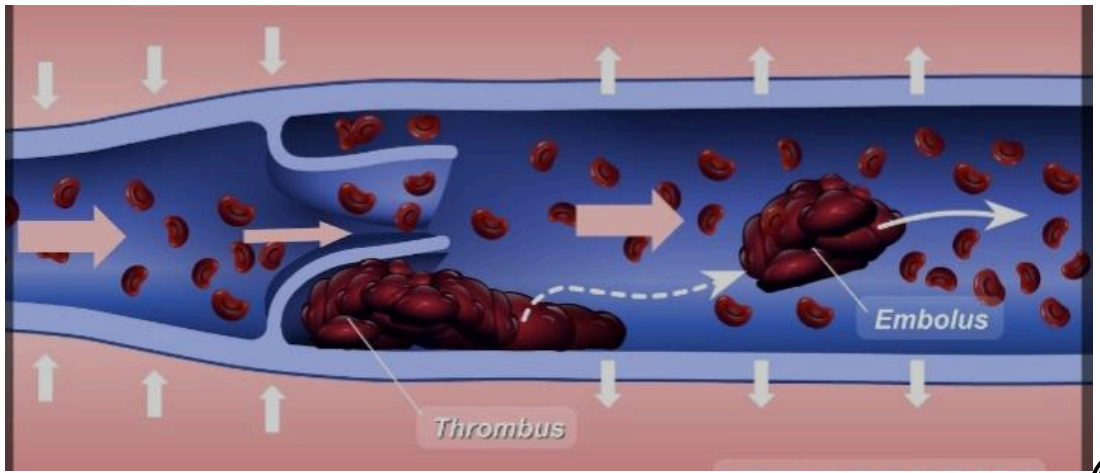
thrombophilia).



VENOUS THROMBOEMBOLISM (VTE)

Blood clots that form within the venous vascular system, dislodge, and travel to a distant location, e.g., the pulmonary arteries via the right heart .

o Examples include deep venous thrombosis, pulmonary embolism, portal vein thrombosis, cerebral venous thrombosis.



RISK FACTORS FOR VTE

Transient (Reversible)	Persistent (irreversible)
1. surgical factors : - Surgery under general anesthesia . - cesarean delivery .	**1. Patient factors :** - Age > 60 years. - Personal or family history of DVT or PE. - Anatomic predisposition to venous stasis (e.g., compression of the iliac veins due to pelvic malignancy).
2. immobilization : - Acute illness requiring complete bed rest . - Lower extremity injury with restricted mobility for ≥ 3 days. - Long-distance travel.	**2. Prothrombotic chronic illnesses :** - Active cancer . - Nephrotic syndrome . - Autoimmune disorders : APLA syndrome, inflammatory bowel disease. - Hereditary thrombophilia : Factor V Leiden , Antithrombin III deficiency, protein C deficiency, and protein S deficiency , Prothrombin mutation.
3. Estrogen-related factors : - Pregnancy. - Use of OCPs or HRT.	
4. Patient factors: - Obesity , smoking , IV drug use .	

DEEP VEIN THROMBOSIS (DVT)

The formation of one or more blood clots in a deep vein, typically of the lower extremities.

Proximal DVT: DVT of the lower extremity affecting the femoral vein, profunda

femoris vein, and/or the popliteal vein (up to the calf vein trifurcation) .

- Distal DVT: DVT of the lower extremity that is confined to the veins beyond the

calf vein trifurcation (i.e., below the knee joint).

Clinical features :

Deep vein thrombosis may be asymptomatic.

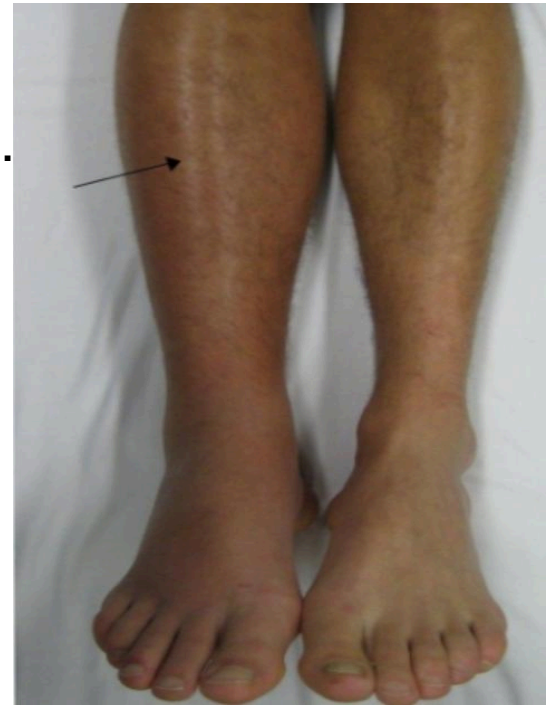
A. Localized unilateral symptoms:

- Typically affects deep veins of the legs, thighs, or pelvis .
- Swelling, feeling of tightness or heaviness.
- Warmth, erythema, and possibly livid discoloration .
- Progressive tenderness, dull pain.
- Distention of superficial veins .
- Distal pulses are normal.

B. **General symptoms**: fever.

C. **Possible signs** of **pulmonary embolism**:

dyspnea, chest pain, dizziness, weakness.



DIAGNOSIS

A. **Doppler analysis and Duplex ultrasound**

o Initial test for DVT; noninvasive.

o High sensitivity and specificity for detecting proximal thrombi (popliteal and

femoral) .

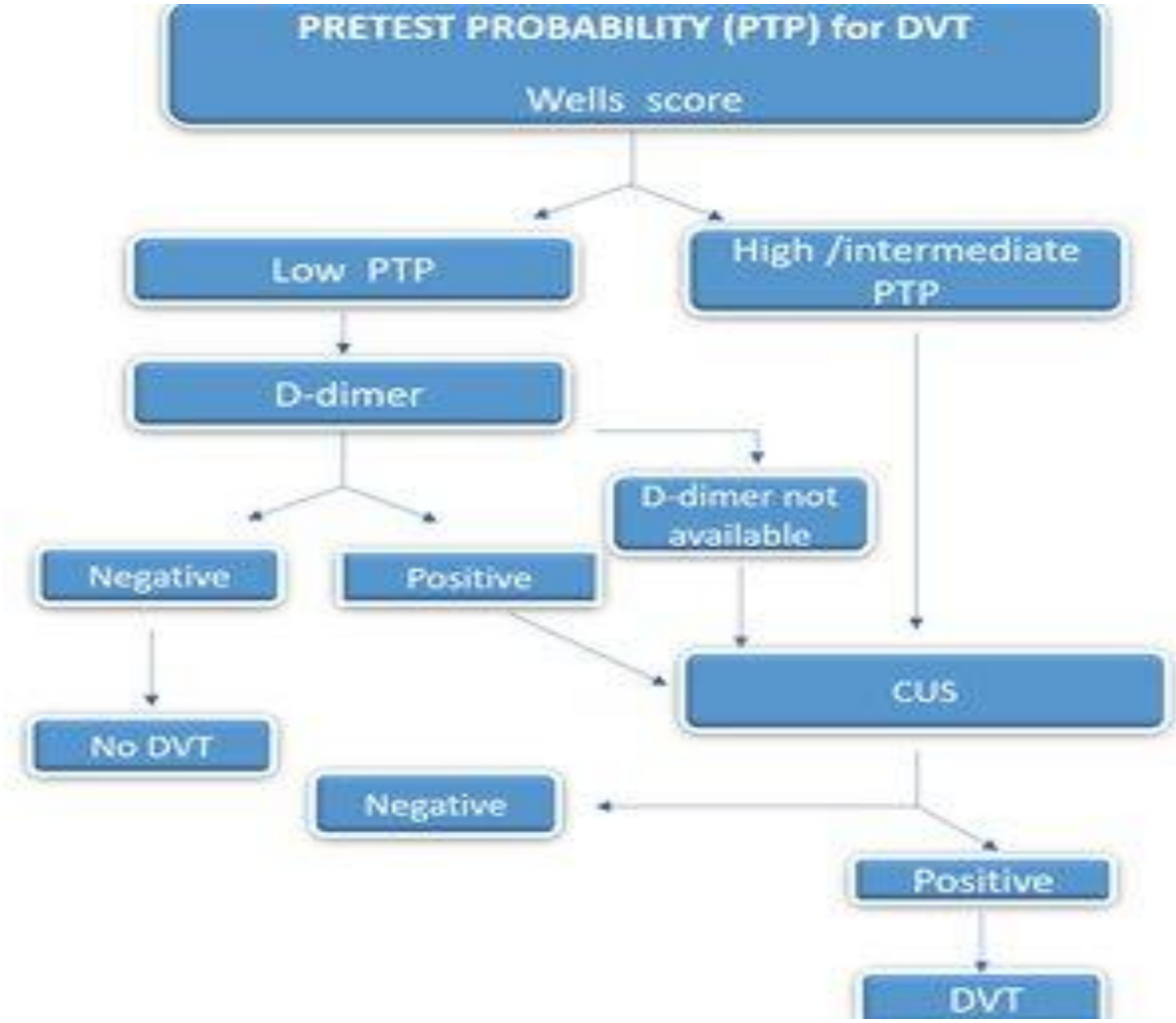
B**. Venography**

o Invasive and infrequently used.

C. **Impedance plethysmography**

D. **D-dimer testing**

o Has a very high sensitivity (95%), but low specificity (50%); can be used to rule out DVT when combined with Doppler and clinical suspicion. In patients with a low pretest probability of DVT, a negative D-dimer (< 500 ng/mL) rules out DVT. A positive D-dimer alone does not confirm DVT.


PRETEST PROBABILITY (PTP) for DVT
Wells score
Low PTP
High /intermediate PTP
D-dimer
D-dimer not available
Negative
Positive
CUS
No DVT
Negative
Positive
DVT

TREATMENT

1. Anticoagulation

a. Prevents further propagation of the thrombus.

b. Multiple options: Injectable LMWH (e.g., enoxaparin, dalteparin) (best choice for DVT/

PE associated with malignancy), DOACs (e.g., apixaban, rivaroxaban, edoxaban, dabigatran), and warfarin (requires IV heparin bridge). LMWH was the preferred

choice for DVT/PE treatment in patients with malignancy.

2. Thrombolytic therapy (streptokinase, urokinase, tissue plasminogen activator [tPA])

3. Inferior vena cava filter placement
Effective only in preventing PE, not DVT

COMPLICATIONS

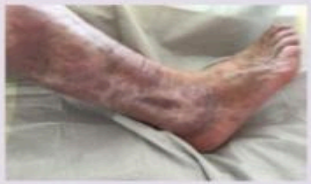
Pulmonary embolism:

Pulmonary emboli most commonly originate in the proximal deep veins of the lower extremities (e.g., iliac, femoral, or popliteal veins).

Post thrombotic syndrome

(chronic venous insufficiency)

A syndrome characterized by features of chronic venous insufficiency (e.g., pain, edema, venous dilation, ulceration, abnormal skin pigmentation) that develop following a deep vein thrombosis.

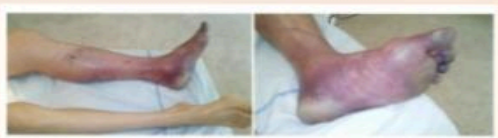


Phlegmasia cerulea dolens

(painful, blue, swollen leg)

Severe leg edema compromises arterial supply to the limb, resulting in impaired sensory and motor function.

Venous thrombectomy is indicated.



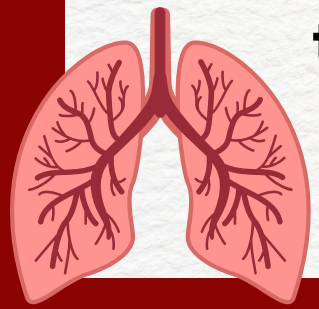
Venous gangrene

(rare complication)

The development of ischemic necrosis of a distal extremity despite palpable or doppler pulses, occurring as a complication of deep vein thrombosis.

Pulmonary Embolism

Luminal obstruction of one or more pulmonary arteries, typically due to blood thrombi from deep vein thrombosis (DVT) .



SOURCES OF EMBOLI

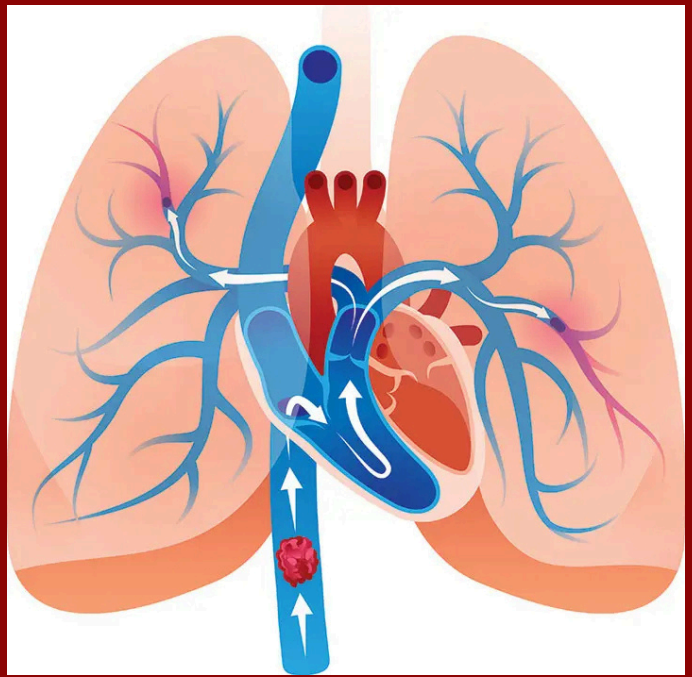
a. Lower extremity DVT - PE is the major complication of DVT. Most pulmonary emboli arise from thromboses in the deep veins of lower extremities above the knee (iliofemoral DVT).

o Pulmonary emboli can also arise from the deep veins of the pelvis.

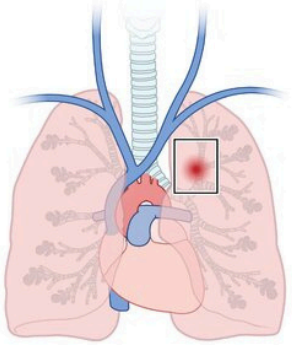
o Although calf vein thrombi have a low incidence of embolizing to the lungs, in many patients these thrombi progress into the proximal veins, increasing the incidence of PE

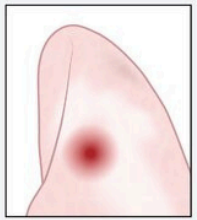
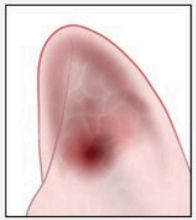
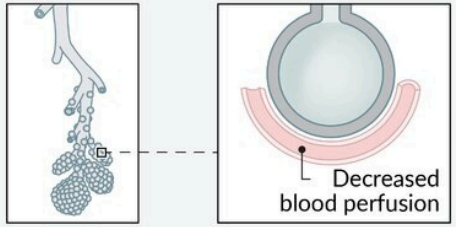
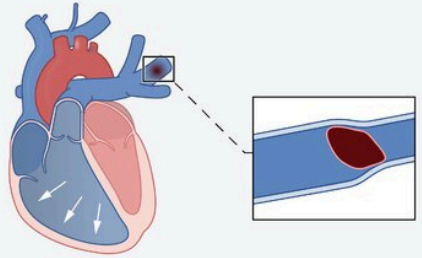
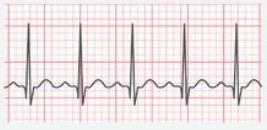
b. Upper extremity DVT, is a rare source of emboli (it may be seen .in IV drug abusers)

Pthophysiology of PE



- Emboli block a portion of pulmonary vasculature, leading to increased pulmonary vascular resistance, pulmonary artery pressure, and right ventricular pressure. If it is severe (large blockage), acute cor pulmonale may result.
- Blood flow **decreases** in some areas of the lung. Dead space is created in areas of the lung in which there is **ventilation but no perfusion**. The resulting **hypoxemia** and **hypercarbia** drive respiratory effort, which leads to **tachypnea.**
- If the size of the dead space is large (large PE), clinical signs are more overt (SOB, tachypnea).



	Pathophysiology	Presentation
Local infarction	  Ischemia → Pleura and lung inflammation	 Fever  Hemoptysis  Pleuritic chest pain
Impaired gas exchange	 Decreased blood perfusion ↑ V:Q → ↓ PaO_2 ↑ A-a gradient ↓ $PaCO_2$ Respiratory alkalosis	 Dyspnea  Cough  Wheezing
Cardiac compromise	 Right ventricular overload → ↑ Pulmonary artery pressure ↓ Cardiac output	 Tachycardia  Shock/syncope

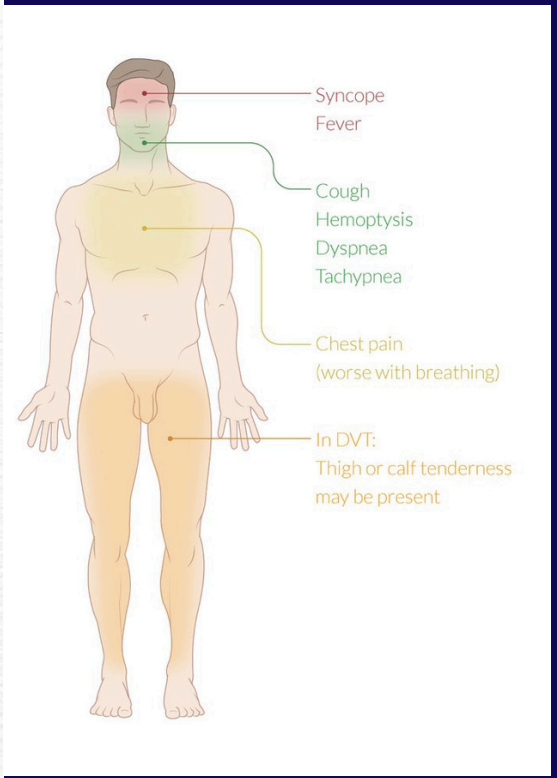
Clinical features

Common features of PE :

a. Acute onset of symptoms.
b. Dyspnea (> 75% of cases).
c. Tachycardia and tachypnea (up to 50% of cases).
d. Sudden pleuritic chest pain (~ 20% of cases).
e. Cough and hemoptysis.
f. Associated features of DVT: e.g., unilaterally painful leg swelling

Less common features of PE :

a. Decreased breath sounds.
b. Dullness to percussion.
c. Low-grade fever.
d. Rarely, upper abdominal pain



pretest probability of PE (wells criteria)

Wells criteria for PE [15][16]	
Criteria	Points
Clinical symptoms of DVT	3
PE more likely than other diagnoses	3
Previous PE/DVT	1.5
Tachycardia (heart rate > 100/min)	1.5
Surgery or immobilization in the past 4 weeks	1.5
Hemoptysis	1
Malignancy	1

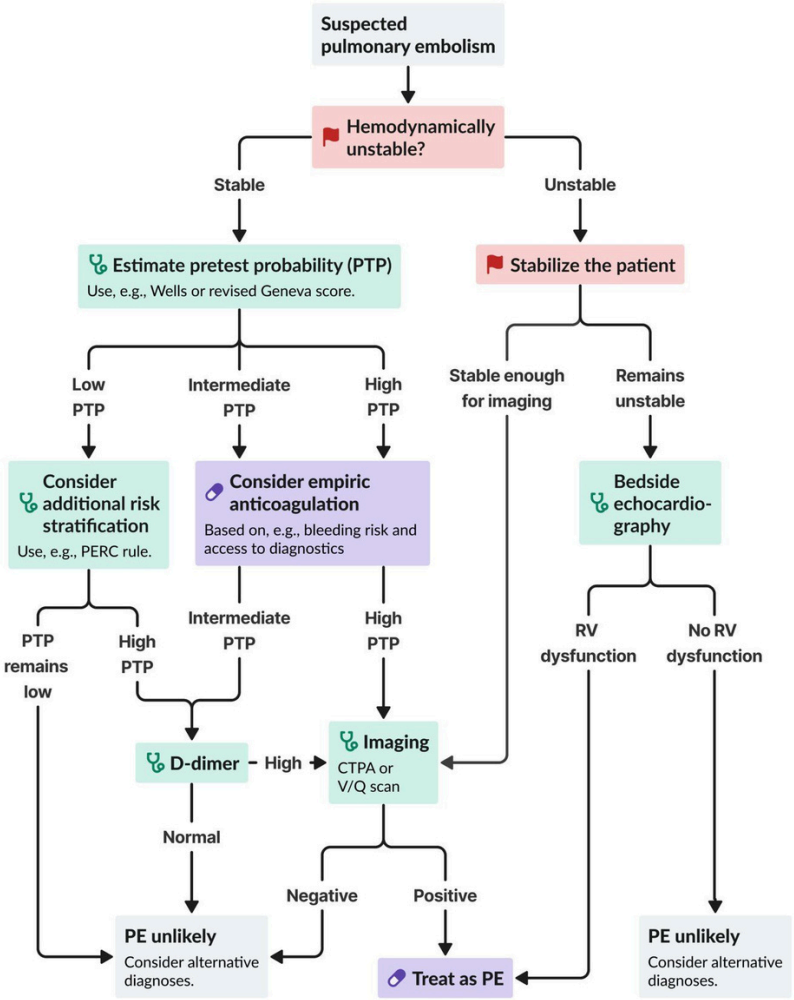
Original Wells score (clinical probability) [15]

- Total score 0–1: low probability of PE (6%)
- Total score 2–6: intermediate probability of PE (23%)
- Total score ≥ 7: high probability of PE (49%)

Modified Wells score (simplified clinical probability) [16]

- Total score ≤ 4: PE unlikely (8%)
- Total score > 4: PE likely (34%)

Diagnostic approach to pulmonary embolism (PE)



Diagnosis

1)arterial blood gas (ABG)

- Often normal .
- Common findings:

a. Alveolar-arterial gradient .

b. Hypoxemia (e.g., SaO_2, $PaO_2 < 80$ mm Hg).

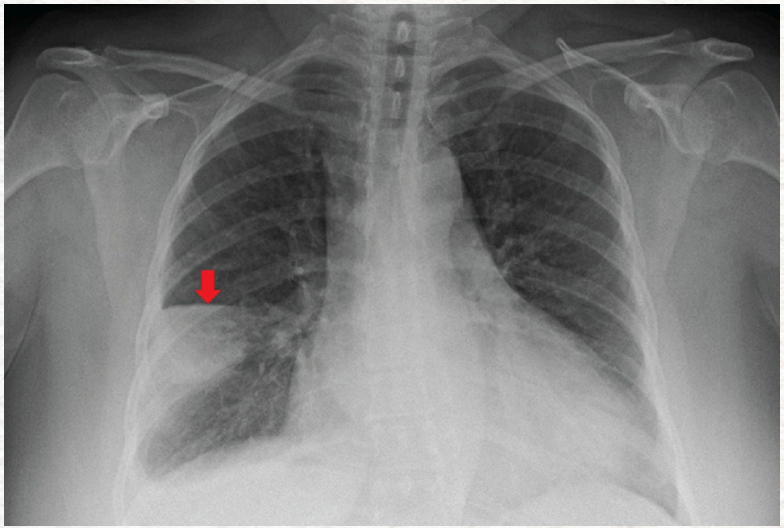
c. Respiratory alkalosis. “Normal oxygen saturation and/or normal PaO_2 do not rule out PE”

2) CHEST X-RAY

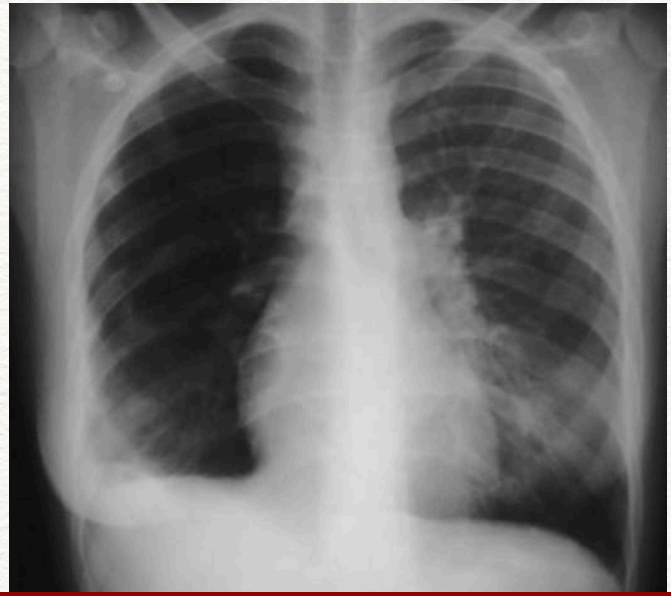
(usually normal)

- Atelectasis or pleural effusion may be present.
- The main usefulness is in excluding alternative diagnoses.
- Classic radiographic signs, such as Hampton hump or Westermark sign, are rarely present

Hampton hump:
a wedge shaped opacity in the peripheral lung
with its base at the thoracic wall
caused by pulmonary infarction and not specific for PE.



Westermark sign:
an area of lung parenchyma lucency caused by oligemia secondary to occlusion of blood flow.



3. Lower extremity venous ultrasound

- Indications a. Symptoms of deep venous thrombosis and pulmonary embolism b. Contraindications to CTPA (e.g., contrast allergy and renal insufficiency) c. Pregnancy . - Findings: e.g., no compressible deep vein

4. D-dimer

Interpretation a. High sensitivity and negative predictive value: A negative D-dimer test most likely rules out PE. b. Low specificity

5. CT pulmonary angiography (CTPA) (Gold standard)

CTPA is the preferred test for the diagnosis of acute PE.

6. CTA

- Has been found to have good sensitivity (>90%) and specificity. - Can visualize very small clots (as small as 2 mm); may miss clots in small subseg-mental vessels (far periphery). - The test of choice in most medical centers

7. Ventilation/perfusion scintigraphy (V/Q scan)- Indication: alternative to CTPA in patients with contraindications for iodinated IV contrast

Treatment

1. Supplemental oxygen

2. Acute anticoagulation therapy

with either unfractionated or low-molecular-weight heparin to prevent another PE. Anticoagulation prevents further clot formation, but does not lyse existing emboli or diminish thrombus size. Start immediately on the basis of clinical suspicion. Do not wait for studies to confirm PE if clinical suspicion is high

3. Oral anticoagulation with warfarin

a. Can start warfarin with heparin on day 1.

b. Target INR with warfarin for PE treatment is 2 to 3.

c. If using a novel oral anticoagulant (e.g., rivaroxaban or apixaban), concurrent treatment with heparin during initiation is not necessary as these medications are effective immediately.

d. Continue for 3 to 6 months or more, depending on risk factors.

4. Thrombolytic therapy-for example, streptokinase, tPA.

Speeds up the lysis of clots

Situations in which thrombolysis should be considered: - Patients with massive PE who are hemodynamically unstable (persistent hypotension). - Patients with evidence of right heart failure (thrombolysis can reverse this).

5. Inferior vena cava interruption (IVC filter placement)

Patients who have IVC filter placed are at higher risk of recurrent DVT (but lower risk of recurrent PE).

Indications include:

- Contraindication to anticoagulation in a patient with documented DVT or PE.

- A complication of current anticoagulation.

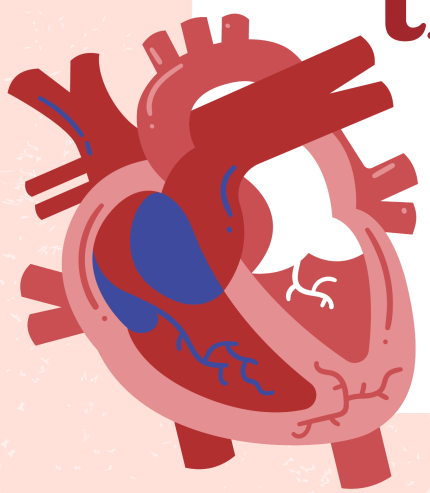
- Failure of adequate anticoagulation as reflected by recurrent DVT or PE.

- A patient with low pulmonary reserve who is at high risk for death from PE

6. Surgical thrombectomy

Consider in patients with hemodynamic compromise, a large, proximal thrombus, and who are poor candidates for fibrinolytics

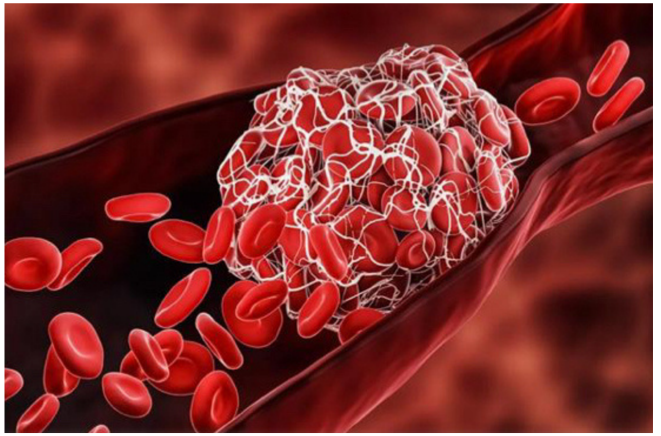
Arterial thromboembolism



Arterial thromboembolism is a blood clot that travels and blocks blood flow in distant arteries.

Arterial embolism results when a mass of tissue or a foreign substance travels through the vascular tree, ultimately lodging in a distal artery where it obstructs blood flow. This obstruction leads to ischemia, organ dysfunction and potential infarction.

Manifestations of this complex disease include medical and surgical emergencies such as stroke, acute limb ischemia, mesenteric ischemia and renal failure.



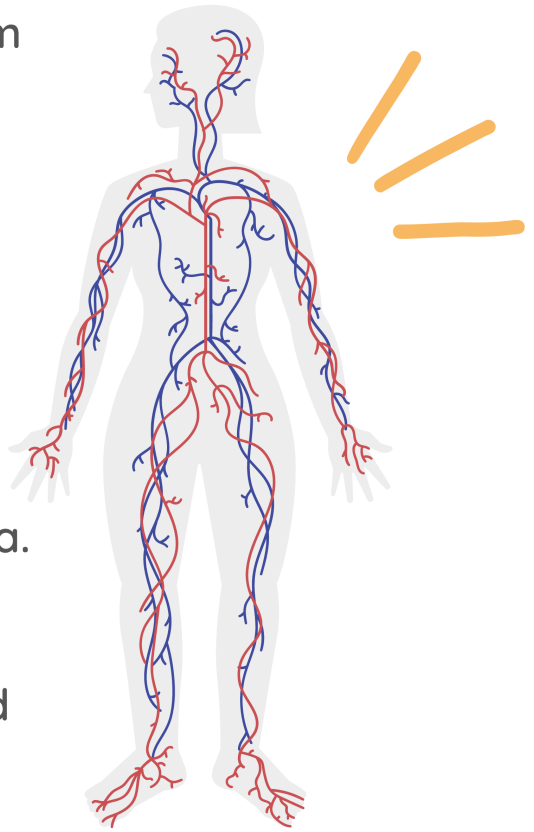
Key points:

- The thrombus obstructs the artery's lumen, causing narrowing or complete blockage.
- Reduced blood flow leads to decreased oxygen and nutrient delivery to downstream tissues or organs.
- In severe cases, this can result in tissue ischemia and necrosis (cell death).

Most arterial emboli originate in the left heart, forming due to structural or functional abnormalities. Some emboli form in the arterial tree itself.

Key Points on Embolism Pathways:

1. Proximal Clots (e.g., from the heart or aortic arch):
   • Can travel to any arterial branch in the body.
2. Distal Clots (e.g., from carotid or infra-renal aorta):
   • Carotid plaques: Likely to cause strokes or transient ischemic attacks (TIAs).
   • Infra-renal plaques: Commonly cause lower limb ischemia.
3. Retrograde Embolization:
   • Rarely, during slow heart rates and late diastolic flow reversal, clots in the descending aorta can move backward and cause strokes.



Factors Affecting Embolus Destination

• Blood Flow: Arterial beds with higher blood flow are more likely targets.
• Anatomy: The structure of arterial branches influences where emboli lodge.
• Size of Embolus:
• Larger emboli: Often block areas with narrowing, such as bifurcations or stenotic arteries.
• Smaller emboli: Travel further, blocking smaller arterioles.

Causes

The likelihood of each cause contributing to an arterial embolism can vary depending on population demographics and clinical context

1. Atrial fibrillation – A common cause due to irregular heartbeats that increase the risk of clot formation in the atria.
2. Atherosclerosis – The most frequent underlying cause of emboli formation, particularly in patients with coronary artery or peripheral artery disease.
3. Hypertension (high blood pressure) – A major contributing factor to atherosclerosis and arterial damage.
4. Heart disease – Conditions such as myocardial infarction or heart failure can lead to thrombus formation.
5. Tobacco use disorder (smoking) – A significant modifiable risk factor that exacerbates atherosclerosis and vascular damage.
6. Post-surgical complications – Especially following cardiac or vascular procedures.
7. Arterial trauma or injury – Less common, but direct damage to the arteries can lead to thrombus or embolus formation.

Singe and symptoms

You may notice these symptoms in an arm or leg after an embolism has formed:

Sudden pain in the affected limb.
Pale or bluish skin over the area.
Coldness in the limb.
Weakness or numbness in the limb.
Reduced or absent pulses below the blockage.
Difficulty moving the affected limb.

Symptoms that may occur if an embolism is not treated or worsens

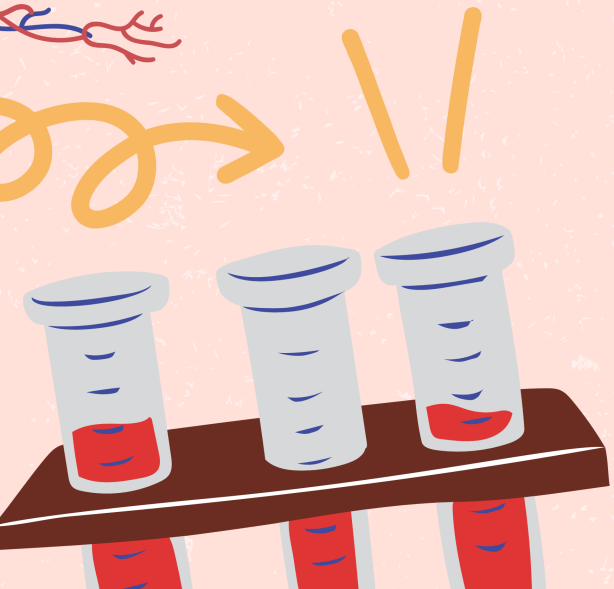
Complication

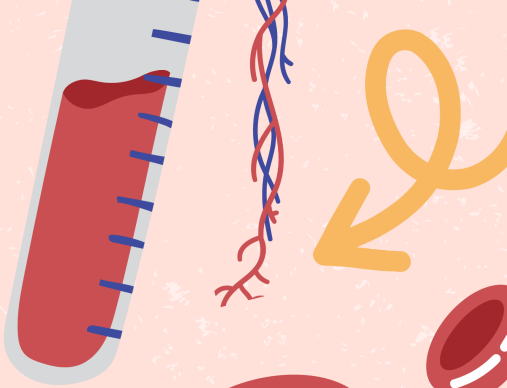
The complications of arterial emboli vary based on their size and location:

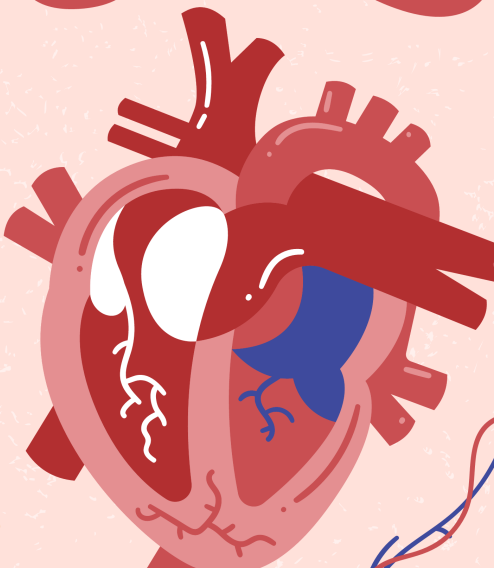
- A blockage in the coronary arteries can cause a **heart attack.**
- If the embolism occurs in the brain, it can lead to a stroke.
- Reduced blood flow to a limb can result in gangrene.
- A clot in the renal arteries can cause kidney failure, which may be temporary or permanent.
- In severe cases, an embolism can lead to sudden death.

Diagnosis

- Angiogram: Identifies the site of arterial blockage.
- ECG: Detects myocardial infarction (MI) or atrial fibrillation (AFib).
- Echocardiogram: Evaluates the heart as a possible source of emboli.
- Doppler ultrasound: Assesses blood flow and blockages in arteries.
- MRI: Pinpoints the location of blood clots within the body.
-







Treatment

Medication

- Thrombolytics: Dissolve existing clots and restore blood flow.
- Anticoagulants: Prevent further clot formation or growth (e.g., warfarin, heparin, apixaban).

Surgery

- Angioplasty: Opens blocked arteries with a balloon catheter, sometimes using a stent.
- Embolectomy: Removes the clot directly.
- Arterial bypass: Creates an alternative route for blood flow around the blockage.