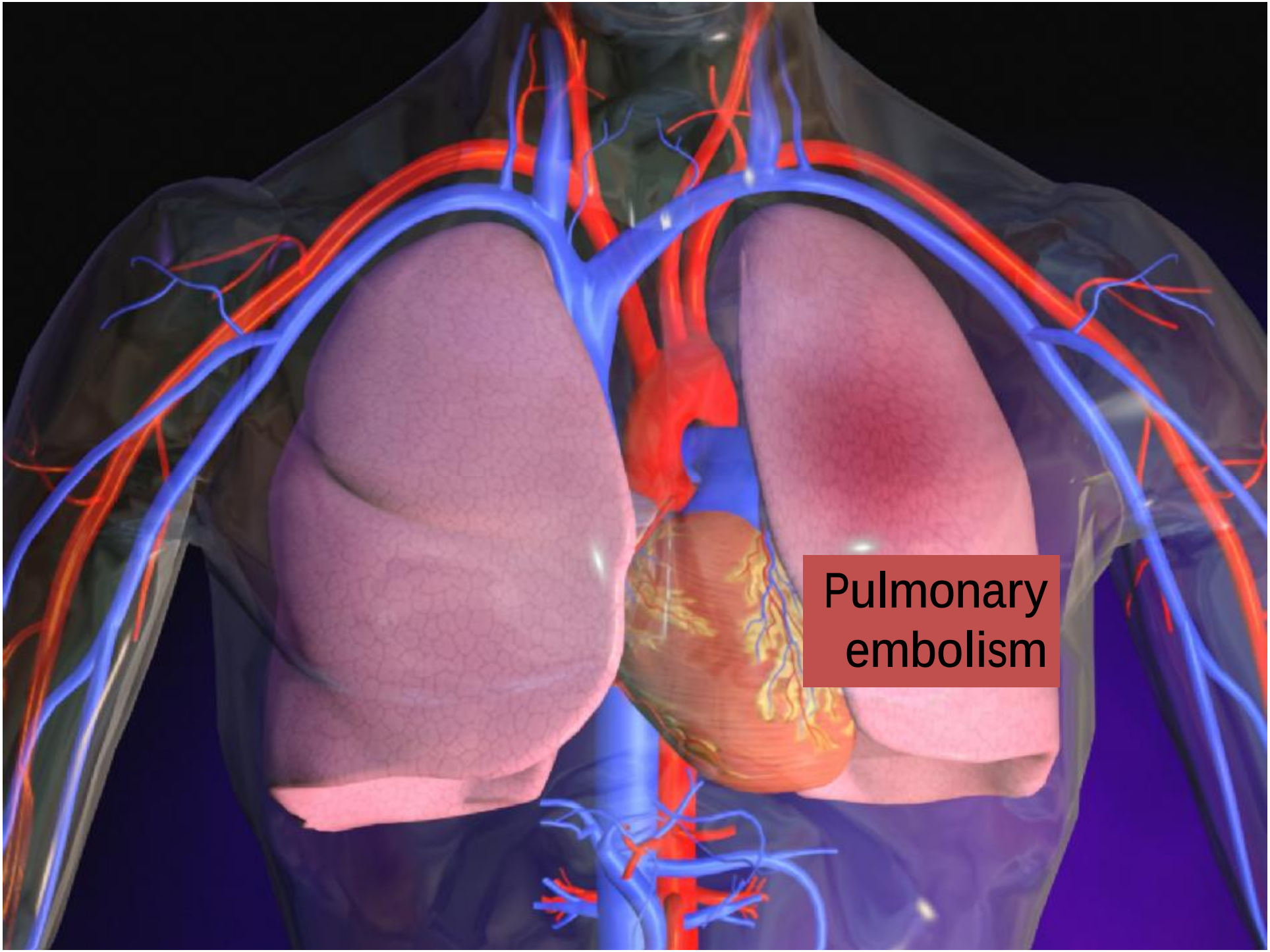
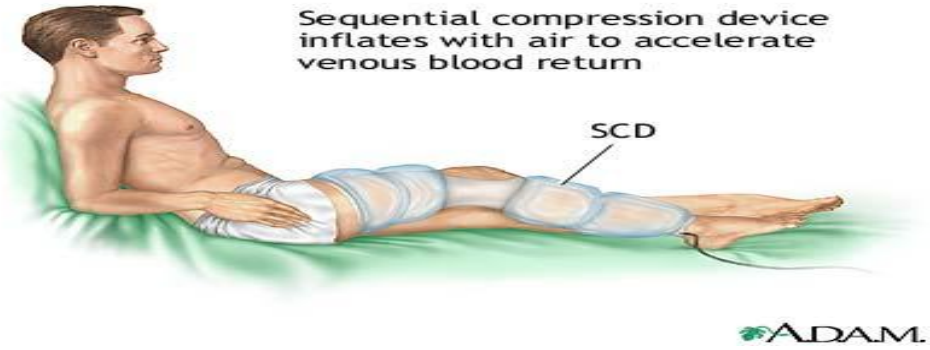


Pulmonary embolism

- () PE refers to obstruction of the pulmonary artery or one of its branches by material (eg, thrombus, amniotic, placenta, septic, tumor, air, or fat) that originated elsewhere in the body.
- () Acute pulmonary embolism (PE) is a common and often fatal disease. Mortality can be reduced by prompt diagnosis and therapy. Unfortunately, the clinical presentation of PE is variable and nonspecific, making accurate diagnosis difficult.
- () 75% of PE derives from DVT in the lower limb & 60% of patients with DVT will have evidence of PE on scanning even in the absence of symptoms.
- () PE occurs in 1% of patients admitted to hospital & is responsible for about 5% of all hospital death.

Risk factors



PE is a common complication

of deep vein thrombosis (DVT). As a result, most factors that favor the development of DVT probably increase the risk for PE.

These include:

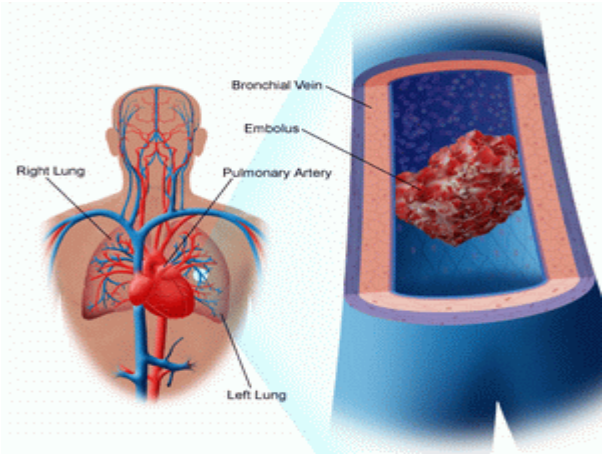
- *** immobilization, surgery within the last three months, stroke, paresis, paralysis, history of venous thromboembolism, malignancy, central venous instrumentation within the last three months, and chronic heart disease. Additional risk factors identified in women may include obesity (BMI ≥ 29 kg/m2), heavy cigarette smoking (>25 cigarettes per day), and hypertension.

Classification & Clinical features:

PE can be classified as acute or chronic.

Patients with acute PE typically develop symptoms and signs immediately after obstruction of pulmonary vessels.

- In contrast, patients with chronic PE tend to develop slowly progressive dyspnea over a period of years due to pulmonary hypertension.
- Acute PE can be further classified as massive or submissive.



1- Acute massive PE:

- Is due to acute obstruction of more than 50% of either the main or the proximal pulmonary artery, leading to an acute reduction of COP & RV dilatation.
- Symptoms: sudden collapse, syncope, central chest pain, apprehension & severe dyspnea.
- Signs: hypotension, tachycardia, tachypnea with central cyanosis, elevated JVP, RV gallop wide-splitting of S2 & low COP.

2- Acute sub-massive PE:

- It due to occlusion of segmental pulmonary artery leading to infarction &/ effusion,
- So called pulmonary infarction syndrome.
- Symptoms: SOB, pleurisy with peripheral chest pain & haemoptysis.
- Signs: tachycardia, signs of pleurisy or effusion, raised hemi diaphragm, crackles & low-grade fever.

3- Chronic PE:

- Chronic venous thromboembolism leading to thromboembolic pulmonary hypertension due to chronic occlusion of pulmonary microvasculature with RV failure.
- Symptoms: typically present with a history of exertional SOB, syncope & chest pain developing over months or years.
- Signs: may be minimal early in disease, later on RV heave, loud split P2, elevated JVP.

Diagnosis:

A- Laboratory: •

1- Routine laboratory findings are nonspecific and • include leukocytosis, an increase in the erythrocyte sedimentation rate (ESR), and an elevated serum LDH or AST (SGOT) with a normal serum bilirubin.

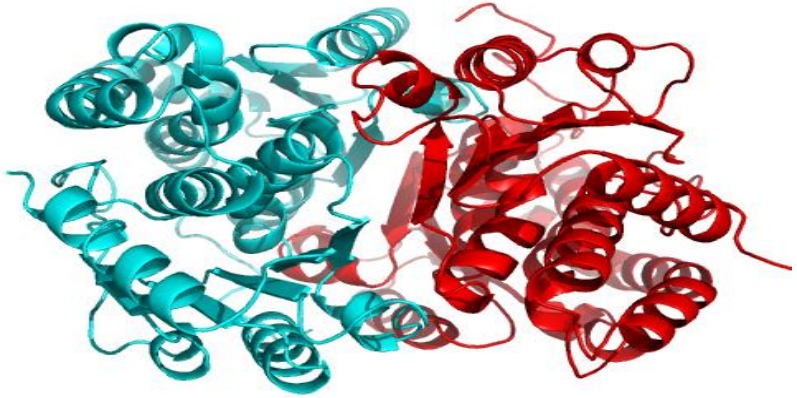
2- Arterial blood gas — Arterial blood gas (ABG) • measurements and pulse oximetry have a limited role in diagnosing PE.

() acute massive PE== hypoxia, hypocapnea & • respiratory alkalosis followed by M acidosis.

() acute sub massive==hypocapnea. •

() chronic PE== exertional hypoxia •

- 3- BNP — brain natruitic peptide (BNP) levels are typically greater in patients with PE compared to patients without PE.
- 4- Troponin — Serum troponin I and troponin T are elevated in 30 to 50 % of patients with pulmonary embolism.
- The presumed mechanism is acute right heart overload.
- The troponin elevations usually resolve within 40 hours with pulmonary embolism in contrast to the more prolonged elevation with acute myocardial injury.



- 5- D-dimers: is a specific degradation product released into the circulation when cross-linked fibrin undergoes endogenous fibrinolysis.
- In patients with suspected PE a low plasma D-dimer (<500mg/ml) has a 95% predictive power for excluding PE & hence the D-dimer can be used as an initial screening investigation.
- A positive D-dimer, however, dose not positively diagnoses PE since raised level may be seen in a whole range of inflammatory conditions as pneumonia.

B- ECG:
() acute massive PE:
- S1, Q3, T3
- T wave inversion V1-4
- RBBB

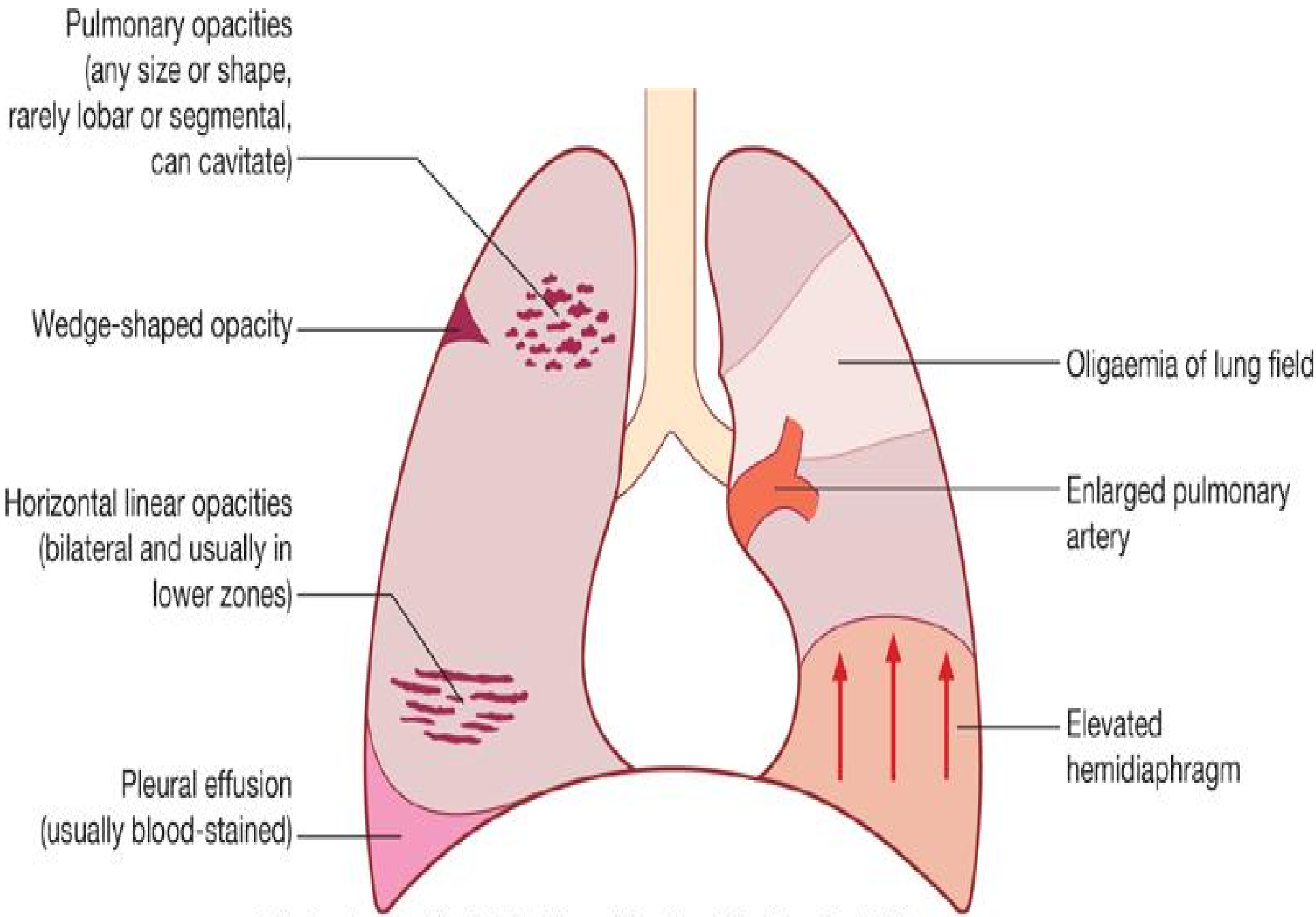
() acute sub massive:
- Sinus tachycardia

() chronic PE:
- RV hypertrophy &/ strain.

C- Chest X-ray:
() acute massive PE:
- Oligamic lung fields.
- Increase hilar shadows.

() acute sub massive:
- Pulmonary opacities (lobar or segmental &/or wedge-shaped)
- Pleural effusion
- Elevated hemidiaphragm

() chronic PE:
- Enlarged pul artery trunk
- Enlarged heart
- Prominent RV.



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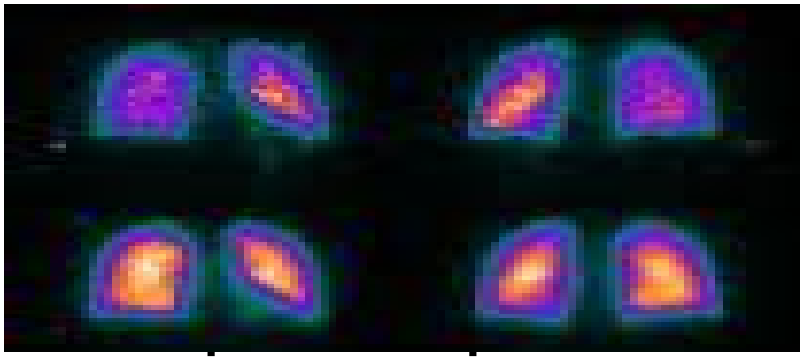


- D- V/Q scan: ventilation-perfusion scanning remains useful in patients with no previous lung disease & should be carried out within 24 hr of presentation since some scans revert to normal very quickly & 50% do so by 1 week.
- () acute massive PE: major area of decrease perfusion.
- () acute sub massive: perfusion defect(s) not matched on the ventilation scan.
- () chronic PE: may be normal.
- E-Ultrasound: colure Doppler Lower extremity venous ultrasound is sometimes performed in the diagnostic evaluation of PE.

F- Spiral CT angiography: has good sensitivity & specificity for central or segmental thrombi.

G- Echocardiography:

Can be used to diagnose a major central PE & exclude other diagnosis as MI:

Only 30 to 40 percent of patients with PE have echocardiographic abnormalities suggestive of acute PE including:

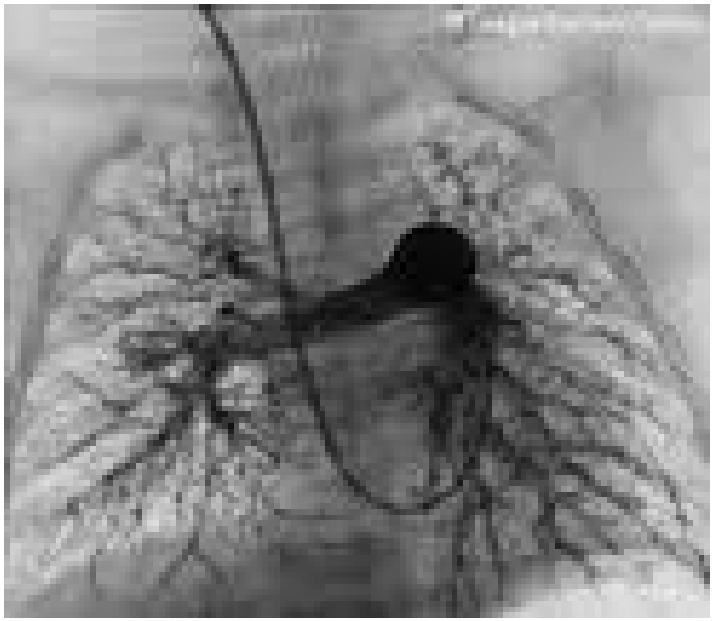
() Increased right ventricular (RV) size

() Decreased RV function

() Tricuspid regurgitation.

H- Pulmonary angiography:

Definitive diagnosis in acute PE.



The modified Wells Criteria include the following:

- () Clinical symptoms of DVT (3 points)
- () Other diagnosis less likely than PE (3 points)
- () Heart rate >100 (1.5 points)
- () Immobilization or surgery in previous four weeks (1.5 points)
- () Previous DVT/PE (1.5 points)
- () Hemoptysis (1 point)
- () Malignancy (1 point)
- SO == PE is unlikely (score <4) or likely (score >4).

Management:

A- General measures:

1- opiates: relieve pain & distress.

2- O2: to restore arterial O2 saturation to over 90%.

3- External cardiac massage may be successful by breaking up a large central embolus.

4- Diuretics & vasodilators should be avoided in acute setting.

B- anticoagulation:

1- Heparin should be given to all patients with a high clinical suspicion of PE.

2- LMW heparin SC as effective as IV unfractionated heparin.

3- Heparin reduces mortality by reducing further emboli.

4- Heparin should be administered for at least 5 days & then continue on oral warfarin. & the heparin should not be discontinued until the INR is over 2.

5- The duration of warfarin therapy is:

6 weeks----for patients with reversible cause for DVT as hip surgery.

3 months--- for patients with no identified cause.

For life-----in patients with prothrombic risk or a history of previous emboli.

C- Thrombolytic therapy: streptokinase or alteplase can be used.

A dose of 60 mg IV administered over 15 minutes is sufficient, used within 24 hr.

Indicated in:

1- Patients with an acute massive PE.

2- RV dysfunction on Echo.

3- Hypotension.

D- Caval filters:

1- Recurrent PE.

2- When anticoagulation is contraindicated.