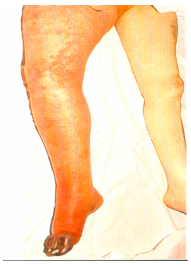
Team Medicine

Pulmonary Embolism

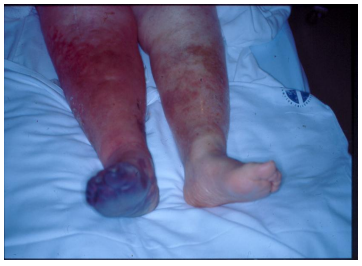
Writer: Saud Alawad
Reviser: Sama Al Ohali
Leader: Alanood Asiri

■ Important notes ■ doctor notes ■ step up to medicine

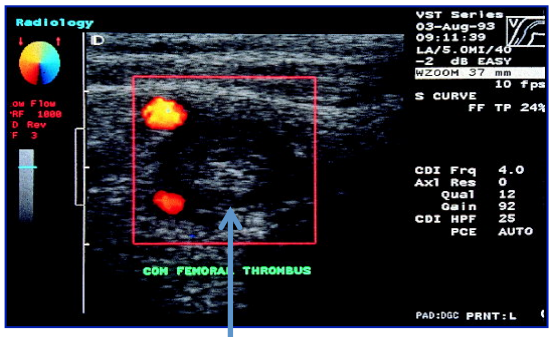
431 Teams
medical students



Phlegmasia cerulea dolens

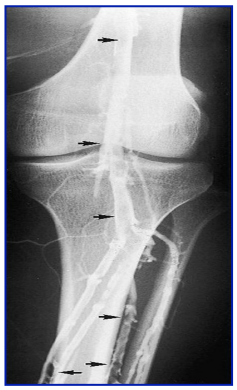


Venous gangrene

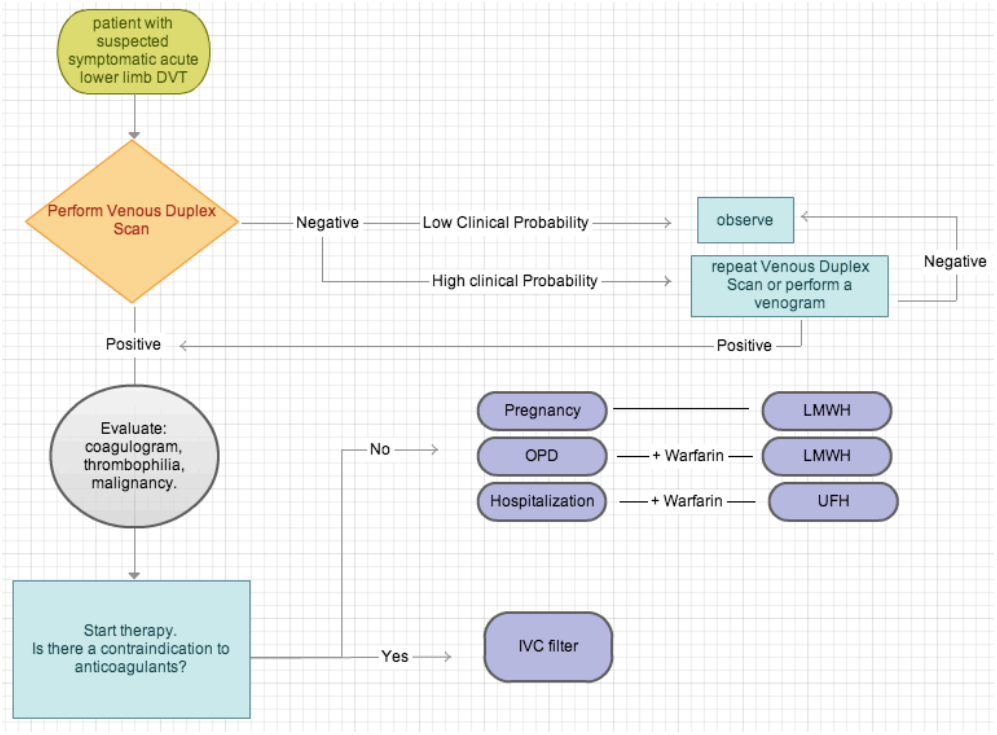


Color duplex scan of DVT

White shadow represents a clot surrounded by the vein.



Venogram shows DVT



Diagnosis and management of a patient with suspected acute lower limb DVT

Thrombophilia = prothrombotic state = hypercoagulability: an abnormality of blood coagulation that increases the risk of thrombosis
Examples:
- Factor V leiden
- Protein C/S deficiency
- Antithrombin III deficiency

Thrombophilia screening: Factor V leiden, Protein C/S deficiency Antithrombin III deficiency

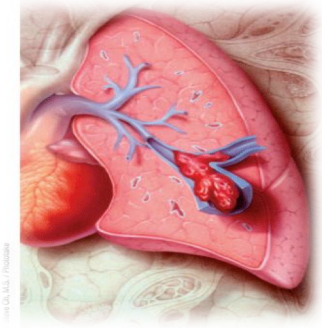
- Idiopathic DVT < 50 years must investigate about thrombophilia.
- Family history of DVT
- Thrombosis in an unusual site
- Recurrent DVT

Recommendation for duration of warfarin:

- 3-6 months first DVT with reversible risk factors
- At least 6 months for first idiopathic DVT
- 12 months to lifelong for recurrent DVT or first DVT with irreversible risk factors malignancy or thrombophilic state.

Catheter directed-thrombolysis:

- Consider in: Acute< 10 days iliofemoral DVT.
- Long-term benefit in preventing post-phebitic syndrome is unknown.



Pulmonary Embolism :

- 50,000 individuals die from PE each year in USA
- The incidence of PE in USA is 500,000 per year
- It is a medical emergency.
- If you suspect it you have to investigate.
- If we have clear risk factor, we don't need to investigate. e.g(if a patient has symptoms of PE and a DVT is found, one can make the diagnosis of PE without further testing.

Step up medicine pg105: Consider PE and deep venous thrombosis (DVT) as a continuum of one clinical entity (venous thromboembolism)—diagnosing either PE or DVT is an indication for treatment.

PE + DVT have the same risk factors and treatment

Most of sudden death maybe because of massive PE
The incidence of PE in hospitals is decreased because of prophylaxis .

Step up medicine pg106:
Signs and symptoms are not a reliable indicator of the presence of PE. This often leads to confusion and delay in diagnosis and treatment. If, however, a patient has symptoms of a PE and a DVt is found, one can make the diagnosis of PE without fur- ther testing.

1. **Step up medicine pg106:** Course and prognosis

Most often, PE is clinically silent. **Recurrences are common,** which can lead to development of chronic pulmonary HTN and chronic cor pulmonale.

When PE is undiagnosed, mortality approaches 30%. A significant number of cases are undiagnosed (as many as 50%).

When PE is diagnosed, mortality is 10% in the first 60 minutes. **Of those who survive the initial event, approximately 30% of patients will die of a recurrent PE if left untreated. Most deaths are due to recurrent PE within the first few hours of the initial PE. Treatment with anticoagulants decreases the mortality to 2% to 8%.**

Risk factor for venous thrombosis :

- Stasis . like heart failure
- Injury to venous intima
- Alterations in the coagulation-fibrinolytic system
- Sometimes when a women shift to another contraceptive pills.

Source of emboli :

- Deep venous thrombosis (>95%). Most commonly
- Other veins:
  - Renal
  - Uterine
  - Right cardiac chambers

Risk factors for DVT: (important) → the same as for PE

- General anesthesia
- Lower limb or pelvic injury or surgery
- Congestive heart failure
- Prolonged immobility
- Pregnancy
- Postpartum
- Oral contraceptive pills
- Malignancy . consider it when young patient with recurrent DVT .
- Obesity
- Advanced age
- Coagulation problems
- Renal disease (like nephrotic syndrome) → producing high amounts of proteins including coagulation factors

Clinical features: are not specific

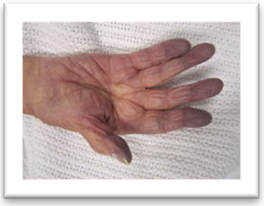
- Sudden onset dyspnea
- Pleuritic chest pain
- Hemoptysis . not common
- Clinical clues cannot make the diagnosis of PE; their main value lies in suggesting the diagnosi

Signs or symptoms observed in patients with thromboembolism			
		Study	
		Stein et al., % (n= 117)	Anderson et al., % (n= 131)
Pulmonary embolism	Dyspnea	73	77
	Tachypnea	70	70
	Chest pain	66	55
	Cough	37	—
	Tachycardia	30	43
	Cyanosis	1	18
	Hemoptysis	13	13
	Wheezing	9	—
	Hypotension	—	10
	Syncope	—	10
	Elevated jugular venous pulse	—	8
	Temperature >38.5°C	7	—
	S-3 gallop	3	5
	Pleural friction rub	3	2
Deep vein thrombosis	Swelling	28	88*
	Pain	26	56
	Tenderness	—	55
	Warmth	—	42
	Redness	—	34
	Homan's sign	4	13
	Palpable cord	—	6

The 2 studies show different percentages of signs and symptoms of PE which supports the above statement (Signs and symptoms are not a reliable indicator of the presence of PE.)

Massive Pulmonary Embolism:

- It is a catastrophic entity which often results in acute right ventricular failure and death
- Frequently undiscovered until autopsy
- Fatal PE typically leads to death within one to two hours of the event
- You could see cyanosis. (Cyanosis is caused by an increase in the deoxygenated haemoglobin level to above 5 g/dL.)
- It can result in obstructive shock.



Pathophysiology:

- Massive PE causes an increase in PVR → right ventricular outflow obstruction → decrease left ventricular preload → Decrease CO
- In patients without cardiopulmonary disease, occlusion of 25-30 % of the vascular bed → increase in Pulmonary artery pressure (PAP)
- Hypoxemia ensues → stimulating vasoconstriction → increase in PAP
- More than 50% of the vascular bed has to be occluded before PAP becomes substantially elevated
- When obstruction approaches 75%, the RV must generate systolic pressure in excess of 50mmHg to preserve pulmonary circulation
- The normal RV is unable to accomplish this acutely and eventually fails

Investigation:

- CXR (The main usefulness is in excluding alternative diagnoses.)
- ABG:
- V/Q
- Spiral CT it is the diagnostic test nowadays. (Most commonly used)
- Echo
- Angio (the gold standard)
- Fibrin Split Products/D-dimer
- ECG: S1 Q3 T3 Pattern – Right ventricular Strian – Right Bundle Branch Block – T wave inversion (suggestive but not diagnostic)

The gold standard test for diagnosis is pulmonary angiography. However, it is invasive. So the diagnostic test of choice is Spiral CT. If contraindicated eX: renal impairment, do V/Q

ABG:
PaO2 and PaCO2 are low (the latter due to hyperventilation) and pH is high; thus, there is typically a respiratory alkalosis

Diagnosis:

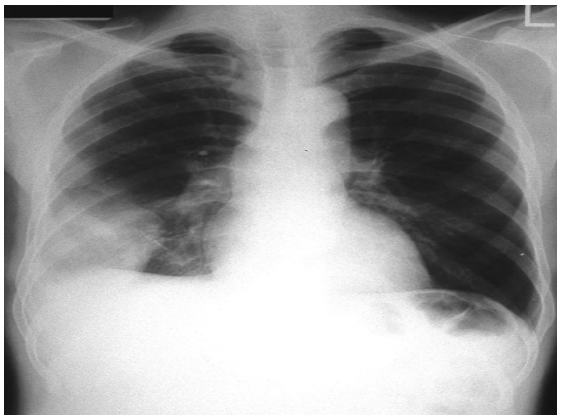
The diagnosis of massive PE should be explored whenever oxygenation or hemodynamic parameters are severely compromised without explanation

- CXR : you may see wedge shape opacity .
- ABG:

Significant hypoxemia is almost uniformly present when there is a hemodynamically significant PE

- V/Q
- Spiral CT it is better than chest CT and MRI
- Echo
- Angio is not usually done nowadays.
- D-Dimer: if + doesn't help , if – make diagnosis less likely .
- BNP
- Troponin

Chest radiograph showing pulmonary infarct in right lower lobe. This patient had low-grade fever, hemoptysis, and pleuritic chest pain. The ventilation-perfusion scan was read as high probability for pulmonary embolism. A pleural-based density in the lower lobe with the convexity directed toward the hilum signifies pulmonary infarction. This sign is also known as ?Hampton's hump."

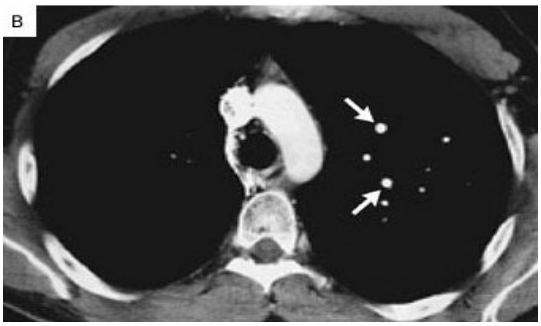


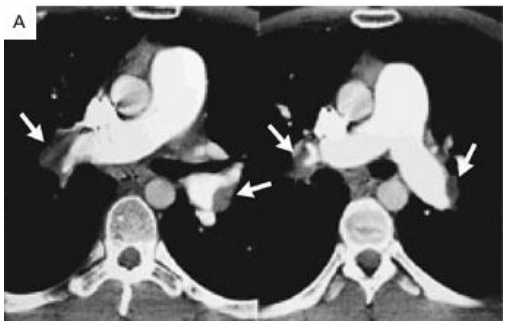
Usually there is no infarction in PE because lung has dual circulation. "bronchial and pulmonary:
You could see infarction in HF (rare)
Low blood supply → low surfactant → Atelactasis

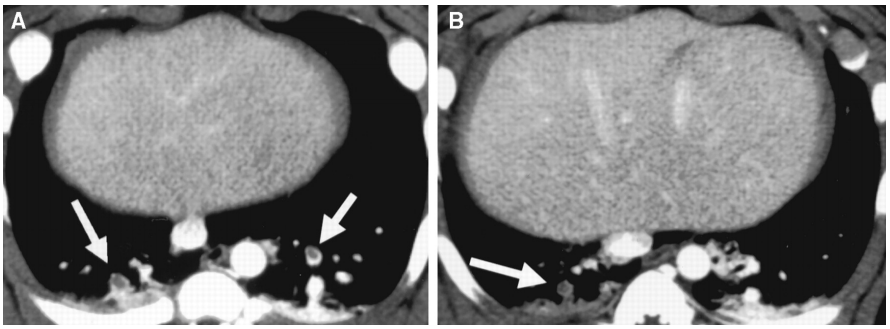
Chest radiographic findings in patients with pulmonary embolism

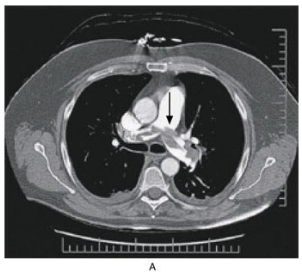
	COPD, % (n= 21)	No prior cardiopulmonary disease, % (n= 117)
Atelectasis or pulmonary parenchymal abnormality	76	68
Pleural effusion	52	48
Pleural-based opacity	33	35
Elevated diaphragm	14	24
Decreased pulmonary vascularity	38	21
Prominent central pulmonary artery	29	15
Cardiomegaly	19	12
Westermark's sign*	5	7
Pulmonary edema	14	4

Spiral (helical) CT :









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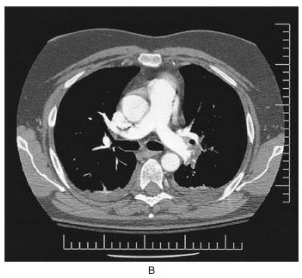

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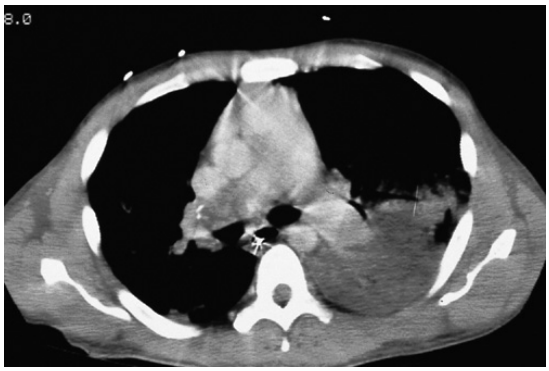


Tomographic scan showing infarcted left lung, large clot in right main pulmonary artery

The use of ventilation perfusion scan in diagnosing pulmonary embolism

High probability

=2 large segmental (>75% of a segment) perfusion defects without corresponding ventilation or radiographic abnormalities or substantially larger than matching ventilation or radiologic abnormalities

OR

=2 moderate segmental (>25% and <75% of a segment) perfusion defects without matching ventilation or chest radiographic abnormalities plus one large unmatched segmental defect

OR

=4 moderate segmental perfusion defects without matching ventilation or chest radiologic abnormalities

The use of ventilation perfusion scan in diagnosing pulmonary embolism

Intermediate probability

Scans that do not fall into normal, very low, low, or high probability categories

The use of ventilation perfusion scan in diagnosing pulmonary embolism

Very low probability

Three or fewer small segmental perfusion defects with a normal chest radiograph

Normal

No perfusion defects present

The use of ventilation perfusion scan in diagnosing pulmonary embolism

Low probability

Nonsegmental perfusion defects

OR

Single moderate mismatched segmental perfusion defect with normal chest radiograph

OR

Any perfusion defect with a substantially larger abnormality on chest radiograph

OR

Large or moderate segmental perfusion defects involving no more than four segments in one lung and no more than three segments in one lung region with matching or larger ventilation/radiographic abnormalities

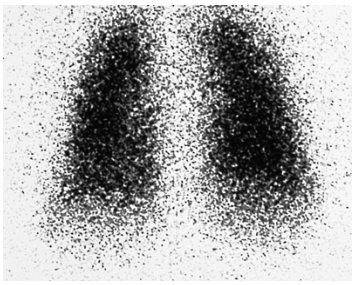
OR

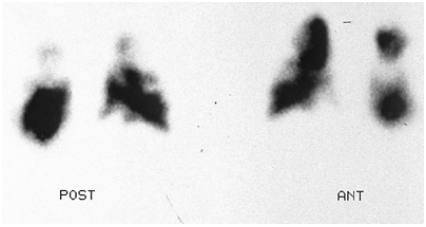
More than three small segmental perfusion defects (<25% of a segment) with a normal chest radiograph

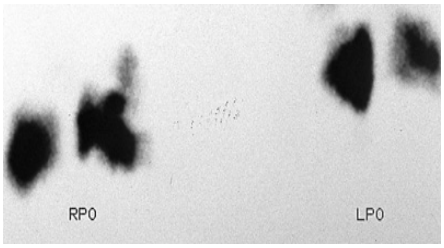
Explanation:

The criteria used for interpreting ventilation-perfusion scans are listed in the schedule above. In patients suspected to have pulmonary embolism, this study is ordered routinely. The perfusion scan is usually performed first. Macroaggregates of albumin labeled with technetium-99m are injected, and images are obtained in anterior, posterior, and right and left lateral and oblique views. If the perfusion scan is normal, there is no need to perform a ventilation scan. Defects in perfusion are assessed and quantified. The ventilation scan is performed by inhalation of a radioactive gas, usually 133 Xe is mixed with air. The patient breathes this radioactive mixture until a state of equilibrium has been reached between the spirometer and lungs, then the patientbreathes room air. Images taken in this wash-out phase are useful in detecting ventilation abnormalities.

High-probability ventilation-perfusion scan :





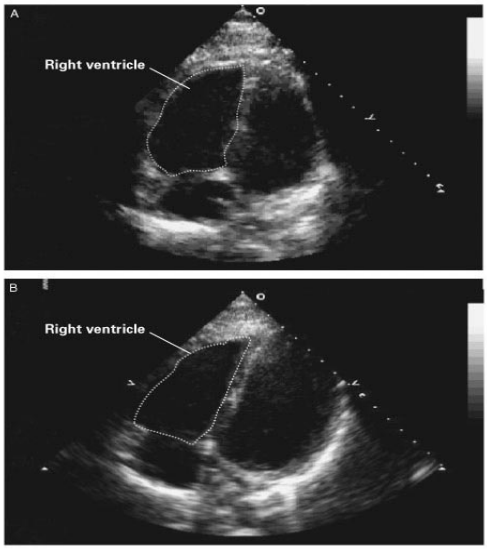


Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) results

Prospective investigation of pulmonary embolism diagnosis results

Scan category	PE present	PE absent	PE uncertain	No angiogram	Total
High probability	102	14	1	7	124
Intermediate probability	105	217	9	33	364
Low probability	39	199	12	62	312
Near normal or normal	5	50	2	74	131
Total	251	480	24	176	931

Explanation :
This prospective study was designed to study the accuracy of ventilation-perfusion (õ/õ) scan in the diagnosis of pulmonary embolism. Results of õ/õ scan were compared with pulmonary angiography, which was used as a gold standard. From the results it is obvious that more than two thirds of patients have scans of low or intermediate probability that are nondiagnostic. Although a high-probability scan usually indicates pulmonary embolism, only a minority of patients with pulmonary embolism have a high-probability scan. Near-normal lung scans make the diagnosis of pulmonary embolism very unlikely.

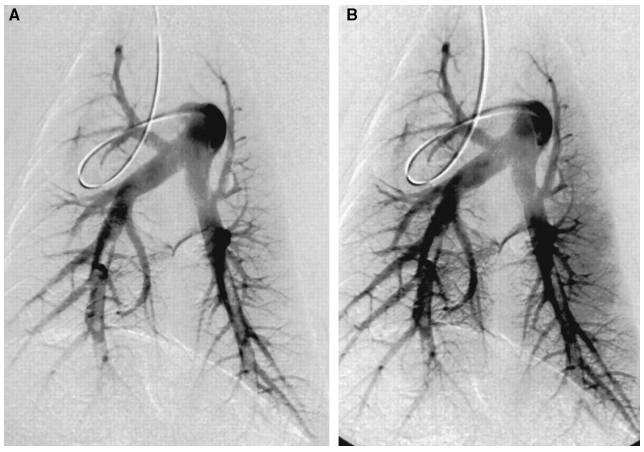


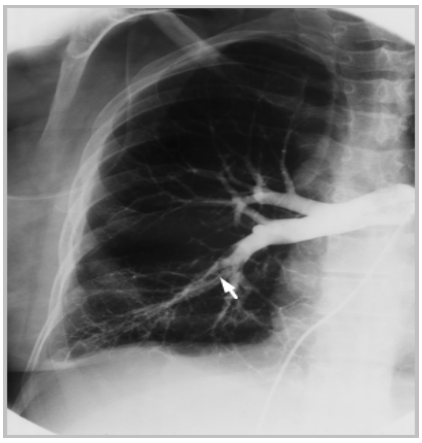
before

after

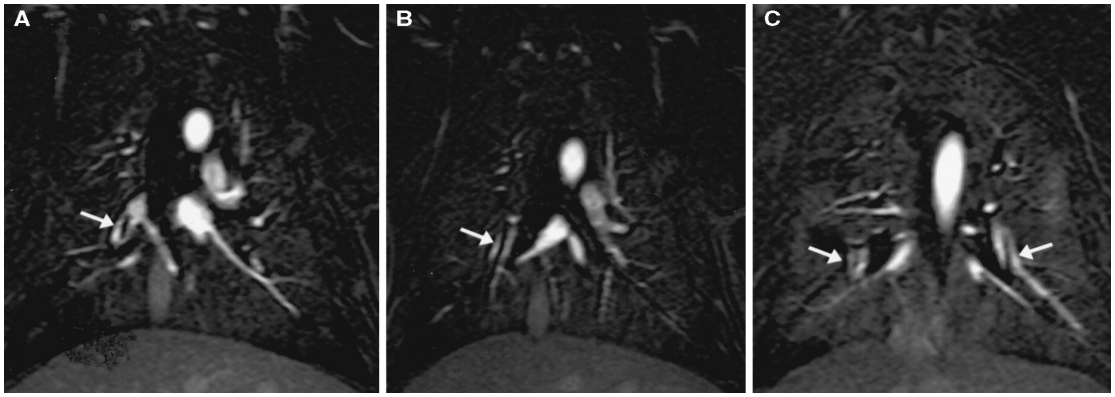
Echocardiography.
Helpful in differential diagnosis and assessment of acute circulatory collapse.

Pulmonary angiogram :

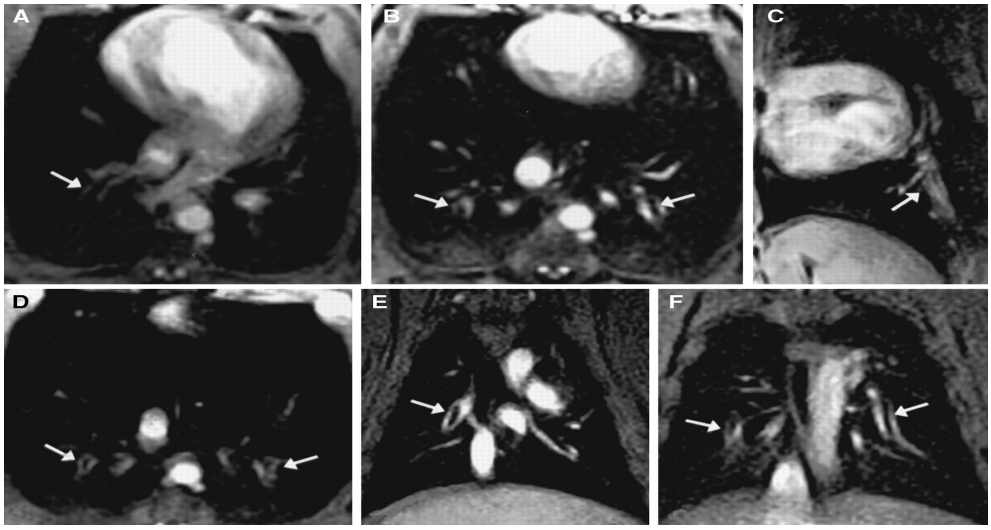




MRA with contrast :

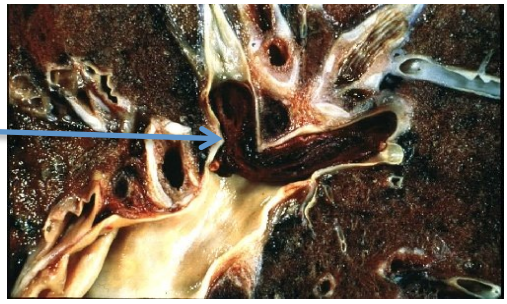


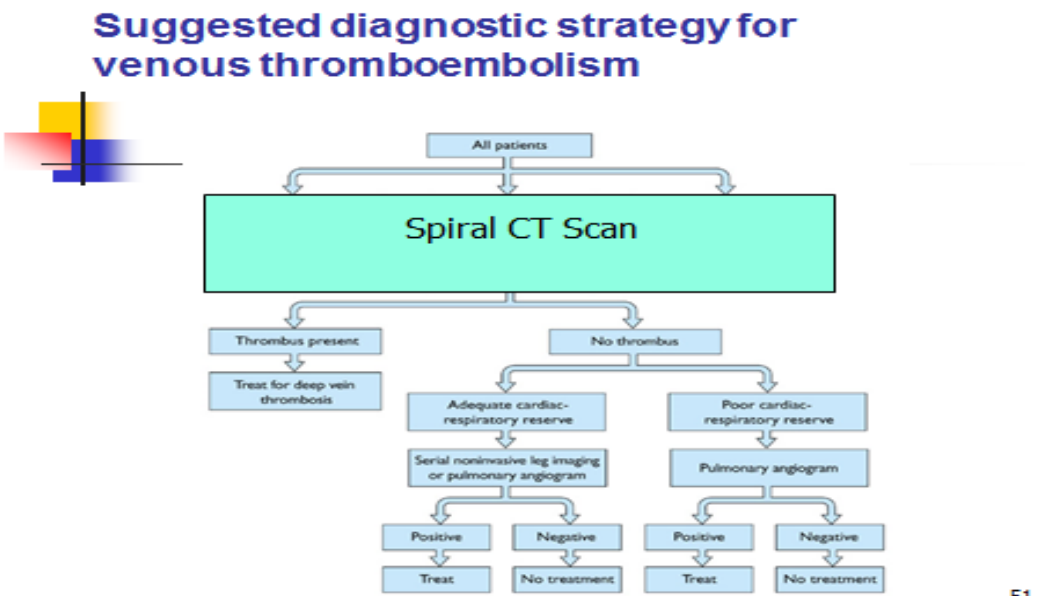
MRA Real Time :



PULMONARY EMBOLISM

Saddle embolism "A large embolism that straddles the arterial bifurcation and thus blocks both branches."





Treatment :

- Respiratory support
- Hemodynamic Support
- Anticoagulation

Dosage and monitoring of anticoagulant therapy

Dosage and monitoring of anticoagulant therapy

After initiating heparin therapy, repeat APTT every 6 h for first 24 h and then every 24 h when therapeutic APTT is achieved

Warfarin 5 mg/d can be started on day 1 of therapy; there is no benefit from higher starting doses

Platelet count should be monitored at least every 3 d during initial heparin therapy

Therapeutic APTT should correspond to plasma heparin level of 0.2–0.4 IU/mL

Heparin is usually continued for 5–7 d

Heparin can be stopped after 4–5 d of warfarin therapy when INR is in 2.0–3.0 range

Important drug interactions with warfarin

Drugs that decrease warfarin requirement	Drugs that increase warfarin requirement
Phenylbutazone	Barbiturates
Metronidazole	Carbamazepine
Trimethoprim-sulfamethoxazole	Rifampin
Amiodarone	Penicillin
Second- and third-generation cephalosporins	Griseofulvin
Clofibrate	Cholestyramine
Erythromycin	
Anabolic steroids	
Thyroxine	

The figure includes only a short list of commonly used agents that are known to have clinically significant interactions with warfarin; several other drugs have pharmacokinetic and pharmacodynamic interactions with warfarin.

· Careful review of medications, alcohol consumption, and dietary factors is mandatory in patients who are on warfarin therapy.

Complications of anticoagulation

	Complication	Management
Heparin	Bleeding	Stop heparin infusion. For severe bleeding, the anticoagulant effect of heparin can be reversed with intravenous protamine sulfate 1 mg/100 units of heparin bolus or 0.5 mg for the number of units given by constant infusion over the past hour; provide supportive care including transfusion and clot evacuation from closed body cavities as needed.

Complications of anticoagulation

	Complication	Management
Heparin	Heparin-induced thrombocytopenia and thrombosis	Carefully monitor platelet count during therapy. Stop-heparin for platelet counts <75,000. Replace heparin with direct inhibitors of thrombin-like desirudin if necessary. These agents do not cause heparin-induced thrombocytopenia. Avoid platelet transfusion because of the risk for thrombosis.

Complications of anticoagulation

	Complication	Management
Heparin	Heparin-induced osteoporosis (therapy >1 mo)	LMWHs may have lower propensity to cause osteoporosis as compared with unfractionated heparin; consider LMWH if prolonged heparin therapy is necessary.

Complications of anticoagulation

	Complication	Management
Warfarin	Bleeding	Stop therapy. Administer vitamin K and fresh-frozen plasma for severe bleeding; provide supportive care including transfusion and clot evacuation from closed body cavities as needed
	Skin necrosis (rare)	Supportive care.
	Teratogenicity	Do not use in pregnancy or in patients planning to become pregnant.

Risks and benefits of thrombolytics vs heparin therapy for pulmonary embolism

	Thrombolytic therapy	No difference	Heparin
Improved resolution at 2-4 h after onset of therapy			
Angiography	+	-	-
Pulmonary artery pressure	+	-	-
Echocardiography	+	-	-
Resolution at 24 h			
Lung scan	+	-	-
Angiography	+	-	-

Risks and benefits of thrombolytics vs heparin therapy for pulmonary embolism

	Thrombolytic therapy	No difference	Heparin
Echocardiography	+	-	-
Pulmonary artery pressure	+	-	-
Resolution at 1 wk and 30 d (lung scan)	-	+	-
Rate of confirmed recurrent pulmonary embolism	-	+	-

Risks and benefits of thrombolytics vs heparin therapy for pulmonary embolism

	Thrombolytic therapy	No difference	Heparin
Hospital mortality	-	+	-
Late mortality	-	+	-
Less severe bleeding	-	-	+
Less intracranial hemorrhage	-	-	+
Lower cost	-	-	+

Approved thrombolytics for pulmonary embolism

Approved thrombolytics for pulmonary embolism

Streptokinase

250,000 IU as loading dose over 30 min, followed by 100,000 U/h for 24 h

Urokinase

4400 IU/kg as a loading dose over 10 min, followed by 4400 IU/kg/h for 12-24 h

Recombinant tissue-plasminogen activator

100 mg as a continuous peripheral intravenous infusion administered over 2 h

Indications for thrombolytic therapy in pulmonary embolism:

- Hemodynamic instability.
- Hypoxia on 100% oxygen .
- Right ventricular dysfunction by echocardiography
- Massive PE

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)

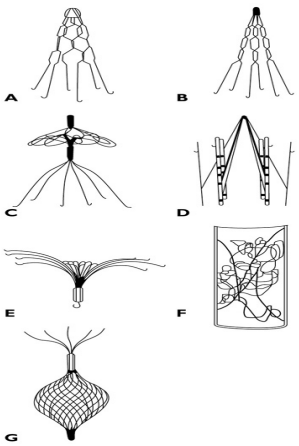
Contraindications :

Relative

Recent surgery within last 10 d Previous arterial punctures within 10 d
Neurosurgery within 6 mo Bleeding disorder (thrombocytopenia, renal failure, liver failure)
Ophthalmologic surgery within 6 wk
Hypertension >200 mm Hg systolic or 110 mm Hg diastolic Placement of central venous catheter within 48 h
Hypertensive retinopathy with hemorrhages or exudates Intracerebral aneurysm or malignancy
Cardiopulmonary resuscitation within 2 wk
Cerebrovascular disease
Major internal bleeding within the last 6 mo Pregnancy and the 1st 10 d postpartum
Infectious endocarditis Severe trauma within 2 mo
Pericarditis

Absolute

Active internal bleeding



Various inferior vena caval filters

Indications for inferior vena caval (IVC) filters:

- Absolute contraindication to anticoagulation (eg, active bleeding)
- Recurrent PE despite adequate anticoagulant therapy
- Complication of anticoagulation (eg, severe bleeding)
- Hemodynamic or respiratory compromise that is severe enough that another PE may be lethal

Embolectomy:

- Embolectomy (ie, removal of the emboli) can be performed using catheters or surgically.
- It should be considered when a patient's presentation is severe enough to warrant thrombolysis (eg, persistent hypotension due to PE), but this approach either fails or is contraindicated.

Conclusions :

- PE is common and under-recognized serious medical problem
- Early diagnosis and treatment is essential for good outcome
- High index of suspicion is needed in high risk patients

Summary : from step up

- Pulmonary embolism is a medical emergency .
- Diagnosis of DVT or PE is an indication for treatment .
- Lower extremities are the main source of emboli .
- In severe cases, acute cor pulmonale may result .
- Clinical symptoms become more overt as the size of dead space in lung increases.
- Symptoms of PE are not specific .
- Most often PE is silent.
- Dyspnea , pleuritic chest pain , tachypnea are the most common manifestations.
- Recurrences are common .
- Spiral CT is the test of choice in diagnosing PE.
- DVT is diagnosed by ultra sound and clinical suspicion.
- V/Q scan plays an important role in diagnosing PE if helical CT is contraindicated.
- Pulmonary angiography can make a definite diagnosis but, it is invasive.
- Start therapeutic heparin as initial treatment. Also start warfarin at the same time.