



Review Article

Pulmonary Embolism- A New Challenge In Diagnosis

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Abstract

Pulmonary embolism is one of the dreadful situations demanding immediate interventions. Any delay in instituting treatment would lead to death. We have dearth of idea about the condition, many patients go undiagnosed and underevaluated. Dyspnoea, tachypnoea, hypoxemia are cardinal features of PE. Deep vein thrombosis (DVT) and PE have almost similar risk factors. Among the risk factors recent immobilization, major surgery, recent limb trauma /or surgery, pregnancy post partum etc. Important diagnostic tools like VQ scanning, assay of D-dimer could only be done only at highly specialized centers. If immediate treatment is undertaken many deaths could be averted. An X-ray chest, ECG, echocardiography could give some important diagnostic clues.

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Introduction

With the advancement of laboratory diagnostic facilities many of the probable diagnostic diseases could now be diagnosed. Pulmonary Embolism (PE) is such a disease which poses a great diagnostic dilemma. PE has a high mortality if appropriate and adequate measures are not taken promptly. Many cases of PE is wrongly diagnosed due to inadequate study by the researchers and lack of idea about the disease.

Pulmonary Embolism (PE) is one of the most common acute severe pulmonary illnesses and account for at least 30,000 deaths per annum in U.K. Symptomatic PE occurs in up to 1% of all post operative patients. A significant proportion are not suspected in life. Thus continued awareness of the possibility of PE in high risk group and willingness to pursue the diagnosis on the ground of clinical suspicion are likely to reduce morbidity and save lives.

PE most commonly result from detachment of vascular thrombus from the leg (70-80%) or pelvis (10-15%). Other causes of PE include amniotic fluid, placenta, air, fat, tumors (eg. Choriocarcinoma), parasites eg. Schistosomiasis and septic emboli from endocarditis affecting pulmonary and tricuspid valves.

Major risk factors

(Relative risk >5)

1. Recent immobilization or major surgery
2. Recent lower limb trauma and/or surgery
3. Clinical DVT
4. Previous proven DVT or PE
5. Pregnancy or post partum
6. Major medical illnesses including malignancy

Minor risk factors

1. Oral oestrogens
2. Long distance travel
3. Minor surgery
4. Thrombophilia
5. Obesity or smoking

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Clinical assessment

PE is a disease of pulmonary circulation, hence indirectly affecting the lung parenchyma whereas cough and haemoptysis often are due to diseases of airways.

Chest pain: The lung has no pain fibers, including the visceral pleura, PE can only cause chest pain if there are peripheral parenchymal changes spreading outside the lung to the (very sensitive) parietal pleura. Where CxR shows normal lung parenchyma, acute pleuritic chest pain most likely arises from the parietal pleura (perhaps with an effusion) or chest wall, making PE unlikely—but there are exceptions, in massive PE chest pain is central and nonpleuritic.

Dyspnoea, tachypnoea, and hypoxia common in PE, and if CxR is normal suggest that this is the probable diagnosis. Conversely, a patient without dyspnoea and also respiratory rate <20/min is unlikely to have PE, especially if normoxic (both sentences evidence-based). Circulatory collapse is noted in massive PE.

Fever may occur, but such patients never volunteer this, thus PE is very unlikely in a patient with febrile symptoms. Similarly an acute chest illness associated with anorexia or upper respiratory tract onset will be a chest infection.

Cough: If a prominent symptom, is far more likely to be due to airway disease, particularly if there is wheeze.

Haemoptysis PE is unlikely if (a) moderate or large volume or (b) no other features of PE or (c) normal chest X-ray. Haemoptysis in PE is rarely the main presenting clinical feature.

Standard investigations

Chest X-ray, if necessary repeat it. Look for the following:

1. The more abnormal -the less likely is PE
2. If normal in a breathless hypoxic patient - the more likely is PE
3. If no pleural reaction in spite of chest pain - PE unlikely (but can occur)

4. Inspect carefully for consolidation, pleural effusion, lung cancer (all easy to overlook when PE is being considered). Pneumonia is the most frequent oversight.

ECG: Although changes are common in PE, none is suggestive (even S₁, Q₃, T₃). Tachycardia, right axis deviation, right bundle branch block (RBBB).

However very useful at revealing alternative diagnosis (eg, pericarditis, IHD).

OXYMETRY: Ensure that the patient has been in room air before assessment.

1. If normal, any PE is likely to be small
2. If low, and patient tachypnoic and normal chest X-ray, PE must be seriously considered.
3. Remember, anemia does not cause hypoxia.
4. If SAT >98% on air, consider hyper-ventilation (if so, high PaO₂ + low PACo₂ on blood gases).

D-dimer: (Reflecting plasmin breakdown of fibrin). Remember, do not do it if:

1. Patient is febrile, or any evidence of inflammatory disease.
2. You already have a clear diagnosis.
3. High clinical probability- these will need imaging anyway.

If it already has been done in this circumstances, and positive, ignore it.

Blood D-dimer assay should only be considered following assessment of clinical probability.

A negative D-dimer test reliably excludes PE in patients.

D-dimer might be positive in MI, sepsis, pregnancy, post operative.

Echocardiography; (Transthoracic):

1. May show other diagnosis
2. May show RV clot
3. Acute right heart strain
4. Raised TR velocity
5. Non collapsible IVC
6. Paradoxical septal movement

Blood gas analysis : \uparrow PaO₂, \downarrow PaCO₂.

VQ Scanning (Ventilation Perfusion test):

Lung scanning is particularly useful if the result are normal or near normal or if there is high probability for PE. The diagnosis of PE is very unlikely in patients with normal or near normal scans, but in contrast, it is about 90% certain in patients with high probability scans.

Pulmonary Angiography

Selective pulmonary angiography is the most specific examination available for establishing definitive diagnosis of PE and can detect emboli as small as 1 to 2 mm. Pulmonary angiography is most useful or when the lung scan is intermediate probability for PE.

The discussion would remain incomplete if deep vein thrombosis (DVT) is not discussed.

Deep Vein Thrombosis

Deep veins	Calf	Peroneal, tibial, gastrocnemus
	Proximal	Popliteal, femoral (superficial/common) Iliac (external common)
Superficial veins	Long saphenous, short saphenous (DVT rare)	

DVT, even proximal with PE, can occur with no symptoms or abnormality in the legs. Always use a tape to measure if DVT suspected.

A. Clinical signs are not sufficient to make a sure diagnosis, suggestions are:

Tenderness most marked along the deep and/or:

1. maximum calf diameter >2 cm than the other leg
2. measurable swelling of both thigh and calf
3. unilateral pitting oedema
4. unilateral visible collaterals (uncommon)

B. Commonest differential diagnosis are:

- Superficial thrombophlebitis, redness & tenderness of saphenous veins
- Cellulitis, circumferential redness & tenderness, demarcated (DVT rare)
- Rupture Bakers cyst: history of recent knee swelling (often detectable)
- Calf hematoma, history of trauma may be due to warfarin.
- Ruptured muscle: uncommon, confirmed on standing & walking.

C. Clinical probability is assessed similar to PE, i.e. by a consideration of:

- signs suggestive of DVT
- alternative diagnosis unlikely
- major risk factors (described earlier)

D. Indication of compression ultrasound:

- if low/ intermediate probability plus negative D-dimer, not DVT
- if high probability, D-dimer unnecessary and wastage of time
- if high probability but unremarkable ultrasound, LMWH, APTT test in 3-7 days
- In a few cases with unremarkable ultrasound, early venography may be wise.

E. Reducing post phlebotic syndrome:

If clinical changes of proximal DVT are marked always consider:

- First dose of unfractionated heparin IV (works much quicker than LMWH)
- Advise for proper elevation of legs and full length graded compression stockings.
- Advise from local vascular surgeon.

If obvious high risk of post-phlebotic syndrome, consider standard thrombolysis. Some centers have vascular radiologists able to give local (lower dose, safer treatment):

1. Myocardial Infarction, unstable angina
2. Pneumonia, bronchitis, COPD exacerbation
3. CCF
4. Severe acute asthma
5. Pericarditis
6. Primary pulmonary hypertension
7. Rib fracture, pneumothorax
8. Costochondritis, musculoskeletal pain, anxiety.

Management

High flow oxygen supplementation, lie patient flat, to attain good venous return

1. Pain relieve (NSAID)
2. Rt. Heart failure and shock, should be treated with Dobutamin.
3. Mechanical ventilation.

Blood clot

Start anticoagulation with low molecular weight Heparin. Consider thrombolysis if there a major

embolus. Embolectomy is necessary if the patient remains moribund at 24-48 hours. Commence warfarin (oral anticoagulant) but continue Heparin for further 2-3 days after adequate oral dosing. Low molecular weight heparin regime. Deltaparin, Enoxaparin, Tinzaparin

Thrombolytic Regime

Rt-PA (100mg over 90 min.) should be given followed by Heparin infusion (24000-36000 unit/day) to maintain partial Thromboplastin time.

The duration of treatment with Warfarin is controversial, depends on age, co morbidity, etc. Adjustment of INR (International Normalization Ratio) should be checked frequently and kept to <3. Recurrence rate of PE once attacked is high, so patient should be kept under surveillance.

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