ЗМІСТ

Internal Medicine: Critical Care: textbook

Про книгу

The “Internal Medicine: Critical Care” provides the depth and breadth of coverage that reflects the complexity and expertise needed to practice emergency medicine successfully in today’s fast—paced environments. It is an important contemporary clinical emergency care resource for students of higher education eshtablishments — medical universities, institutes and academies. The textbook was published in the English language, illustrated with pictures and tables, which are easy to learn and to store in memory for a long time. This textbook also gives possibility to find answers quickly when you are faced with a difficult diagnosis or need the latest treatment recommendations, step-by-step guidelines and new pharmacologic considerations.

INTERNAL MEDICINE: Critical Care

TEXTBOOK

Edited by O.Ya. BABAK, O.M. BILOVOL

RECOMMENDED
by the Academic Board of Kharkiv National Medical University as a textbook for students of higher education establishments — medical universities, institutes and academies

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Contents

Chapter 3

PULMONARY EMBOLISM

Pulmonary embolism occurs when some substance (usually a thrombus, fat, air, or amniotic fluid) lodges in the pulmonary vasculature resulting in obstruction of the blood flow to that portion of the lung.

Epidemiology

PE is a relatively common disease, with an annual incidence of 23 cases per 100,000 persons in the United States. PE is an extremely common and highly lethal condition that is a leading cause of death in all age groups.

Etiology and Pathogenesis

The most common type of pulmonary embolism is a blood clot, usually one that forms in a leg or pelvic vein (deep vein thrombosis) when the blood flow slows down or stops, as may occur in the leg veins when a person stays in one position for a long time (table 11). People who have been on prolonged bed rest and those recovering from major surgery are at risk. Those sitting for long time periods without moving around (as may happen during air travel) are at a slightly increased risk. Far less often, blood clots form in the veins of the arms or in the right side of the heart. Once a clot breaks free into the bloodstream, it usually travels to the lungs.

TABLE 11

Risk Factors for Venous Thromboembolism
(British Thoracic Society Standards of Care Committee Pulmonary Embolism Guideline Development Group, 2003)

Major risk factors (relative risk 5–20): Surgery*	• Major abdominal/pelvic surgery • Hip/knee replacement • Postoperative intensive care
Obstetrics	• Late pregnancy • Caesarian section • Puerperium
Lower limb problems	• Fracture • Varicose veins

Table 11 continued

Malignancy	• Abdominal/pelvic • Advanced/metastatic
Reduced mobility	• Hospitalisation • Institutional care
Miscellaneous	• Previous proven VTE
Minor risk factors (relative risk 2–4): Cardiovascular	• Congenital heart disease • Congestive cardiac failure • Hypertension • Superficial venous thrombosis • Indwelling central vein catheter
Oestrogens	• Oral contraceptive • Hormone replacement therapy
Miscellaneous	• COPD • Neurological disability • Occult malignancy • Thrombotic disorders • Long distance sedentary travel • Obesity • Other†

*Where appropriate prophylaxis is used, relative risk is much lower.
†Inflammatory bowel disease, nephrotic syndrome, chronic dialysis, myeloproliferative disorders, paroxysmal nocturnal haemoglobinuria, Behçet's disease

Unusual Types of Emboli

Fat can escape into the blood from the bone marrow when a long bone is fractured or during bone surgery and form an embolus.

Amniotic fluid that is forced into the pelvic veins during a tumultuous childbirth can form an embolus.

Cancer cells in clumps may break free into the circulation to form tumour emboli.

Air bubbles may form emboli if a catheter in one of the large veins (central veins) is inadvertently opened to air. Air emboli may also form when a vein is operated on (such as when a blood clot is being removed) or when a person is being resuscitated (because of the force of chest compressions). An additional risk is underwater diving (pulmonary barotrauma).

Infected material may also form emboli and travel to the lung. Causes include intravenous drug use, certain heart valve infections, parasites and inflammation of a vein with blood clot formation and infection (septic thrombophlebitis).

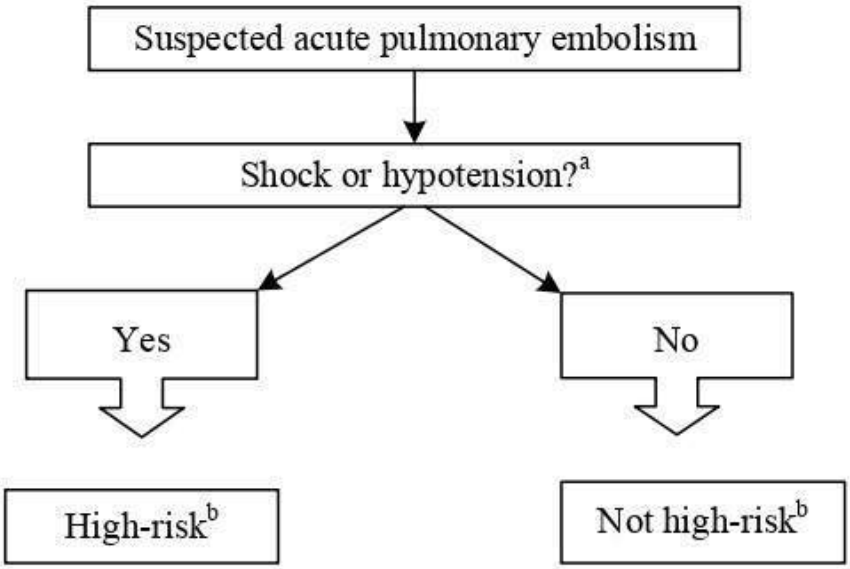
A foreign substance can be introduced into the bloodstream, usually by intravenous injection of inorganic substances such as talc by injection drug users, where it can form emboli and travel to the lung, venous catheters etc.

Classification

The clinical classification of the severity of an episode of acute PE is based on the estimated PE-related early mortality risk defined by in-hospital or 30-day mortality (Fig. 2). This stratification, which has important implications both for the diagnostic and therapeutic strategies, is based on the patient's clinical status at presentation, with high-risk PE being suspected or confirmed in the presence of shock or persistent arterial hypotension and not high-risk PE in their absence.

Clinical Presentation

Symptoms that should provoke a suspicion of PE must include chest pain, chest wall tenderness, back pain, shoulder pain, upper abdominal pain, syncope, haemoptysis, shortness of breath, painful respiration, new



[a] Defined as systolic blood pressure < 90 mm Hg or systolic pressure drop by ≥ 40 mm Hg for > 15 minutes if not caused by new-onset arrthymia, hypovolaemia or sepsis.

[b] Based on the estimated PE-related in-hospital or 30-day mortality.

Fig. 2. **Initial risk stratification of acute PE**

onset of wheezing, any new cardiac arrhythmia, or any other unexplained symptom referable to the thorax.

The classic triad of signs and symptoms of PE (haemoptysis, dyspnoea, chest pain) are neither sensitive nor specific. They occur in fewer than 20 % of patients in whom the diagnosis of PE is made, and most patients with those symptoms are found to have some etiology other than PE to account for them. Of patients who go on to die from massive PE, only 60 % have dyspnoea, 17 % have chest pain and 3 % have haemoptysis. Nonetheless, the presence of any of these classic signs and symptoms is an indication for a complete diagnostic evaluation.

Many patients with PE are initially completely asymptomatic, and most of those who do have symptoms have an atypical presentation.

Patients with PE often present with primary or isolated complaints of seizure, syncope, abdominal pain, high fever, productive cough, new onset of reactive airway disease (adult-onset asthma), or hiccoughs. They may present with new-onset atrial fibrillation, disseminated intravascular coagulation, or any of a host of other signs and symptoms.

Pleuritic or respirophasic chest pain is a particularly worrisome symptom. PE has been diagnosed in 21 % of young, active patients who come to the ED complaining only of pleuritic chest pain. These patients usually lack any other classical signs, symptoms, or known risk factors for pulmonary thromboembolism. Such patients often are dismissed inappropriately with an inadequate workup and a nonspecific diagnosis, such as musculoskeletal chest pain or pleurisy.

Signs. Examination may reveal tachycardia and tachypnoea only. Look for postural hypotension (in the presence of raised JVP).

Signs of raised right heart pressures and cor pulmonale (raised JVP, tricuspid regurgitation, parasternal heave, right ventricular S3, loud pulmonary closure sound with wide splitting of S2, pulmonary regurgitation). Cyanosis suggests a large pulmonary embolism.

Pleural rub (may be transient) or effusion. Examination of the lower limbs for obvious thrombophlebitis. Mild fever (> 37.5 °C) may be present. There may be signs of co-existing COPD.

Diagnosis

The diagnosis of PE is based primarily on validated clinical criteria combined with selective testing because the typical clinical presentation (shortness of breath, chest pain) cannot be definitively differentiated from other causes of chest pain and shortness of breath.

The decision to do medical imaging is usually based on clinical grounds, i. e. the medical history, symptoms and findings on physical examination.

The most commonly used method to predict clinical probability, the Wells score, is a clinical prediction rule, whose use is complicated by multiple versions being available (table 12).

TABLE 12

The Wells Score (ESC 2008)

Variables	Points
Predisposing factors	
Previous PE or DVT	+1.5
Recent surgery or immobilisation	+1.5
Cancer	+1.0
Symptoms	
Haemoptysis	+1.0
Clinical signs	
Heart rate > 100 beats per minute	+1.5
Clinical signs of DVT	+3.0
Clinical judgment	
Alternative diagnosis is less likely than PE	+3.0
Clinical probability (2-level)	Total
PE unlikely	0–4
PE likely	>4
Clinical probability (3-level)	Total
Low	0 to1
Intermediate	2 to 6
High	≥ 7

There are additional prediction rules for PE, such as the Geneva score (table 13).

TABLE 13

Geneva Score (Revised ESC 2008)

Variables	Points
Predisposing factors	
Age > 65 years	+1
Previous PE or DVT	+3
Surgery or fracture within one month	+2
Active malignancy	+2
Symptoms	
Unilateral lower limb pain	+3
Haemoptysis	+2

Table 13 continued

Variables	Points
Clinical signs *Heart rate* 75 to 94 beats per minute ≥ 95 beats per minute Pain on lower limb deep vein at palpation and unilateral	 +3 +5 +4
Clinical probability Low Intermediate High	Total 0 to 3 4 to 10 ≥11

Blood Tests

D-dimer levels are elevated in plasma in the presence of acute thrombosis because of simultaneous activation of coagulation and fibrinolysis. The negative predictive value of D-dimer testing is high and a normal D-dimer level renders acute PE or DVT unlikely. On the other hand, fibrin is also produced in a wide variety of conditions such as cancer, inflammation, bleeding, trauma, surgery and necrosis. Accordingly, the positive predictive value of elevated D-dimer levels is low and D-dimer testing is not useful for confirmation of PE. In low/moderate suspicion of PE, a normal D-dimer level (shown in a blood test) is enough to exclude the possibility of thrombotic PE.

Markers of Myocardial Injury. Transmural RV infarction despite patent coronary arteries has been found at autopsy of patients who died of massive PE. Elevated plasma troponin concentrations on admission have been reported in connection with PE and were associated with worse prognosis. Heart-type fatty acid-binding protein, an early marker of myocardial injury, was also found to possess prognostic value in acute PE.

Other (Non-cardiac) Laboratory Biomarkers. Elevated serum creatinine levels and a decreased (calculated) glomerular filtration rate are related to 30-day all-cause mortality in acute PE. Elevated neutrophil gelatinase-associated lipocalin (NGAL) and cystatin C, both indicating acute kidney injury, have also been found to be of prognostic value.

When a PE is being suspected, a number of blood tests are done, in order to exclude important secondary causes of PE. This includes a full blood count, clotting status (PT, APTT, TT) and some screening tests (erythrocyte sedimentation rate, renal function, liver enzymes, electrolytes). If one of these is abnormal, further investigations might be warranted.

Medical Imaging

The gold standard for diagnosing PE is pulmonary angiography. Pulmonary angiography is used less often due to wider acceptance of CT scans, which are non-invasive.

Non-invasive imaging:

— ***High-resolution multidetector CT pulmonary angiography*** is a pulmonary angiogram obtained using CT with radiocontrast rather than right heart catheterisation. Its advantages are clinical equivalence, its non-invasive nature, its greater availability to patients and the possibility of identifying other lung disorders from the differential diagnosis in case there is no pulmonary embolism. Assessing the accuracy of CT pulmonary angiography is hindered by the rapid changes in the number of rows of detectors available in multidetector CT machines. A study with a mixture of 4 slice and 16 slice scanners reported a sensitivity of 83 % and a specificity of 96 %. The last data suggest that a negative MDCT is an adequate criterion for excluding PE in patients with a non-high clinical probability of PE. Whether patients with a negative CT and a high clinical probability should be further investigated is controversial. MDCT showing PE at the segmental or more proximal level is an adequate proof of PE in patients with a non-low clinical probability; however, the positive predictive value of MDCT is lower in patients with a low clinical probability of PE and further testing may be considered, especially if the clots are limited to segmental or sub-segmental arteries.

MCTPA is non-inferior to V/Q scanning and identifies more emboli (without necessarily improving the outcome) compared to V/Q scanning.

— ***Ventilation/perfusion scan*** (or V/Q scan or lung scintigraphy) is an established diagnostic test for suspected PE. The test is based on the intravenous injection of technetium (Tc)-99m-labelled macroaggregated albumin particles, which block a small fraction of the pulmonary capillaries and thereby enable scintigraphic assessment of lung perfusion. Perfusion scans are combined with ventilation studies, for which multiple tracers such as xenon-133 gas, Tc-99m-labelled aerosols, or Tc-99m-labelled carbon microparticles (Technegas) can be used. Being a radiation- and contrast medium-sparing procedure, the V/Q scan may be preferentially applied in outpatients with a low clinical probability and a normal chest X-ray, in young (particularly female) patients, in pregnancy, in patients with a history of contrast medium-induced anaphylaxis and strong allergic history, in severe renal failure, and in patients with myeloma and paraproteinaemia.

— ***Pulmonary angiography*** has for decades remained the 'gold standard' for the diagnosis or PE exclusion, but is rarely performed now as

less-invasive CT angiography offers similar diagnostic accuracy. Pulmonary angiography is more often used to guide percutaneous catheter-directed treatment of acute PE. Digital subtraction angiography requires less contrast medium than conventional cineangiography and has excellent imaging quality for peripheral pulmonary vessels in patients who can hold their breath; it is less useful for imaging of the main pulmonary arteries due to cardiac motion artefacts. The diagnosis of acute PE is based on direct evidence of a thrombus in two projections, either as a filling defect or as amputation of a pulmonary arterial branch. Thrombi as small as 1–2 mm within the sub-segmental arteries can be visualised by DSA, but there is substantial interobserver variability at this level.

— ***Magnetic resonance angiography*** has been evaluated for several years in suspected PE but is not yet ready for clinical practice due to its low sensitivity, high proportion of inconclusive MRA scans and low availability in most emergency settings.

— ***Chest X-rays*** are often done on patients with shortness of breath to help rule out other causes, such as congestive heart failure and rib fracture. The initial CXR findings of a patient with PE are virtually always normal, although on rare occasions they may show the Westermark sign (i. e. a dilatation of the pulmonary vessels proximal to an embolism along with collapse of the distal vessels, sometimes with a sharp cutoff). Over time, an initially normal CXR often begins to show atelectasis, which may progress to cause a small pleural effusion and an elevated hemidiaphragm. After 24–72 hours, one third of patients with proven PE develop focal infiltrates that are indistinguishable from an infectious pneumonia. A rare late finding of pulmonary infarction is the Hampton hump, a triangular or rounded pleural-based infiltrate with the apex pointed toward the hilum, frequently located adjacent to the diaphragm.

— ***Ultrasonography.*** Acute PE may lead to RV pressure overload and dysfunction, which can be detected by echocardiography. Given the peculiar geometry of the RV, there is no individual echocardiographic parameter that provides fast and reliable information on RV size or function. This is why echocardiographic criteria for the diagnosis of PE have differed between studies. The specific appearance of the right ventricle on echocardiography is referred to as the McConnell sign. This is the finding of akinesia of the mid-free wall but normal motion of the apex. This phenomenon has a 77 % sensitivity and a 94 % specificity for the diagnosis of acute pulmonary embolism. Because of the reported negative predictive value of 40–50 %, a negative result cannot exclude PE. On the other hand, signs of RV overload or dysfunction may also be found in the absence of acute PE and be due to concomitant cardiac or respiratory disease.

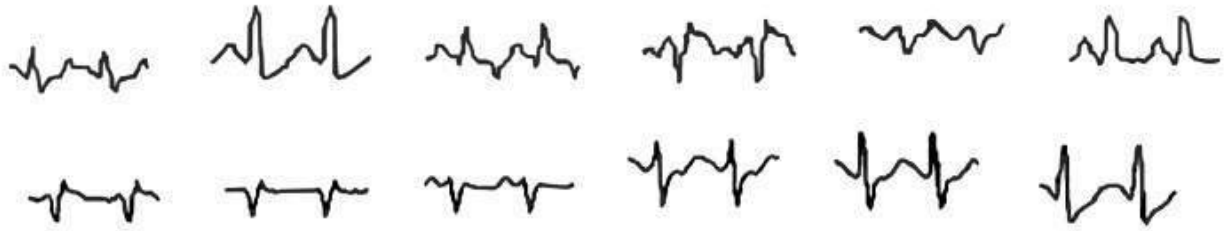


Fig. 3. **Pulmonary embolism, ECG, S1Q3T3**

— ***Compression Venous Ultrasonography.*** DVT was found in 70 % of patients with proven PE. Nowadays, lower limb CUS has largely replaced venography for diagnosing DVT. CUS has a sensitivity > 90 % and a specificity of approximately 95 % for symptomatic DVT. The presence of DVT, as shown on ultrasonography of the legs, is in itself enough to warrant anticoagulation, without requiring the V/Q or spiral CT scans (because of the strong association between DVT and PE).

— ***Electrocardiogram*** findings. An electrocardiogram is routinely done on patients with chest pain to quickly diagnose myocardial infarctions (heart attacks). An ECG may show signs of right heart strain or acute cor pulmonale in cases of large PEs — the classic signs are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III (S1Q3T3, Fig. 3). This is occasionally (up to 20 %) present, but may also occur in other acute lung conditions and has therefore limited diagnostic value. The most commonly seen signs in the ECG are sinus tachycardia, right axis deviation and right bundle branch block.

— ***Echocardiography*** findings. Dysfunction of the right side of the heart can be seen on echocardiography, an indication that the pulmonary artery is severely obstructed and the heart is unable to match the pressure. Some studies suggest that this finding may be an indication for thrombolysis. Not every patient with a (suspected) pulmonary embolism requires an echocardiogram, but elevations in cardiac troponins or brain natriuretic peptide may indicate heart strain and warrant an echocardiogram.

Diagnostic Strategies (table 14)

Suspected high-risk and non-high-risk PEs are two distinct situations that must be distinguished because the diagnostic strategies differ. Overall, with adequate clinical awareness the prevalence of PE inpatients in whom the disease is suspected is low (10–35 % in recent large series). Pulmonary angiography, the definitive standard criterion, is invasive, costly and sometimes difficult to interpret. Hence, non-invasive diagnostic approaches are warranted, and various combinations of clinical evaluation, plasma D-dimer measurement, lower limb CUS, V/Q lung scintigraphy and, more re-

cently, CT have been evaluated to obviate the requirement for pulmonary angiography. These strategies were applied to patients presenting with suspected PE in the emergency ward, during a hospital stay or both.

Suspected PE with Shock or Hypotension

This is an immediately life-threatening situation, and patients presenting with shock or hypotension present a distinct clinical problem. The differential diagnosis includes acute valvular dysfunction, tamponade, acute coronary syndrome and aortic dissection. The most useful initial test in this situation is bedside transthoracic echocardiography, which will yield evidence of acute pulmonary hypertension and RV dysfunction if acute PE is the cause of the patient's haemodynamic decompensation. In a highly unstable patient, echocardiographic evidence of RV dysfunction is sufficient to prompt immediate reperfusion without further testing. Ancillary bedside imaging tests include transoesophageal echocardiography which, if available, may allow direct visualisation of thrombi in the pulmonary artery and its main branches and bedside CUS, which can detect proximal DVT.

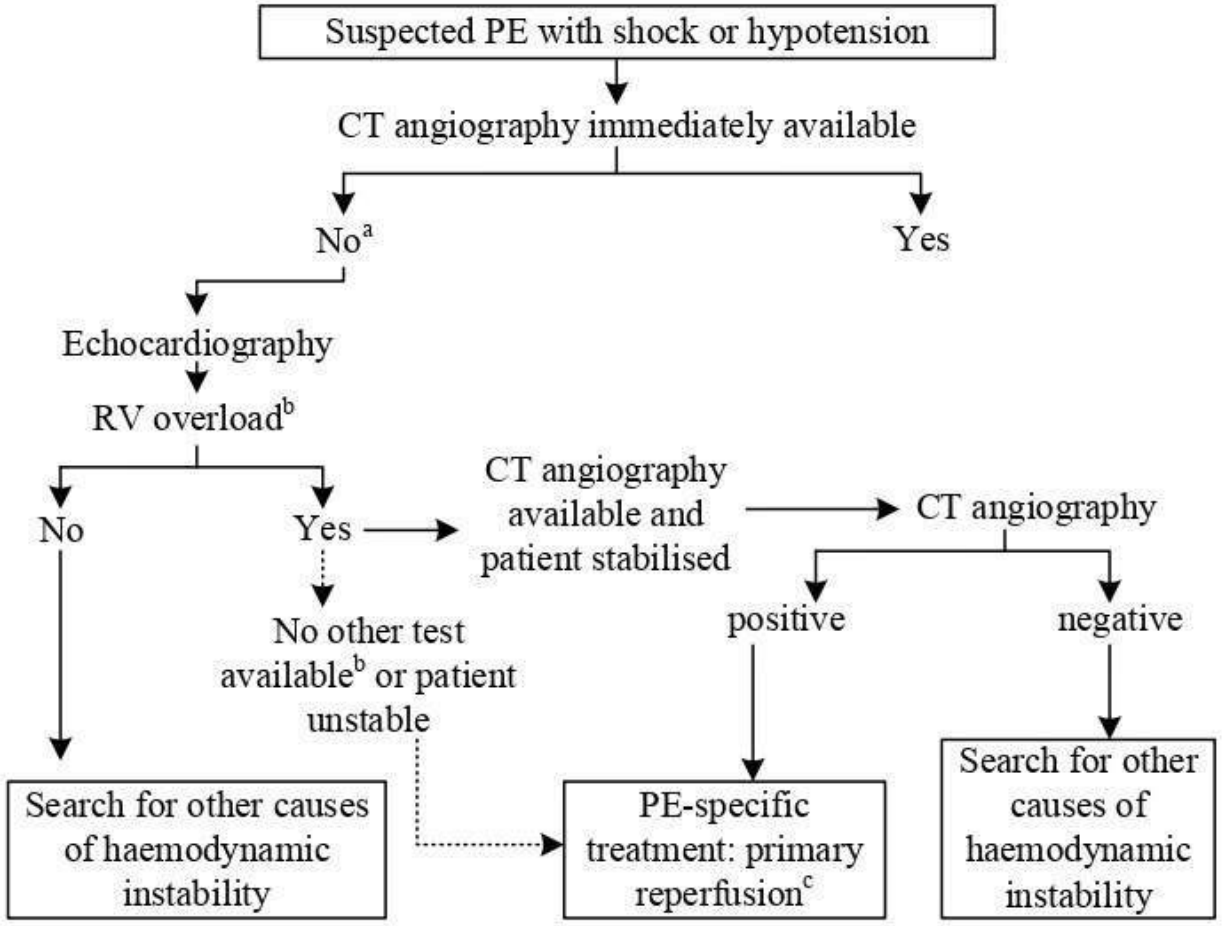
TABLE 14

Validated Diagnostic Criteria (Based on Non-invasive Tests) for Diagnosing PE in Patients without Shock or Hypotension According to Clinical Probability

Diagnostic Criterion	Clinical probability of PE				
	Low	Intermediate	High	PE unlikely	PE likely
Exclusion of PE					
D-dimer					
Negative result, highly sensitive assay	+	+	–	+	–
Negative result, moderately sensitive assay	+	±	–	+	–
Chest CT angiography					
Normal multidetector CT alone	+	+	±	+	±
V/Q scan					
Normal perfusion lung scan	+	+	+	+	+
Non-diagnostic lung scan[a] and negative proximal CUS	+	±	–	+	–
Confirmation of PE					
Chest CT angiogram showing at least segmental PE	+	+	+	+	+
High probability V/Q scan	+	+	+	+	+
CUS showing proximal DVT	+	+	+	+	+

+ — valid diagnostic criterion (no further testing required);
– — invalid criterion (further testing mandatory);
± — controversial criterion (further testing to be considered).
[a] — low or intermediate probability lung scan according to the PIOPED classification.

As soon as the patient can be stabilised by supportive treatment, final confirmation of the diagnosis by CT angiography should be sought. For unstable patients admitted directly to the catheterisation laboratory with suspected ACS, pulmonary angiography may be considered as a diagnostic procedure after the ACS has been excluded, provided that PE is a probable diagnostic alternative and particularly if percutaneous catheter-directed treatment is a therapeutic option.



[a] Includes the cases in which the patient's condition is so critical that it only allows bedside diagnostic tests.

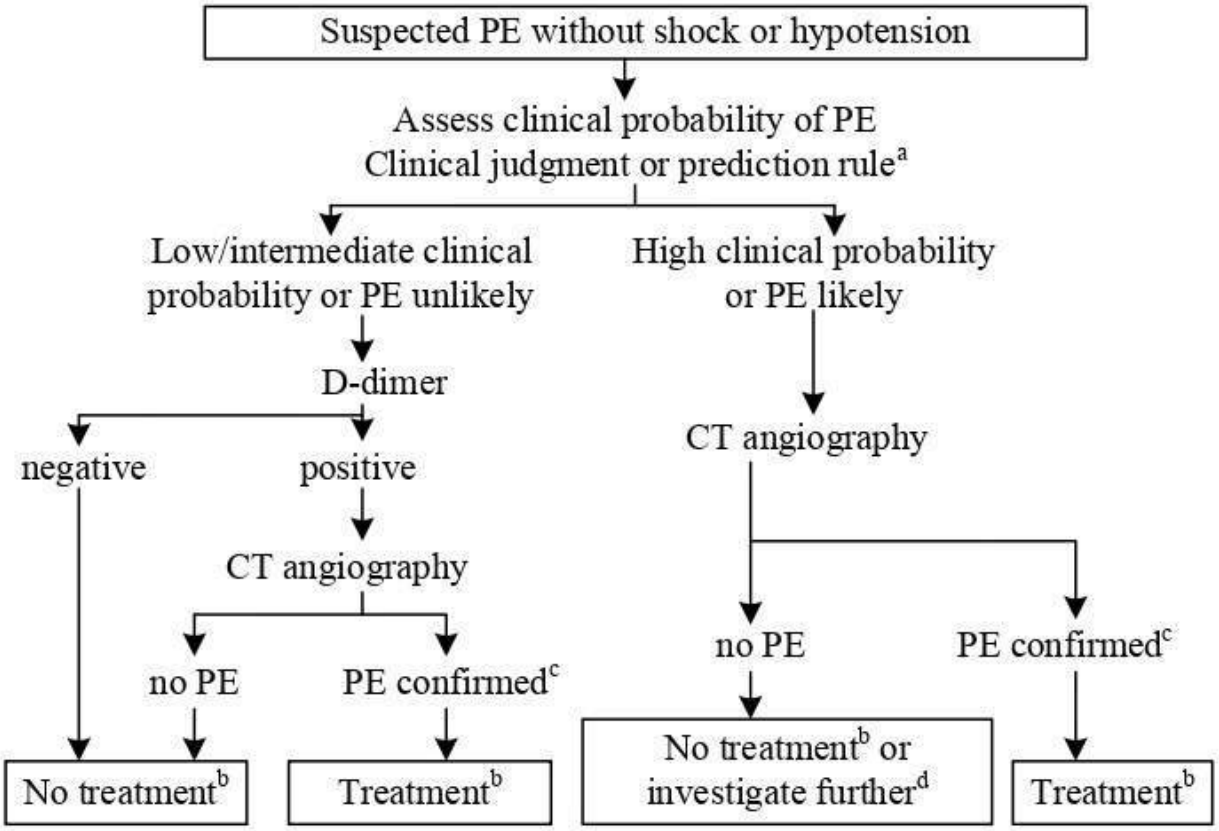
[b] Apart from the diagnosis of RV dysfunction, bedside transthoracic echocardiography may in some cases directly confirm PE by visualising mobile thrombi in the chambers. Ancillary bedside imaging tests include transoesophageal echocardiography, which may detect emboli in the pulmonary artery and its main branches, and bilateral compression venus ultrasonography which may confirm deep vein thrombosis and thus be on help in emergency management decisions.

[c] Thrombolysis; alternatively, surgical embolectomy or catheter-directed treatment.

Fig. 4. **Proposed diagnostic algorithm for patients with suspected high-risk PE (2014 ESC Guidelines on the Diagnosis and Management of Acute Pulmonary Embolism)**

Suspected PE without Shock or Hypotension

In patients admitted to the emergency department, plasma D-dimer measurement, combined with clinical probability assessment, is the logical first step and allows PE to be ruled out in around 30 % of patients in most centres; MDCT angiography is the second-line test in patients with an elevated D-dimer level and the first-line test in patients with a high clinical probability. CT angiography is considered to be diagnostic of PE when it shows a clot at least at the segmental level of the pulmonary arterial tree.



[a] Two alternative classification schemes may be used for clinical probability assessment, i. e. a three-level scheme (clinical probability defined as low, intermediate or high) or a two-level scheme (PE unlikely or PE likely). When using a moderately sensitive assay, D-dimer measurement should be restricted to patients with low clinical probability or a PE-unlikely classification while highly sensitive assays may also be used in patients with intermediate clinical probability of PE. Note that plasma D-dimer measurement is of limited use in suspected PE occurring in hospitalised patients.

[b] Treatment refers to anticoagulation treatment for PE.

[c] CT angiogram is considered to be diagnostic of PE if it shows PE at the segmental or more proximal level.

[d] In case of negative CT angiogram in patients with high clinical probability, further investigation may be considered before withholding PE-specific treatment.

Fig. 5. **Proposed diagnostic algorithm for patients with suspected not high-risk pulmonary embolism**
(2014 ESC Guidelines on the Diagnosis and Management of Acute Pulmonary Embolism)

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Перейти до категорії
Внутрішня медицина