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Pulmonary embolism

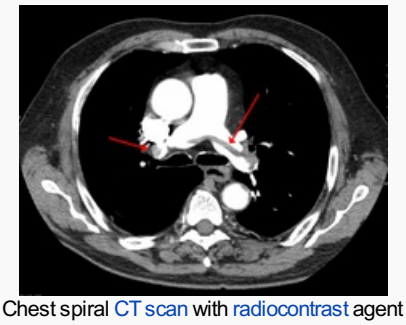
From Wikipedia, the free encyclopedia

Pulmonary embolism (**PE**) is a blockage of the lung's main artery or one of its branches by a substance that has traveled from elsewhere in the body through the bloodstream (embolism). PE results from deep vein thrombosis (commonly a blood clot in a leg) that breaks off and migrates to the lung, a process termed *venous thromboembolism* (VTE). A small proportion of cases are caused by the embolization of air, fat, or talc in drugs of intravenous drug abusers or amniotic fluid. The obstruction of the blood flow through the lungs and the resultant pressure on the right ventricle of the heart lead to the symptoms and signs of PE. The risk of PE is increased in various situations, such as cancer or prolonged bed rest.[1]

Symptoms of pulmonary embolism include difficulty breathing, chest pain on inspiration, and palpitations. Clinical signs include low blood oxygen saturation and cyanosis, rapid breathing, and a rapid heart rate. Severe cases of PE can lead to collapse, abnormally low blood pressure, and sudden death.[1]

Diagnosis is based on these clinical findings in combination with imaging studies, usually CT pulmonary angiography. If suspicion of a pulmonary embolus is lower, a negative result from a D-dimer test can be used to rule out a pulmonary embolus. Treatment is typically with anticoagulant medication, including heparin and warfarin. Severe cases may require thrombolysis using medication such as tissue plasminogen activator (tPA), or may require surgical intervention via pulmonary thrombectomy.[1]

Pulmonary embolism



Chest spiral CT scan with radiocontrast agent showing multiple filling defects both at the bifurcation ("saddle" pulmonary embolism) and in the pulmonary arteries.

Classification and external resources

Specialty	Hematology, cardiology, pulmonology
ICD-10	I26
ICD-9-CM	415.1
DiseasesDB	10956
MedlinePlus	000132
eMedicine	med/1958 emerg/490 radio/582
Patient UK	Pulmonary embolism
MeSH	D011655

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Signs and symptoms [edit]

Symptoms of pulmonary embolism are typically sudden in onset and may include one or many of the following: dyspnea (shortness of breath), tachypnea (rapid breathing), chest pain of a "pleuritic" nature (worsened by

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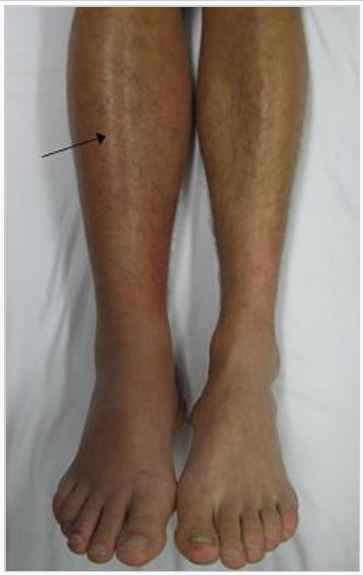
breathing), cough and hemoptysis (coughing up blood). More severe cases can include signs such as cyanosis (blue discoloration, usually of the lips and fingers), collapse, and circulatory instability because of decreased blood flow through the lungs and into the left side of the heart. About 15% of all cases of sudden death are attributable to PE.[1]

On physical examination, the lungs are usually normal. Occasionally, a pleural friction rub may be audible over the affected area of the lung (mostly in PE with infarct). A pleural effusion is sometimes present that is exudative, detectable by decreased percussion note, audible breath sounds and vocal resonance. Strain on the right ventricle may be detected as a left parasternal heave, a loud pulmonary component of the second heart sound, and raised jugular venous pressure.[1] A low-grade fever may be present, particularly if there is associated pulmonary hemorrhage or infarction.[2]

As smaller pulmonary emboli tend to lodge in more peripheral areas without collateral circulation they are more likely to cause lung infarction and small effusions (both of which are painful), but not hypoxia, dyspnea or hemodynamic instability such as tachycardia. Larger PEs, which tend to lodge more centrally, typically cause dyspnea, hypoxia, hypotension, tachycardia and syncope, but are often painless because there is no lung infarction due to collateral circulation. The classic presentation for PE with pleuritic pain, dyspnea and tachycardia is most likely to be caused by a large embolism that fragments and thus causes both large and small PEs. Thus, small PEs are often missed because they cause pleuritic pain alone without any other findings and large PEs are often missed because they are painless and mimic other conditions often causing EKG changes and small rises in troponin and BNP levels.[3]

PEs are sometimes described as massive, submassive and nonmassive depending on the clinical signs and symptoms. Although the exact definitions of these are unclear, a generally accepted definition of massive PE is one in which there is hemodynamic instability such as sustained hypotension, bradycardia or pulselessness.[4]

Risk factors [edit]


A deep vein thrombosis as seen in the right leg is a risk factor for PE

The most common sources of embolism are proximal leg deep vein thromboses (DVTs) or pelvic vein thromboses. Any risk factor for DVT also increases the risk that the venous clot will dislodge and migrate to the lung circulation, which may happen in as many as 15% of all DVTs.[*citation needed*] The conditions are generally regarded as a continuum termed *venous thromboembolism* (VTE).

The development of thrombosis is classically due to a group of causes named Virchow's triad (alterations in blood flow, factors in the vessel wall and factors affecting the properties of the blood). Often, more than one risk factor is present.

- *Alterations in blood flow*: immobilization (after surgery, injury, pregnancy (also procoagulant), obesity (also procoagulant), cancer (also procoagulant)
- *Factors in the vessel wall*: surgery, catheterizations causing direct injury ("endothelial injury")
- *Factors affecting the properties of the blood* (procoagulant state):
  - Estrogen-containing hormonal contraception
  - Genetic thrombophilia (factor V Leiden, prothrombin mutation G20210A, protein C deficiency, protein S deficiency, antithrombin deficiency, hyperhomocysteinemia and plasminogen/fibrinolysis disorders)
  - Acquired thrombophilia (antiphospholipid syndrome, nephrotic syndrome, paroxysmal nocturnal hemoglobinuria)
  - Cancer (due to secretion of pro-coagulants)

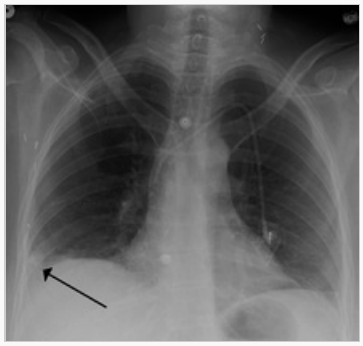
Underlying causes [edit]

After a first PE, the search for secondary causes is usually brief. Only when a second PE occurs, and especially when this happens while still under anticoagulant therapy, a further search for underlying conditions is undertaken. This will include testing ("thrombophilia screen") for Factor V Leiden mutation, antiphospholipid antibodies, protein C and S and antithrombin levels, and later prothrombin mutation, MTHFR mutation, Factor VIII concentration and rarer inherited coagulation abnormalities.[*citation needed*]

Diagnosis [edit]

To diagnose pulmonary embolism, medical societies recommend a review of clinical criteria to determine the need for testing, followed by testing to determine a likelihood of being able to confirm a diagnosis by imaging, followed by imaging if other tests have shown that there is a likelihood of a PE diagnosis.[5][6][7]

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, followed by an assessment of clinical probability.[1]



A Hampton hump in a person with a right lower lobe pulmonary embolism

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. In 1995, Philip Steven Wells, initially developed a prediction rule (based on a literature search) to predict the likelihood of PE, based on clinical criteria.[8] The prediction rule was revised in 1998[9] This prediction rule was further revised when simplified during a validation by Wells *et al.* in 2000.[10] In the 2000 publication, Wells proposed two different scoring systems using cutoffs of 2 or 4 with the same prediction rule.[10] In 2001, Wells published results using the more conservative cutoff of 2 to create three categories.[11] An additional version, the "modified extended version", using the more recent cutoff of 2 but including findings from Wells's initial studies[8][9] were proposed.[12] Most recently, a further study reverted to Wells's earlier use of a cutoff of 4 points[10] to create only two categories.[13]

There are additional prediction rules for PE, such as the Geneva rule. More importantly, the use of *any* rule is associated with reduction in recurrent thromboembolism.[14]

The Wells score:[15]

- clinically suspected DVT — 3.0 points
- alternative diagnosis is less likely than PE — 3.0 points
- tachycardia (heart rate > 100) — 1.5 points
- immobilization (≥ 3d)/surgery in previous four weeks — 1.5 points
- history of DVT or PE — 1.5 points
- hemoptysis — 1.0 points
- malignancy (with treatment within 6 months) or palliative — 1.0 points

Traditional interpretation[10][11][16]

- Score >6.0 — High (probability 59% based on pooled data[17])
- Score 2.0 to 6.0 — Moderate (probability 29% based on pooled data[17])
- Score <2.0 — Low (probability 15% based on pooled data[17])

Alternative interpretation[10][13]

- Score > 4 — PE likely. Consider diagnostic imaging.
- Score 4 or less — PE unlikely. Consider D-dimer to rule out PE.

Blood tests [edit]

In people with a low or moderate suspicion of PE, a normal D-dimer level (shown in a blood test) is enough to exclude the possibility of thrombotic PE, with a three-month risk of thromboembolic events being 0.14%.[18] D-dimer is highly sensitive but not very specific (specificity around 50%). In other words, a positive D-dimer is not synonymous with PE, but a negative D-dimer is, with a good degree of certainty, an indication of absence of a PE.[19] The typical cut off is 500 ug/L.[20] However, in those over the age of 50, changing the cut-off value to the persons age multiplied by 10 ug/L decreases the number of falsely positive tests without missing any additional cases of PE.[20]

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.[*citation needed*]

Imaging [edit]

In typical people who are not known to be at high risk of PE, imaging is helpful to confirm or exclude a diagnosis of PE after simpler first-line tests are used.[5][6][21] Medical societies recommend tests such as the D-dimer to

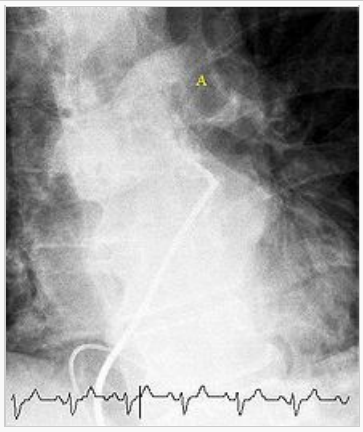
first provide supporting evidence for the need for imaging, and imaging would be done if other tests confirmed a moderate or high probability of finding evidence to support a diagnosis of PE.[6][21]

CT pulmonary angiography is the recommended first line diagnostic imaging test in most people.[22] Historically, the gold standard for diagnosis was pulmonary angiography, but this has fallen into disuse with the increased availability of non-invasive techniques.[*citation needed*]

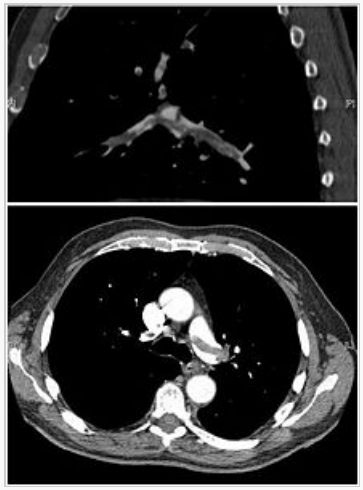
Non-invasive imaging [edit]

CT pulmonary angiography (CTPA) is a pulmonary angiogram obtained using computed tomography (CT) with radiocontrast rather than right heart catheterization. Its advantages are clinical equivalence, its non-invasive nature, its greater availability to people, 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 (MDCT) machines.[23] According to a cohort study, single-slice spiral CT may help diagnose detection among people with suspected pulmonary embolism.[24] In this study, the sensitivity was 69% and specificity was 84%. In this study which had a prevalence of detection was 32%, the positive predictive value of 67.0% and negative predictive value of 85.2% (click here to adjust these results for people at higher or lower risk of detection). However, this study's results may be biased due to possible incorporation bias, since the CT scan was the final diagnostic tool in people with pulmonary embolism. The authors noted that a negative single slice CT scan is insufficient to rule out pulmonary embolism on its own. A separate study with a mixture of 4 slice and 16 slice scanners reported a sensitivity of 83% and a specificity of 96%. This study noted that additional testing is necessary when the clinical probability is inconsistent with the imaging results.[25] CTPA is non-inferior to VQ scanning, and identifies more emboli (without necessarily improving the outcome) compared to VQ scanning.[26]

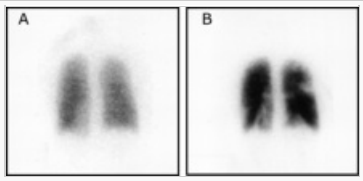
A ventilation/perfusion scan (or V/Q scan or lung scintigraphy) shows that some areas of the lung are being ventilated but not perfused with blood (due to obstruction by a clot). This type of examination is as accurate as multislice CT, but is less used, due to the greater availability of CT technology. It is particularly useful in people who have an allergy to iodinated contrast, impaired renal function, or are pregnant (due to its lower radiation exposure as compared to CT).[27][28] The test can be performed with planar two-dimensional imaging, or single photon emission tomography (SPECT) which enables three-dimensional imaging.[22] Hybrid devices combining SPECT and CT (SPECT/CT) further enable anatomic characterization of any abnormality.



Selective pulmonary angiogram revealing significant thrombus (labelled A) causing a central obstruction in the left main pulmonary artery. ECG tracing shown at bottom.



CT pulmonary angiography (CTPA) showing a "saddle embolus" at the bifurcation of the pulmonary artery and substantial thrombus burden in the lobar branches of both main pulmonary arteries.



Ventilation-perfusion scintigraphy in a woman taking hormonal contraceptives and valdecoxib.
(A) After inhalation of 20.1 mCi of Xenon-133 gas, scintigraphic images were obtained in the posterior projection, showing uniform ventilation to lungs.
(B) After intravenous injection of 4.1 mCi of Technetium-99m-labeled macroaggregated albumin, scintigraphic images were obtained, shown here in the posterior projection. This and other views showed decreased activity in multiple regions.

Low probability diagnostic tests/non-diagnostic tests [edit]

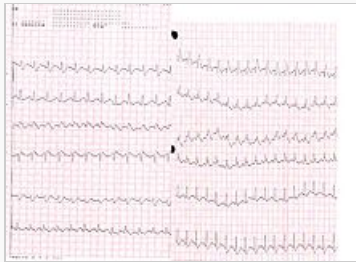
Tests that are frequently done that are not sensitive for PE, but can be diagnostic.

- Chest X-rays are often done on people with shortness of breath to help rule-out other causes, such as congestive heart failure and rib fracture. Chest X-rays in PE are rarely normal,[29] but usually lack signs that suggest the diagnosis of PE (e.g. Westermark sign, Hampton's hump).
- Ultrasonography of the legs, also known as leg doppler, in search of deep venous thrombosis (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). This may be valid approach in pregnancy, in which the other modalities would increase the risk of birth defects in the unborn child.

However, a negative scan does not rule out PE, and low-radiation dose scanning may be required if the mother is deemed at high risk of having pulmonary embolism.[*citation needed*]

Electrocardiogram [edit]

The primary use of the ECG is to rule out other causes of chest pain.[30] An electrocardiogram (ECG) is routinely done on people with chest pain to quickly diagnose myocardial infarctions (heart attacks), an important differential diagnosis in an individual with chest pain. While certain ECG changes may occur with PE, none are specific enough to confirm or sensitive enough to rule out the diagnosis.[30] 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), which occurs in 12-50% of people with the diagnosis, yet also occurs in 12% without the diagnosis.[31][32]



Electrocardiogram of a person with pulmonary embolism, showing sinus tachycardia of approximately 150 beats per minute and right bundle branch block.

This is occasionally present (occurring in up to 20% of people), but may also occur in other acute lung conditions, and, therefore, has limited diagnostic value. The most commonly-seen signs in the ECG are sinus tachycardia, right axis deviation, and right bundle branch block.[33] Sinus tachycardia, however, is still only found in 8–69% of people with PE.[34]

Echocardiography [edit]

In massive and submassive PE, dysfunction of the right side of the heart may be seen on echocardiography, an indication that the pulmonary artery is severely obstructed and the right ventricle, a low pressure pump, is unable to match the pressure. Some studies (see below) suggest that this finding may be an indication for thrombolysis. Not every person 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,[35] and be important in prognosis.[36]

The specific appearance of the right ventricle on echocardiography is referred to as the *McConnell's 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 in the setting of right ventricular dysfunction.[37]

Algorithms [edit]

Probability testing [edit]

Recent recommendations for a diagnostic algorithm have been published by the PIOPED investigators; however, these recommendations do not reflect research using 64 slice MDCT.[17] These investigators recommended:

- Low clinical probability. If negative D-dimer, PE is excluded. If positive D-dimer, obtain MDCT and based treatment on results.
- Moderate clinical probability. If negative D-dimer, PE is excluded. *However*, the authors were not concerned that a negative MDCT with negative D-dimer in this setting has an 5% probability of being false. Presumably, the 5% error rate will fall as 64 slice MDCT is more commonly used. If positive D-dimer, obtain MDCT and based treatment on results.
- High clinical probability. Proceed to MDCT. If positive, treat, if negative, additional tests are needed to exclude PE.

Pulmonary embolism rule-out criteria [edit]

The pulmonary embolism rule-out criteria (PERC) help assess people in whom pulmonary embolism is suspected, but unlikely. Unlike the Wells score and Geneva score, which are clinical prediction rules intended to risk stratify patients with suspected PE, the PERC rule is designed to rule out risk of PE in patients when the physician has already stratified them into a low-risk category.

People in this low risk category without any of these criteria may undergo no further diagnostic testing for PE: Hypoxia — Sa_{O_2} <95%, unilateral leg swelling, hemoptysis, prior DVT or PE, recent surgery or trauma, age >50, hormone use, tachycardia. The rationale behind this decision is that further testing (specifically CT angiogram of the chest) may cause more harm (from radiation exposure and contrast dye) than the risk of PE.[38] The PERC rule has a sensitivity of 97.4% and specificity of 21.9% with a false negative rate of 1.0% (16/1666).[39]

Prevention [edit]

Pulmonary embolism may be preventable in those with risk factors. For instance, people admitted to hospital may receive preventative medication and anti-thrombosis stockings to reduce the risk.[40]

Following the completion of warfarin in those with prior PE, long term aspirin is useful to prevent re occurrence.[41][42]

Treatment [edit]

Anticoagulant therapy is the mainstay of treatment. Acutely, supportive treatments, such as oxygen or analgesia, may be required. People are often admitted to hospital in the early stages of treatment, and tend to remain under inpatient care until the INR has reached therapeutic levels. Increasingly, however, low-risk cases are managed at home in a fashion already common in the treatment of DVT.[43] Evidence to support one approach versus the other is weak.[44]

Anticoagulation [edit]

In most cases, anticoagulant therapy is the mainstay of treatment. Unfractionated heparin, low molecular weight heparin (LMWH), or fondaparinux is administered initially, while warfarin, acenocoumarol, or phenprocoumon therapy is commenced (this may take several days, usually while the patient is in the hospital). LMWH may reduce bleeding among people with pulmonary embolism as compared to heparin according to a systematic review of randomized controlled trials by the Cochrane Collaboration.[45] The relative risk reduction was 40%. For people at similar risk to those in this study (2.0% had bleeding when not treated with low molecular weight heparin), this leads to an absolute risk reduction of 0.8%. 125 people must be treated for one to benefit.

Warfarin therapy often requires frequent dose adjustment and monitoring of the international normalized ratio (INR). In PE, INRs between 2.0 and 3.0 are generally considered ideal. If another episode of PE occurs under warfarin treatment, the INR window may be increased to e.g. 2.5–3.5 (unless there are contraindications) or anticoagulation may be changed to a different anticoagulant e.g. LMWH.[*citation needed*]

In patients with an underlying malignancy, therapy with a course of LMWH is favored over warfarin; it is continued for six months, at which point a decision should be reached as to whether ongoing treatment is required.[46]

Similarly, pregnant women are often maintained on low molecular weight heparin until at least 6 weeks after delivery to avoid the known teratogenic effects of warfarin, especially in the early stages of pregnancy.[47]

Warfarin therapy is usually continued for 3–6 months, or "lifelong" if there have been previous DVTs or PEs, or none of the usual risk factors is present. An abnormal D-dimer level at the end of treatment might signal the need for continued treatment among patients with a first unprovoked pulmonary embolus.[48] For those with small PEs (known as subsegmental PEs) the effects of anticoagulation is unknown as it has not been properly studied as of 2014.[49]

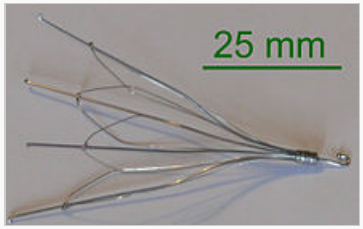
Thrombolysis [edit]

Massive PE causing hemodynamic instability (shock and/or hypotension, defined as a systolic blood pressure <90 mmHg or a pressure drop of 40 mmHg for >15 min if not caused by new-onset arrhythmia, hypovolemia or sepsis) is an indication for thrombolysis, the enzymatic destruction of the clot with medication. In this situation it is the best available treatment in those without contraindications and is supported by clinical guidelines.[7][46][50]

The use of thrombolysis in non-massive PEs is still debated.[51][52] Some have found that the treatment decreases the risk of death and increases the risk of bleeding including intracranial hemorrhage.[53] Others have found no decrease in the risk of death.[52]

Inferior vena cava filter [edit]

If anticoagulant therapy is contraindicated (e.g. shortly after a major operation), an inferior vena cava filter may be implanted to prevent new emboli from entering the pulmonary artery and combining with an existing blockage.[46] It should be removed as soon as it becomes safe to start using anticoagulation.[46]



Used inferior vena cava filter.

Surgery [edit]

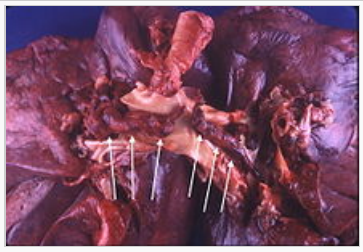
Surgical management of acute pulmonary embolism (pulmonary thrombectomy) is uncommon and has largely been abandoned because

of poor long-term outcomes. However, recently, it has gone through a resurgence with the revision of the surgical technique and is thought to benefit certain people.[54] Chronic pulmonary embolism leading to pulmonary hypertension (known as *chronic thromboembolic hypertension*) is treated with a surgical procedure known as a pulmonary thromboendarterectomy.

Epidemiology [edit]

Pulmonary embolisms occur in more than 600,000 people in the United States each year.[55] It results in between 50,000[55] and 200,000 deaths per year in the United States.[56] The risk in those who are hospitalized is around 1%.[57] The rate of fatal pulmonary embolisms has declined from 6% to 2% over the last 25 years in the United States.[56]

Prognosis [edit]


Large saddle embolus seen in the pulmonary artery (white arrows).

Mortality from untreated PE is said to be 26%. This figure comes from a trial published in 1960 by Barrit and Jordan,[58] which compared anticoagulation against placebo for the management of PE. Barritt and Jordan performed their study in the Bristol Royal Infirmary in 1957. This study is the only placebo controlled trial ever to examine the place of anticoagulants in the treatment of PE, the results of which were so convincing that the trial has never been repeated as to do so would be considered unethical. That said, the reported mortality rate of 26% in the placebo group is probably an overstatement, given that the technology of the day may have detected only severe PEs. More recent evidence suggests that up to 10% of symptomatic PEs are fatal within the first hour of symptoms.[7]

There are a number of markers used for risk stratification and these are also independent predictors of adverse outcome. These include hypotension, cardiogenic shock, syncope, evidence of right heart dysfunction, and elevated cardiac enzymes.[7] A number of ECG changes including S1Q3T3 also correlate with worse short-term prognosis.[4] There have been a number of other patient-related factors such as COPD and chronic heart failure thought to also play a role in prognosis.[7]

Prognosis depends on the amount of lung that is affected and on the co-existence of other medical conditions; chronic embolisation to the lung can lead to pulmonary hypertension. After a massive PE, the embolus must be resolved somehow if the patient is to survive. In thrombotic PE, the blood clot may be broken down by fibrinolysis, or it may be organized and recanalized so that a new channel forms through the clot. Blood flow is restored most rapidly in the first day or two after a PE.[59] Improvement slows thereafter and some deficits may be permanent. There is controversy over whether small subsegmental PEs need to be treated at all[60] and some evidence exists that patients with subsegmental PEs may do well without treatment.[25][61]

Once anticoagulation is stopped, the risk of a fatal pulmonary embolism is 0.5% per year.[62]

Predicting mortality [edit]

The PESI can estimate mortality of patients. The Geneva prediction rules and Wells criteria are used to calculate a pre-test probability of patients to predict who has a pulmonary embolism. These scores are tools to be used in conjunction with clinical judgement in deciding diagnostic testing and types of therapy.[63]

References [edit]

1. ^ [a] [b] [c] [d] [e] [f] Goldhaber SZ (2005). "Pulmonary thromboembolism". In Kasper DL, Braunwald E, Fauci AS et al. *Harrison's Principles of Internal Medicine* (16th ed.). New York, NY: McGraw-Hill. pp. 1561–65. ISBN 0-07-139140-1.
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