

Diagnosis of Pulmonary Embolism

Lynn M. Cloutier, RN, MSN, ACNP, AOCN®

Case 1: A 77-year-old Caucasian woman with a probable diagnosis of ovarian cancer developed an acute onset of shortness of breath two days after being discharged for a workup of symptomatic ascites. Her medical history was significant for hypertension and atrial fibrillation, and she was taking therapeutic doses of coumadin. She presented to the emergency department; was found to have a large, left pleural effusion; and was admitted to the hospital. A thoracentesis removed 2.1 L of fluid. The patient's respiratory distress improved; however, she experienced a second episode of sudden onset shortness of breath prior to her anticipated discharge. She was afebrile, with a room air pulse oximetry of 80%, pulse of 121 beats per minute, and respiratory rate of 28. A chest x-ray (CXR) showed only a small pleural effusion remaining on the left. An electrocardiogram (EKG) showed atrial fibrillation. A chest computed tomography (CT) was performed and showed bilateral pulmonary emboli.

Case 2: A 57-year-old, obese, African American woman with newly diagnosed endometrial cancer presented to clinic with a palpable left supraclavicular lymph node. A CXR revealed mediastinal adenopathy, and a chest CT was performed to further delineate the size of the mass and evaluate for metastases. The CT revealed a Hampton's hump and also was positive for pulmonary embolism (PE). The patient's vital signs were normal, and she did not have any respiratory symptoms. An ultrasound of her lower extremities was negative for deep vein thrombosis (DVT).

Case 3: A 31-year-old Caucasian woman receiving chemoradiation for cervical cancer was admitted to the hospital with diffuse abdominal pain that occurred two days after completion of brachytherapy with after-loading tandem and ovoids, a treatment requiring complete bed rest for 48 hours. On admission, further review of systems was negative; however, the patient mentioned that she lived in a two-story house and had shortness of breath after climbing stairs—a symptom she attributed to her body habitus and anemia. Her height was 162.5 cm, and her weight was 135.9 kg. During the

patient's hospitalization, she developed a rapid heart rate of 139 beats per minute. An EKG showed sinus tachycardia, and a CXR revealed atelectasis and diminished lung volumes. Laboratory evaluation revealed leukocytosis with a white blood cell count of 21.5/mm³, and a cardiac panel was negative. Her room air pulse oximetry was 100%. Following a diagnosis of pneumonia, the patient developed respiratory distress, was intubated, and was transported to the intensive care unit. Continued workup with a chest CT revealed bilateral pulmonary emboli and pulmonary metastases.

Case 4: A 58-year-old Caucasian woman with a history of cervical cancer underwent surgical debulking after an aborted pelvic exenteration. She developed a wound dehiscence and pelvic abscesses postoperatively. A pelvic drain was placed, and she was admitted to the hospital for IV antibiotic therapy. During her hospitalization, she developed a second surface abscess and a CT of the

abdomen and pelvis was performed to delineate the extent of the abscess and to reassess the initial abscess. The CT showed an incidental finding of a pulmonary embolus in the right lower lobe, which was confirmed by chest CT. The patient was completely asymptomatic. She never displayed shortness of breath, she did not require oxygen, and her pulse rate was normal.

These patients all have a diagnosis of cancer or an anticipated diagnosis of cancer and a diagnosis of PE. Their cases are more different than similar, which illustrates how the presentation of PE is sometimes unusual, often varied, and usually found incidentally.

Discussion and Analysis

PE is an extremely common and highly lethal condition that is a leading cause of death in all age groups and the first- or second-most common cause of unexpected death in most age groups.

Lynn M. Cloutier, RN, MSN, ACNP, AOCN®, is a nurse practitioner at the University of Texas M.D. Anderson Cancer Center in Houston.

Digital Object Identifier: 10.1188/07.CJON.343-348

PE is the most serious complication of venous thrombosis and the third-most common cardiovascular problem after coronary artery disease and stroke (Stein & Firth, 2003). More than 400,000 cases are undiagnosed in the United States annually, resulting in 100,000 patient deaths (Feied, 2002). PE is associated with 300,000–600,000 hospitalizations each year (Garg, 2005).

Prompt diagnosis and treatment of PE can dramatically reduce morbidity and mortality; therefore, a workup should be initiated as soon as any suspicion exists. Unfortunately, the condition is undiagnosed far more often than it is diagnosed because patients with PE often present with vague and nonspecific complaints (Feied, 2002; Kline & Runyon, 2006) and classic symptoms are not present in many cases (Goldhaber, 2004). PE is not an isolated disease of the chest, but a complication of DVT (see Figure 1). DVT and PE are part of the same venous thromboembolism process (Riedel, 2004). Although as many as 90% of pulmonary emboli arise from the veins of the lower extremities, many patients with PE have no signs or symptoms of DVT (Desai, 2002; Stein & Firth, 2003).

In approximately 25% of patients with PE, the initial clinical manifestation is sudden death (Heit, 2006).

Virchow described PE in the mid-1800s, when he showed how venous blood clots travel to the pulmonary artery and form emboli. He noted that the clinical manifestation of PE was asphyxiation, caused by blockage of the main trunks of the pulmonary artery. He proposed that all primary thromboembolism resulted from three factors known as Virchow's triad—stasis of blood flow, injury to the vein, and hypercoagulability (Cardin & Marinelli, 2004; Dalen, 2002; Riedel, 2004). At least one component of the triad is present in all categories of risk factors.

Risk Factors

The assessment of PE begins with a careful physical examination and determination of risk factors (see Figure 2) (Ramzi & Leeper, 2004). Most patients with PE have a combination of risk factors (Dalen, 2002; Desai, 2002). Immobility, irrespective of the cause, is the most frequent predisposing factor. Immobilization for even one or two days may predis-

pose a patient for PE, and most patients who develop PE are immobilized for less than two weeks (Stein & Firth, 2003).

Pathophysiology

PE may result from the blockage of an artery in the lungs by fat, air, a tumor, or a blood clot. This discussion will focus on blood clots. Normally, microscopic clots are formed and destroyed constantly in venous circulation, allowing for local hemostasis in response to an injury without enabling a clot to form. When the system is disrupted, microthrombi escape the normal fibrinolytic system. Most clots originate in the lower extremities; the clots may build in the venous system, break loose, and travel in the venous system through the vena cava and into the right side of the heart, causing blockage of the pulmonary blood vessels (Feied, 2002; Kline & Runyon, 2006).

Presentation

The presentation of patients with PE can vary greatly (see Table 1). Young, previously healthy patients with excellent cardiac reserve may have subtle signs and symptoms. PE tends to mimic other illnesses (Goldhaber, 2004). The classic triad of symptoms, which occur in fewer than 20% of patients, are dyspnea, often with accompanying tachypnea (> 20 per minute); hemoptysis; and chest pain (pleuritic or substernal) (Riedel, 2004). PE is so lethal that the diagnosis should be sought actively in every patient who presents with any chest symptom that cannot be proven to have another cause (see Figure 3).

Clinical Evaluation

Prior to the development of diagnostic imaging techniques, the diagnosis of PE was based on dyspnea, hemoptysis, and chest pain (Colp & Stein, 2001). Currently, the diagnosis can be made through a combined approach of physical examination, assessment of risk factors, and imaging studies.

Clinicians should assign a pretest probability for PE before proceeding with objective testing (Hyers, 2000). Wells et al.'s (2001) clinical decision rule is based on a seven-feature assessment, assigning points based on physical examination and



Figure 1. Deep Vein Thrombosis Caused By Venous Stasis, Resulting in Pulmonary Embolism

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Air travel—greatest risk at distances > 5,000 km or 3,100 miles	Lower-extremity paralysis
Atrial fibrillation	Lupus anticoagulant
Burns	Major trauma
Cancer	Myocardial infarction
Cardiomyopathy	Nephrotic syndrome
Childbirth	Obesity
Disseminated intravascular coagulation	Oral contraceptive use
Estrogen or hormone replacement therapy	Pregnancy
Family history of deep vein thrombosis or pulmonary embolism	Personal history of deep vein thrombosis or pulmonary embolism
Fracture of the hip or femur	Prolonged bed rest or inactivity
Genetic or acquired hematologic disorders	Prosthetic cardiac valves
Cardiac surgery	Recent surgery, especially for cancer
Immobilization	Reduced cardiac output
Increasing age	Sickle cell anemia
Indwelling venous catheters	Smoking
Large pelvic tumors	Stroke

Figure 2. Risk Factors for Pulmonary Embolism

history. The tool can be used to rapidly assess the clinical probability of PE (see Table 2). A diagnosis of PE is unlikely in patients given a score of four points or less (van Belle et al., 2006). Within that group, about 5% of patients will be diagnosed with PE.

Physical Examination

Physical examination of patients with suspected PE should be performed in a systematic, thorough manner because the clinical picture varies widely. Some patients may present only with a change in vital signs; others are dyspneic, hypoxic, acutely ill, and unstable. If the presentation is not obvious, astute clinicians will look for subtle changes in patients' conditions. Tachypnea, tachycardia, and a complaint of chest pain may be present. Auscultation of the lungs may elicit a pleural friction rub or wheeze. A cough with blood-tinged sputum may be present (Stein & Firth, 2003). Clinicians should examine patients for a positive Homan's sign and evidence of DVT, which includes extremity swelling, pain, redness, or a palpable cord of a thrombosed vein. Patients may exhibit anxiety or cyanosis because of hypoxemia and have a low room-air pulse oximetry. Further evaluation with an arterial blood gas is recommended.

Pulse Oximetry

Pulse oximetry is not helpful in the diagnosis of PE but will identify patients

who require supplemental oxygen and monitor patients who have a drop in oxygen saturation (Feied, 2002; Kline & Runyon, 2006). Room air pulse oximetry will fall lower than 92% in most patients with PE. Patients with a room air pulse oximetry of 95% or more at diagnosis of PE will have a significantly lower probability of in-hospital complications (Kline et al., 2003).

Arterial Blood Gas

Arterial blood gas should be performed to help establish a PE diagnosis. Characteristic changes associated with PE include a reduced arterial oxygen pressure and an arterial carbon dioxide pressure that is normal or reduced because of hyperventilation. Arterial oxygen pressure may be normal in patients with a minor pulmonary embolus but is almost never normal in patients with massive pulmonary emboli when more than 50% of the pulmonary circulation is obstructed suddenly. Widening of the alveolar to arterial gradient (> 20 mm/Hg) may be more sensitive than arterial oxygen pressure alone (Dalen, 2002; Hyers, 2000; Riedel, 2004). Although hypoxemia and respiratory alkalosis are nonspecific, they are the classic findings on arterial blood gas. A normal arterial oxygen pressure is found in 15% of patients, and a normal alveolar to arterial gradient is found in 10%-15% of patients (Desai, 2002).

Electrocardiogram and Chest X-Ray

An EKG and CXR should be performed in all patients to support the clinical suspicion of PE and exclude alternative

Table 1. Prevalence of Presenting Signs and Symptoms of Pulmonary Embolism

SIGN OR SYMPTOM	PRESENTATION (%)
Sudden onset of shortness of breath at rest or with exertion; dyspnea or worsening of chronic dyspnea	73–84
Tachypnea	70–96
Chest pain under the breastbone or on one side	66–90
• Pleuritic	66–74
• Nonpleuritic	14
Rales	51–58
Unexplained anxiety	50–65
Fever	43
Cough of sudden onset	37–73
Third or fourth heart sound gallop	34
Signs and symptoms of thrombophlebitis	32
Tachycardia (heart rate > 100 beats per minute)	30–50
Lower-extremity edema	24–28
Cardiac murmur	23
Cyanosis	19
Hemoptysis	13–30
Diaphoresis	11–36
New onset of wheezing	9
Syncope	5–13

Note. Other presenting symptoms have been reported, including decreased pulse oximetry less than 92% on room air, splinting of ribs with breathing, lightheadedness, dizziness, and new cardiac arrhythmia.

Note. Based on information from Desai, 2002; Feied, 2002; Stein & Firth, 2003.

Acute coronary syndrome
 Angina
 Asthma
 Bronchitis
 Carcinoma
 Chronic obstructive pulmonary disease
 Congestive heart failure
 Myocardial infarction
 Pleurisy or pleuritis
 Pneumonia
 Pneumothorax

Figure 3. Differential Diagnosis for Pulmonary Embolism

diagnoses (Riedel, 2004). EKG findings are abnormal, but nonspecific, in as many as 70% of patients with PE and may be normal in young, previously healthy patients (Dalen, 2002; Piazza & Goldhaber, 2006; Ramzi & Leeper, 2004). In patients with a minor pulmonary embolus, the EKG may show only sinus tachycardia or nonspecific ST-T wave. A normal EKG is very unusual in patients with an acute pulmonary embolus. An EKG also can be used to rule out other diagnoses, such as myocardial infarction or pericarditis (Desai, 2002; Goldhaber, 2004; Riedel, 2004). Twenty-five percent of patients with a proven pulmonary embolus have EKGs that are unchanged from baseline (Feied, 2002).

A CXR often is the first imaging procedure that is obtained when patients present with dyspnea. A normal film in patients with severe, acute dyspnea without wheezing is very suspicious for PE. The CXR is more helpful in excluding other conditions that mimic PE, such as pneumothorax, pneumonia, left-sided heart failure, tumor, rib fracture, massive pleural effusion, and lobar collapse (Riedel, 2004).

Three rare findings indicative of PE (i.e., Westermark sign, Hampton's hump, and Palla's sign) may be present on CXR (Piazza & Goldhaber, 2006). Westermark sign is the focal area of decreased pulmonary vasculature, which commonly occurs when large emboli affect a lobe or entire lung or in patients with pulmonary embolus complicated by infarction of the lung (Dalen, 2002). Hampton's hump is a peripheral wedge-shaped, pleural-based density with apex pointing to the hilum. Palla's sign is the presence of an enlarged, right, descending pulmonary artery.

D-Dimer

D-dimer is a final product of fibrin degradation. A positive serum D-dimer indicates that high levels of fibrin degradation products are present in the blood. Fibrin degradation products are released into the blood as a result of fibrinolysis, which occurs with significant clot formation and degradation or because of an endovascular thrombus (Neff, 2003). The enzyme-linked immunosorbent assay method is the preferred testing process. Almost all patients with a proven pulmonary embolus will have a D-dimer level of more than 500 ng/ml (Desai, 2002).

Measurement of D-dimer is an excellent test to rule out the diagnosis of PE. A negative test is sensitive in low-risk patients, and the workup for PE can stop if the D-dimer level is normal. A normal D-dimer can exclude PE in more than 90% of cases (Martino et al., 2005); however, the test is nonspecific and cannot be used alone to establish the diagnosis. An elevated D-dimer level may be seen in hospitalized patients, especially patients with cancer and those who recently had surgery. Elevated levels also may be seen in patients with inflammatory diseases, pneumonia, myocardial infarction, sepsis, pregnancy, trauma, and disseminated intravascular coagulation (Riedel, 2004; Schoepf, Goldhaber, & Costello, 2004). Further confirmatory testing is required in the setting of a positive D-dimer test (Ebell, 2004; Rathbun, Whitsett, & Raskob, 2004).

Diagnostic Imaging

The confirmation or exclusion of PE may be achieved through multiple diagnostic mechanisms and imaging studies (see Figure 4). The diagnostic tool most appropriate for a particular case should be determined by patients' medical histories, allergy profiles, and conditions.

Ventilation and Perfusion Scan

The ventilation and perfusion (V/Q) scan consists of two sequential tests. The perfusion scan shows the distribution of microvascular blood flow in the lungs after IV injection of a radionuclide, and the ventilation scan shows distribution of ventilation in the small airways after inhalation of a radioactive gas or aerosol

(Hyers, 2000). V/Q scanning does not require IV contrast and therefore is the imaging modality of choice for patients with major renal impairment, anaphylaxis to IV contrast, and pregnancy (Piazza & Goldhaber, 2006).

V/Q scanning is an indirect study of embolism because it shows perfusion abnormality. Defects in tracer uptake when inhaled into the lungs are reported as normal, near normal, or indicating low, moderate, or high probability for PE (Ramzi & Leeper, 2004). In 1990, the Prospective Investigation of Pulmonary Embolism Diagnosis ([PIOPED], 1990) trial, a multi-institutional study of V/Q scanning and pulmonary angiography, assessed diagnostic specificity and sensitivity of V/Q scans for the diagnosis of PE. The study revealed that a V/Q scan with normal findings virtually excludes PE because occlusive PE of all types produces a defect of perfusion. A V/Q scan with high-probability findings is virtually diagnostic for PE and mandates treatment.

Although V/Q scanning is sensitive enough to serve as a screening test, specificity is limited. A low or indeterminate

Table 2. Pretesting Assessment for Probability of Pulmonary Embolism

POINTS	POSITIVE FEATURES
3	Clinical signs and symptoms of deep vein thrombosis
3	Alternative diagnosis less likely than pulmonary embolism
1.5	Heart rate greater than 100 beats per minute
1.5	Immobility or surgery in the previous four weeks
1.5	Previous venous thromboembolism
1	Hemoptysis
1	Malignancy (currently receiving treatment, including palliative therapy; patient received treatment in the past six months.)

Note. Based on information from Neff, 2003; Ramzi & Leeper, 2004; van Belle et al., 2006; Wells et al., 2001.

study does not rule out a diagnosis of PE, and further evaluation should be undertaken (Ramzi & Leeper, 2004; Riedel, 2004). A V/Q scan often is not diagnostic in patients with chronic pulmonary disease or pleural effusion (Ebell, 2004).

Pulmonary Angiogram

Pulmonary angiogram is considered the gold standard for the diagnosis of PE to which sensitivity and specificity of all other radiographic methods for diagnosis of PE have been compared. Pulmonary angiography is an invasive imaging procedure of the body's blood vessels and is unavailable in many clinical settings. It requires placement of a catheter from a peripheral vein, either in the arm or the groin, which is threaded through the vascular system to the right-sided cardiac structures and into the main pulmonary artery. Contrast dye is injected through the catheter, allowing the multiple vessels of the pulmonary system to be visible on x-ray.

Pulmonary angiography should be considered only if all other imaging studies are inconclusive. Passage of the catheter through the right ventricle may provoke nonsustained ventricular tachycardia. Patients feel hot and flushed when the contrast media is injected and also may experience transient hypotension and coughing spasms (Goldhaber, 2005). A negative test essentially excludes the diagnosis of PE (Desai, 2002). Pulmonary angiography is contraindicated in patients who are pregnant and those who have a significant bleeding risk, renal insufficiency, or a known right-sided heart thrombus (Riedel, 2004).

Spiral Computed Tomography Angiography and Chest Computed Tomography Angiogram

High-resolution helical (spiral) CT angiography is gaining wide acceptance as first-line imaging for the detection of PE. The sensitivity and specificity of the test may be as high as 100% (Cobelli et al., 2005). Following the injection of contrast, the entire chest can be scanned for abnormalities. Intraluminal filling defects are identified in segmental or pulmonary arteries, and scanning may diagnose other abnormalities in as many as two-thirds of patients when

PE is absent (Ramzi & Leeper, 2004). CT angiography can resolve increasingly smaller diameter subsegmental pulmonary vessels without the need for invasive pulmonary artery catheters (Garg, 2005). PLOPED II studied patients with suspected PE and concluded that CT angiography was as accurate as invasive pulmonary angiography for the diagnosis of acute PE (Quiroz et al., 2005). Scans that are positive or negative to the level of subsegmental arteries are considered diagnostic, and no other imaging is necessary. A pulmonary angiogram is recommended for patients who have a normal scan and no evidence of lower extremity DVT (Martino et al., 2005; Neff, 2003; Riedel, 2004; Schoepf et al., 2004; Wittram, Maher, Halpern, & Shepard, 2005). Iodinated contrast agents are needed; therefore, CT angiography is contraindicated in patients with renal impairment and severe allergy to the contrast material.

Conclusion

PE often is asymptomatic, has varied presentations, may masquerade as other diseases, and may cause sudden death. Ruling out PE should be a priority to clinicians who believe it is the cause of patients' shortness of breath or tachycardia. If PE is not immediately fatal, the diagnosis should be made quickly and appropriate treatment initiated. Reducing the mortality rate from pulmonary embolus is connected to the prevention of and prophylaxis against venous thromboembolism. Preventive mechanisms include early mobility and ambulation after surgery, leg exercises while in bed and sitting for long periods of time, compression stockings, and sequential compression devices. Compression stockings must provide a gradient of 30–40 mm/Hg or more to be effective. These stockings provide the highest level of pressure at the toes and gradually decrease in pressure up to the level of the thigh (Feied, 2002). Prophylaxis can be obtained with fractionated or unfractionated heparin in individuals who must be confined to bed for long periods of time. Studies show that combinations of drug therapy and compression methods are more effective than either approach alone. Preventive therapy provides tenfold risk reduction

Pulmonary embolism is excluded by

- Normal pulmonary angiogram
- Normal perfusion scan
- Normal thin collimation (multidetector)
- CTPA [computed tomography pulmonary angiogram]
- Low probability perfusion scan and low clinical probability
- Normal D-dimer level (assay with high sensitivity) and low clinical probability
- Normal single detector spiral CTPA and compression ultrasonography (or computed tomography venography)
- Nondiagnostic lung scan and normal results on serial leg testing

Pulmonary embolism is confirmed by

- Intraluminal filling defect on pulmonary angiogram
- Intraluminal filling defect on spiral CTPA
- High probability scan and moderate or high clinical probability
- Apparative evidence of acute DVT [deep vein thrombosis] with nondiagnostic scan or spiral CTPA

Figure 4. Test Results That Effectively Exclude or Confirm Pulmonary Embolism

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for clinical events and death (Hyers, 2000). In a disease state that often is deadly, prevention is paramount.

Author Contact: Lynn M. Cloutier, RN, MSN, ACNP, AOCN®, can be reached at lcloutier@mdanderson.org, with copy to editor at CJONEditor@ons.org.

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