
Diagnosing and Treating Pulmonary Thromboembolism

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Citation

S Hamlin. *Diagnosing and Treating Pulmonary Thromboembolism*. The Internet Journal of Advanced Nursing Practice. 1998 Volume 3 Number 2.

Abstract

Pulmonary thromboembolism (PTE) is a difficult disease to diagnose and yet a missed diagnosis can be fatal. Presenting signs and symptoms mimic numerous conditions such as myocardial infarction and pneumonia. Diagnostic testing therefore is often extensive in an attempt to differentiate the diagnosis. Although pulmonary angiography is the gold standard for definitive diagnosis, it is seldom employed. Initial diagnostic testing often includes non-invasive studies such as lung scanning, echocardiography, and venous ultrasound along with specific laboratory data. Presenting condition as well as diagnostic results, suggest the severity of the disease process, and guide appropriate therapy.

INTRODUCTION

Pulmonary thromboembolism has been described as “the great masquerader” because of the illusive, nonspecific and varied symptoms upon presentation. The definitive modality for diagnosis is a pulmonary angiogram. However, most nurses and physicians would agree, angiography is not an appropriate initial diagnostic test because of cost and increased patient risk. It is therefore, difficult to diagnose and yet a missed diagnosis is often fatal. This article will introduce a patient presenting with pulmonary thromboembolism and discuss assessment, diagnosis and treatment of the disease.

The term pulmonary embolism (PE) is a broad term that includes embolism from many sources such as air, bone marrow, talc, amniotic fluid, arthroplasty cement, tumor, and sepsis.¹ This paper will focus on a thrombotic venous PE that has developed from a distal source such as the deep veins of the legs. The untreated mortality rate for PE is 30%, however only 8% if appropriate treatment is instituted, the challenge is that the disease is difficult to diagnose often because the symptoms are “silent”.²

CASE STUDY

CHIEF COMPLAINT

“My chest hurts and I feel so weak”.

PRESENT PROBLEM

Mr. Jones is a 58-year-old patient who awoke this morning feeling well. Mid-morning, while watching television, he began to feel tightness in his chest especially upon deep inspiration. A short while later “coughing spells” began without sputum production, as well as shortness of breath especially with exertion. Mr. Jones is now presenting to the emergency room (ER) six hours after symptoms began with complaints of extreme weakness and states “I just feel like there is something terribly wrong”. There is no recent history of illness, fever, cough or syncope.

PAST MEDICAL HISTORY

Mr. Jones is a widowed salesman with two grown children. He states he has experienced good health except for “a few aches and pains every now and then”. He does not have a primary physician, and recalls the last time he was examined by a physician was 15 years ago for a right knee replacement as a result of a football injury. Mr. Jones has no allergies, takes no prescription medications and is able to perform all activities of daily living. He takes Tylenol occasionally for pain in his right knee and leg. Family history is significant for his father and two brothers with heart disease and hypertension. Social and personal history is significant for smoking two packs per day for 22 years, sedentary life-style, and occasional “social” alcohol use.

PHYSICAL EXAMINATION

Mr. Jones is a 58-year-old moderately obese white male that appears in moderate distress. Vital signs are blood pressure 158/98 in both arms, heart rate 115, respiratory rate 34, temperature 38.1 C. Height is six foot two inches tall and weight is 265 pounds.

- Neurologic- speech is clear, thought process and memory are intact, orientation is to person, place and time. CN II-XII are intact.
- Integumentary- skin is pale and cool without diaphoresis. Capillary refill less than three seconds, no evidence of nail clubbing or cyanosis.
- HEENT- unremarkable.
- Lymphatic-unremarkable.

Pulmonary- symmetric chest excursion without use of accessory muscles. Respiratory rate regular at 35 breaths per minute of moderate depth and slightly laborious. Course crackles with diminished breath sounds throughout anterior and posterior chest, without friction rub, egophony or tactile fremitus.

Cardiovascular- apical impulse at fifth intercostal space mid-clavicular line with a regular apical rate of 120. S1, S2 and S3 heart sounds are present with a systolic murmur at left lower sternal boarder. No apparent S4 or pericardial rub. + JVD and 1+ pedal edema.

- Blood Vessels-temporal, carotid, and abdominal aorta arteries are without thrill or bruit.
- Gastrointestinal-unremarkable.
- Genitourinary-unremarkable.
- Musculoskeletal-unremarkable.
- Diagnostic Data-(Table 1)

Figure 1

**Table 1
Diagnostic Data**

Test	Result
ABG (Room Air)	PH 7.50 PaO ₂ 82 PCO ₂ 31
CBC	Normal
Serum Electrolytes	Normal
PT/PTT	Normal
CPK-MB Isoenzyme and Troponin I	Normal
Plasma D-dimer (ELISA)	Elevated
Electrocardiogram	Sinus Tachycardia with T-wave Inversion V1 to V4
Chest Radiograph	Normal
Doppler Echocardiography	Right Ventricular Dilaton with Moderate Tricuspid Regurgitation
Doppler Ultrasound of Lower Limbs	Thrombi in Popliteal Vein of Right Leg
V/Q Lung Scan	High Probability

Differential diagnosis for Mr. Jones at this time includes pulmonary thromboembolism, pneumonia and myocardial infarction (MI). The immediate plan involves instituting intravenous anticoagulation and placement in a critical care unit.

PULMONARY THROMBOEMBOLISM

Research indicates that more than 90% of pulmonary thrombotic embolisms (PTE) originate in the legs or pelvis where blood has become stagnant, there is injury to vessels, or a hypercoagulability state exists, all of which promotes thrombus formation.³ Miniati et al report that 50% of the patients with deep vein thrombosis (DVT) have evidence of clinically silent pulmonary embolisms and more importantly, DVT is diagnosed in 70% of the patients with angiographically confirmed PTEs.⁴ Risk factors for development of PTE include among others, surgery, oral contraception, smoking, obesity, high blood pressure, and estrogen replacement therapy.^{1,3} (Table 2)

Figure 2

Table 2: Risk Factors 1, 3

Surgery	Prolonged Immobility
Oral Contraception	Smoking
Obesity	Pregnancy
Age Over 40	Estrogen Replacement Therapy
High Blood Pressure	Heart Disease
Malignancies	Lung Disease
Hypercoagulability State	Renal Disease
Coagulation Deficiencies	Local Trauma to Vessel Wall
Venous Stasis	

Once a venous thrombus forms and dislodges from the site of origination, it will either travel to the pulmonary arterial circulation and produce a PTE or will embolize to the systemic arterial circulation by way of patent foramen ovale.⁵ Obstruction of the pulmonary arterial circulation causes release of vasoactive substances, which in turn increases pulmonary vascular resistance.⁵ An increase in alveolar dead space caused by vascular obstruction and hypoxemia, results in impaired gas exchange.⁵ Alveolar hyperventilation caused by stimulation of irritant receptors and increased airway resistance from reflex bronchoconstriction will also ensue.⁵ The result of this pathophysiology will be a decreased pulmonary compliance and a progressive increase in right ventricular dysfunction.^{1,5} Progressive right ventricular dysfunction is usually the cause of death in most patients with a fatal PTE.⁵ As the pulmonary vascular resistance increases, right ventricular wall tension increases as well, causing the interventricular septum to bulge into and compress the left ventricle.⁵ As the right coronary artery is compressed by the increase in right ventricular pressure, right ventricular infarction may result, further impairing right ventricular function.⁵ The failing right heart subsequently results in decreased left ventricular filling pressures with a decrease in cardiac output and systemic arterial pressure. The result of left ventricular compromise is additional myocardial ischemia with possible circulatory collapse.⁵

DIAGNOSIS

The size and extent of the embolism will determine the clinical signs and symptoms a patient exhibits. However, in general, a patient with pre-existing cardiopulmonary disease or one who is old, frail or debilitated will be more sensitive to the PTE regardless of size, than a patient who is relatively healthy.⁶

Tachypnea and dyspnea are the most frequent presenting signs and symptoms.⁵ Other symptoms include chest pain on inspiration, cough, wheezing, syncope, hemoptysis and apprehension.³ On physical examination, typical findings include bulging neck veins, a left parasternal lift, an accentuated pulmonic component of the second heart sound, systolic murmur at the left sternal boarder that increases on inspiration, diaphoresis and mental changes.^{1,3}

Patients with a massive PTE will invariably present with systemic arterial hypotension with impending circulatory collapse. Moderate to large PTE may exhibit normal arterial blood pressure but will show signs of right ventricular dysfunction on echocardiography as well as physical examination.⁵ Small to moderate PTE often will have normal arterial pressures as well as normal right ventricular function.⁵

Although the gold standard for a definitive diagnosis of PTE is pulmonary angiography, the test is expensive, invasive and often times not available.⁷ Therefore, in the majority of cases the diagnosis of PTE is made on clinical probability and non-invasive methods such as ventilation-perfusion (V/Q) lung scan, echocardiography, venous ultrasound of lower extremities, electrocardiogram, chest roentgenography and finally, laboratory data.⁷ (Table 3)

Figure 3

Table 3: Summary of Diagnostic Test

TEST	SENSITIVITY	SPECIFICITY	ADVANTAGE	DISADVANTAGE
Pulmonary Angiography	100% ⁸	100% ⁸	Direct visualization of embolus	Invasive Expensive Not available at all institutions Mortality 1% ⁹ Morbidity 0.5% ⁹
V/Q Lung Scan	41%	97%	Non-invasive Ability to rule PTE in or out	30% Diagnostic rate Variability in interpretation ¹⁰ No bedside testing
Echocardiography	67%	94%	Non-invasive Bedside testing Predicts severity of PTE Used in treatment determination	Non-specific Low specificity in co-existing pulmonary disease ²
Venous Ultrasound	78% ¹¹	83% ¹¹	Non-invasive Bedside testing Diagnostic for PTE if DVT found	Non-diagnostic with negative results
12-lead EKG	S ₁ , Q ₃ , T ₃ 54% ¹² ST* 36% ¹²	S ₁ , Q ₃ , T ₃ 62% ¹² ST* 100% ¹²	Non-invasive Quick and easy Bedside testing Predicts severity of PTE	Non-specific
Chest Radiograph	Westermarck's Sign 8-14% ¹³ Hampton Hump 22% ¹³	Westermarck's Sign 92% ¹³ Hampton Hump 82% ¹³	Quick and easy Bedside testing Excludes mimicking diagnoses ¹³ Aids in V/Q scan interpretation	Non-specific Lacks diagnostic utility
ABG (no prior CFD**)	88% ¹⁴	53% ¹⁴	Non-invasive Quick and easy	Non-specific Lacks diagnostic utility
Rapid D-Dimer	100% ⁹	35% ⁹	Non-invasive Easily performed Ability to exclude PTE	Non-specific Lacks diagnostic utility

* Sinus Tachycardia

**Cardiopulmonary Disease

V/Q lung scan is the most commonly used non-invasive method of diagnosing PTE. With a sensitivity of 41% and a specificity of 97% the V/Q lung scan yields results ranging from normal to high probability for PTE.4 (Table 4) However, after extensive research by Miniati et al, the researchers concluded that abnormalities other than high probability results should be regarded as non-diagnostic and further testing evoked.4 Less than half of the PTEs confirmed by angiography have high probability lung scans.5 In addition, 40% of patients with low-probability lung scans and high clinical suspicion for PTE, do in fact, have angiography confirmed PTE.5

Figure 4

Table 4
V/Q Scan Interpretation⁴

Result	Interpretation	Significance
Normal	No Perfusion Deficit	Excludes PTE
Low Probability	Perfusion Deficit with Matched Ventilation Deficit	Non-diagnostic
Intermediate Probability	Perfusion Deficit that Corresponds to Parenchymal Abnormality on Chest Radiograph	Non-diagnostic
High Probability	Multiple Segmental Perfusion Deficit with Normal Ventilation	Diagnostic for PTE

Because the V/Q scan yields only a 30% diagnostic rate, other non-invasive methods for diagnosis are employed.7 Perrier et al found doppler-echocardiography to have a 67% sensitivity and a 94% specificity in diagnosing or excluding PTE.7 Reports show that right ventricular dilation confirmed by echocardiography ranges from 50-100% in patients with PTE.2 Therefore, a negative echocardiogram does not exclude a small PTE but it does exclude a potentially significant episode, which results in extreme right ventricular compromise.2

Venous ultrasound of the lower extremities has also evolved into an important diagnostic tool. Because venograms shows thrombus in the deep venous system in at least 70% of patients with confirmed PTE, venous ultrasounds are currently being used to detect DVT in patients with clinical suspicion of PTE.15 If the V/Q scan is non-diagnostic and the venous ultrasound reveals thrombi, then the diagnosis of PTE is likely and anticoagulation should be instituted.15 However, it must be mentioned that negative DVT does not exclude a diagnosis of PTE, because 40% of suspected PTEs do not demonstrate DVT.15 In these situations, “the clot may have already embolized to the lung or the pelvic veins where ultrasonography is usually inadequate”.5 Other diagnostic methods such as electrocardiography, chest roentgenography and laboratory data are much less diagnostic in the typical PTE patient however, may reveal important information. For example, the typical electrocardiogram for the PTE patient shows sinus tachycardia. The “classical changes” of pulmonary embolus-S1, Q3, T3 (S wave in lead I, Q and inverted T waves in lead V3), which suggest right ventricular strain, are absent in the vast majority of patients.5,15 In addition, the chest radiograph is normal in the majority of PTE patients however, diagnostic changes possibly seen on x-ray include, focal oligemia or decreased pulmonary vascular markings-called Westermarck’s sign (see:

<http://www.vh.org/Providers/Textbooks/ElectricPE/RadIma>

ges/16.XFullChest.html), or peripheral wedged-shaped density above the diaphragm-termed Hampton’s hump (see: <http://www.vh.org/Providers/Textbooks/ElectricPE/RadImages/13.XFullChest.html>).

1,5,15,16,17,18

Laboratory tests for suspected PTE include arterial blood gases (ABG) and quantitative plasma D-dimer enzyme-linked immunosorbent assay (ELISA). Arterial hypoxemia in a patient without pre-existing lung disease is suggestive of a PE, however a normal ABG, does not exclude a PTE.¹⁵ The ELISA test however, is elevated in more than 90% of the patients with PTE.⁵ The test reflects plasmin’s breakdown of fibrin and indicates endogenous thrombolysis. Because the ELISA lacks specificity and can be elevated with MI, sepsis or almost any systemic disease, the test is most useful in the healthy patient presenting with unexplained dyspnea.⁵

DIFFERENTIAL DIAGNOSIS

Because PTE’s presentation can represent numerous abnormalities, the differential diagnosis is broad. For Mr. Jones, after a consummate history and physical examination, the differential diagnosis for him include PTE, pneumonia, and MI among others. In this age population, pneumonia must be considered with a presentation of chest pain especially upon inspiration, dyspnea, cough, weakness, tachycardia, tachypnea, and low grade-fever. However, because of the rapid progression of symptoms, no egophony on physical examination, a normal chest x-ray as well as normal white blood cell count (WBC), these findings do not support a diagnosis of pneumonia at this time. Pneumonia however, must remain on the differential diagnosis list while PTE is further evaluated in light of the fact infiltrates seen on the chest x-ray, purulent sputum, high fever and chills may not develop for 12 hours or more.⁵ In addition, it is not uncommon to treat patients with intravenous heparin and antibiotics in the short term until a definitive diagnosis is made.¹⁵

MI must also be considered as a differential diagnosis. MI is relevant due to the patient’s risk factors for cardiovascular compromise namely elderly, male, overweight, sedentary lifestyle, smoker and significant family history for heart disease; but also considering his presenting signs and symptoms, in particular chest pain, tachycardia, and tachypnea. However, MI quickly becomes lower on the differential diagnosis list after ECG reveals t-wave inversion in leads V1 through V4. This pattern suggests right

ventricular strain from progressive right heart failure.¹⁹ This pattern could however, represent anterior-septal ischemia caused by primary cardiovascular compromise or the secondary effects of the PTE. Therefore, serial CPK-MB and troponin1 values become very important in evaluating the state of the heart as well as defining the appropriate diagnosis. In Mr. Jones’s case, these values were negative and therefore suggest PTE.

Mr. Jones’s high probability V/Q result is sufficient to diagnose PTE. Miniati et al report “a high probability ventilation-perfusion scan...justifies the institution of anticoagulant treatment, especially when this scintigraphic pattern is associated with a high clinical probability for pulmonary embolism”.⁴ In addition, DVT, dilated right heart with suggested right ventricular strain and the elevated ELISA test, provides overwhelming evidence for the diagnosis of PTE.

Figure 5

Table 5: Mr. Jones’ Differential Diagnoses

	Pneumonia	Myocardial Infarction	Pulmonary Thromboembolism
Risk Indicators	Elderly	Elderly	Sudden onset of symptoms
	Chest pain with inspiration	Male	Chest pain with inspiration
	Dyspnea	Overweight	Tachycardia
	Cough	Sedentary lifestyle	Tachypnea
	Weakness	Smoker	Cough
	Tachycardia	Family history	Weakness
	Tachypnea	Sudden onset of symptoms	Apprehension
	Low grade fever	Chest pain	+ JVD **
		Tachycardia	S3 heart sound
		Tachypnea	Systolic murmur

Figure 6

Test Results	None support pneumonia	ECG-Sinus Tachycardia T wave inversion in leads V1-V4	V/Q scan-high probability ECG-sinus tachycardia T wave inversion in leads V1-V4 Doppler echocardiography- dilated right heart Doppler ultrasound-DVT*** D-dimer-elevated
Rational	Diagnosis ruled out because sudden onset of symptoms, normal x-ray and normal WBC* count	Diagnosis ruled out because CPK-MB and Troponin I values normal	Diagnosed because high probability V/Q scan

*WBC - White blood cell

**JVD -Jugular vein distention

***DVT - Deep vein thrombosis

TREATMENT

Once a diagnosis of PTE is made, not only will a pulmonary and possible surgical consultation be in order, but also primary or secondary therapy must be instituted immediately after contraindications have been evaluated. Primary therapy involves clot dissolution with thrombolysis or removal of the embolism by surgical embolectomy.⁵ Secondary prevention involves treatment with anticoagulation to prevent a recurrent PTE.²⁰ Echocardiography plays a major role in “risk stratification” to determine which patients would benefit from primary or secondary treatment.⁵

In general patients with normal systemic arterial pressure and normal right ventricular function, secondary therapy with intravenous anticoagulation is the treatment of choice.²⁰ Patients, in whom right ventricular hypokinesia are demonstrated through echocardiography but remain normotensive, primary treatment should be strongly considered.²⁰ Furthermore, patients who exhibit systemic arterial hypotension are generally demonstrating cardiogenic shock, which suggests a massive PTE and without lysis or removal of the clot, death will ensue.²⁰

In the majority of patients, heparin is the initial treatment.²¹ Heparin acts by accelerating the action of antithrombin III, preventing additional clots from forming as well as permitting endogenous fibrinolysis to dissolve some of the pre-existing clot.¹ Anticoagulation with heparin should be

maintained for at least five days to a targeted international normalized ratio (INR) of 2.0-3.0.^{1,21} Warfarin in conjunction with heparin, is started once therapeutic levels of heparin have been achieved.¹

Controversy exists over the optimal long-term management of PTE. Goldhaber, reports warfarin taken for six months as opposed to six weeks, prevents recurrences.¹ However, Agnelli and Sonaglia state three months is sufficient.²¹

Low-molecular-weight heparin (LMWH) has increasingly replaced unfractionated heparin for treatment and prophylaxis of PTE. These agents require no laboratory monitoring, have greater bioavailability and absorption, have longer half-lives and decreased rates of heparin-induced thrombocytopenia.^{1,5,18} LMWH however, has not been approved by the FDA for treatment of PTE.^{5,18}

Thrombolysis on the other hand, has a 14-day window of opportunity for it to be effective.^{5,20} The advantages of thrombolysis over secondary therapy involves not only the dissolution of the pulmonary clot and the remnants of the remaining clot in the pelvis or deep leg veins, but also prevents exacerbation of pulmonary hypertension by preventing the release of serotonin and other neurohumoral factors.⁵ A typical lytic regimen is 100 mg of recombinant tissue plasminogen activator, as a continuous infusion over two hours.⁵

In patients whom antithrombotic or anticoagulation therapy is not appropriate such as with active bleeding, an inferior vena caval filter is necessary to prevent a recurrent PTE.^{1,5} Other indications include recurrent PTE despite adequate prolonged anticoagulation or patients with right heart failure who are not candidates for anticoagulation.^{1,5} Inferior vena caval filters are used for prophylaxis only and do not affect the thrombotic process.^{1,5}

In summary, diagnosing PTE is a perplexing aspect of medicine and yet a missed diagnosis carries a high mortality rate. With the exception of pulmonary angiography, there is no definitive diagnosis for PTE, therefore the search becomes a puzzle. The appropriate pieces of the puzzle must be assembled quickly and accurately to optimize patient outcomes.

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