Risk Factors for the Assessment of Patients with Pulmonary Embolism

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Key Words
Pulmonary embolism, thrombolysis, pulmonary embolectomy, anticoagulation, risk stratification

Abstract
Pulmonary embolism (PE) occurs frequently among cancer patients, with a spectrum ranging from small, clinically insignificant thrombi to life-threatening massive PE. It is fatal in as many as 14% of cancer patients, primarily by producing right ventricular heart failure and cardiogenic shock. PE diagnosis is difficult because the signs and symptoms imitate other commonly occurring diseases. Clinicians must be able to integrate a wide array of diagnostic imaging tools and laboratory tests to ensure rapid assessment and diagnosis. Risk stratification with the use of cardiac biomarkers and imaging tests to evaluate right ventricular function will identify treatment options. Hemodynamically stable patients can be treated effectively with anticoagulation alone, whereas those with right ventricular dysfunction require an aggressive strategy with thrombolysis, surgical embolectomy, or a catheter-based intervention. When anticoagulation is contraindicated, a vena caval filter may be deployed. PE treatment must be customized to the individual and consider the existing thrombus burden, presence of underlying cardiopulmonary disease and right side heart dysfunction, and cancer status of the patient. Clinicians should focus on providing adequate thromboprophylaxis in hospitalized cancer patients to avoid PE treatment. (JNCCN 2006;4:871–880)

The relationship between cancer and venous thromboembolism (VTE), including deep venous thrombosis (DVT) and pulmonary embolism (PE), has been recognized for almost 150 years. The proportion of patients with VTE has increased. Although the incidence of VTE in the United States population has been reported to be 1 event per 1000 patients annually, DVT and PE are estimated to occur in 1 of every 200 cancer patients annually. Cancer patients have a sevenfold higher VTE incidence rate than those hospitalized with other diseases.

VTE rates vary with each type of malignancy. Although the highest rates are associated with cancers of the kidney, stomach, pancreas, and brain, VTE is now recognized as occurring with more frequency in what was thought to be low-risk “liquid” cancer subgroups. In a recent study, patients with neutropenic lymphoma and leukemia represented more than one third of all patients with VTE events. More concerning, hospital mortality approached 15% for these patients. The extent of cancer, type of chemotherapy, presence of catheters, and underlying genetic and acquired thrombophilias all contribute to the development of VTE.

For cancer patients, the presence of VTE is an ominous sign for prognosis and treatment. VTE is the leading cause of death in cancer patients other than the cancer itself. An autopsy study showed that PE occurred in 17% of cancer patients and was fatal in 14%. In more than half of these patients, the cancer was localized or had limited metastases. Patients with cancer and a VTE episode are more likely to have distant metastases than those without. The probability of death or rehospitalization is higher in cancer patients than in those with nonmalignant disease.

Despite anticoagulant treatment, cancer patients have a threefold higher incidence of recurrent VTE events. Managing anticoagulation is complicated in cancer patients. The incidence of major bleeding in these patients is 2.5 to 6 times higher than in patients without malignancy. Therapeutic international normalized ratios (INRs) are more difficult to obtain. Therefore, patients require more frequent monitoring and treatment visits and are less likely to complete their intended course of anticoagulation.
Pathophysiology

PE obstructs the pulmonary artery (Fig. 1). As this obstruction increases, so does pulmonary artery pressure. The presence of thrombus promotes release of vasoconstricting substances, such as serotonin from platelets and histamine from tissue, and generates thrombin in the plasma. These substances all contribute to further increases in pulmonary artery vascular resistance and pulmonary artery pressure.\textsuperscript{15–18}

As pulmonary artery pressure rises, right ventricle afterload increases, leading to tricuspid regurgitation with tricuspid valve annular dilation, elevations in right ventricular wall tension, and increased right ventricular oxygen demand. Right ventricular dilatation and dysfunction ensue. Right ventricular dilatation shifts the interventricular septum, producing underfilling of the left ventricle. Systemic cardiac output and pressure decline, compromising coronary perfusion and producing myocardial ischemia. Right ventricular wall tension compresses the right coronary artery, impairing myocardial perfusion and limiting oxygen supply. Right ventricular microinfarction leads to troponin elevation, and right ventricular overload causes increases in cardiac biomarkers such as N-terminal pro–brain natriuretic peptide (NT-pro BNP) and BNP. While this insidious process progresses, most patients without underlying cardiopulmonary disease can maintain a normal systemic arterial pressure for 12 to 48 hours.\textsuperscript{15–18}

PE impairs the exchange of oxygen and carbon dioxide in the lungs and produces hypoxemia. Venous blood, flowing to alveoli where the ratio of ventilation to capillary blood flow is low, produces arterial hypoxemia. Alveolar dead space, representing ventilation of alveoli that are not involved in gas exchange, increases and further impairs the elimination of carbon dioxide. Atelectasis, caused by loss of surfactant and alveolar hemorrhage, further aggravates arterial hypoxemia. Right to left shunting of venous blood into the arterial system occurs through the heart, the lungs, or both, and bypasses the gas exchange surfaces of the lung. Administering supplemental oxygen may fail to correct arterial hypoxemia. Continuous positive airway pressure will further increase pulmonary vascular resistance and worsen intracardiac shunting.\textsuperscript{15–18}

Diagnosis

PE remains difficult to diagnose; it evades both experienced and inexperienced physicians.\textsuperscript{19} Clinical presentation varies widely. Some patients present with abrupt onset, whereas others suffer progressive deterioration. PE imitates many common diseases and conditions with nonspecific signs and symptoms. Underlying comorbidities may also disguise or cloud the clinical picture. The clinical presentation ranges from stable hemodynamics to cardiogenic shock. Clinical probability of PE should be assessed with a point score system (Table 1). Decision algorithms facilitate diagnosis (Fig. 2).

Clinical suspicion is critical to the diagnosis of PE. Initial workup should include a comprehensive medical history and physical examination.\textsuperscript{15} In the International Cooperative Pulmonary Embolism Registry, the most common symptoms at PE diagnosis were dyspnea (82%), chest pain (49%), cough (20%), syncope (14%), and hemoptysis (7%).\textsuperscript{20} On physical examination, the patient may not appear ill. Arterial hypertension, tachycardia, tachypnea, a parasternal heave, and cyanosis are often present with an accentuated P2 pulmonic heart sound, and tricuspid regurgitation on auscultation.\textsuperscript{21} Signs of right ventricular dysfunction include distended neck veins and an S3 gallop.

PE should be suspected in any patient with current DVT or a history of PE. In a recent U.S. VTE
complete or incomplete right bundle branch block. 

T-wave inversion in leads VI through V4 is particularly helpful. The chest radiograph is often the first imaging study performed and helps exclude pneumonia, pneumothorax, or other thoracic diseases. Most patients with PE have a normal chest radiograph. A near-normal radiograph in the setting of severe respiratory compromise is highly suggestive of a PE.

Chest Computed Tomography
Computed tomography (CT) of the chest with intravenous contrast has become the preferred imaging study in most patients with suspected PE. It has supplanted pulmonary radionuclide perfusion scintigraphy (lung scanning) as the initial imaging test because it provides conclusive and reproducible results. CT examination can include scanning of the venous system from the iliac to the popliteal veins, eliminating the need for venous ultrasound. Multislice CT scanners can image emboli in fifth- or sixth-order subsegmental pulmonary arteries. It may provide an alternative diagnosis to PE, identify concomitant morbidities that were not apparent on chest radiographs, or eliminate suspicion of pulmonary disease.

Table 1  Pulmonary Embolism Clinical Model with a Point Score System

<table>
<thead>
<tr>
<th>Type of Measure</th>
<th>Points</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leg swelling and pain in the deep vein regions</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Heart rate &gt; 100 beats per minute</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>Immobilization or surgery in prior 4 weeks</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>Previous objectively diagnosed DVT or PE</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Malignancy</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Alternative diagnosis less likely than PE</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

Total Point Score Probability

< 2 Low probability
> 2–6 Moderate probability
> 6 High probability

Abbreviations: DVT, deep venous thrombosis; PE, pulmonary embolism.

A prospective study showed that most patients require more than one diagnostic test to confirm the diagnosis of acute PE and that delays in medical evaluation and confirmatory diagnostic testing are common. An integrated diagnostic approach ensures rapid assessment and risk stratification, and will help identify the optimum strategy for PE treatment.

Electrocardiogram and Chest Radiograph
An electrocardiogram (EKG) is helpful in excluding acute coronary syndromes and may raise suspicion of PE if signs of right ventricular strain are present. Typically, EKG changes, if any, are associated with sinus tachycardia or atrial fibrillation with right heart strain, minor ST- and T-wave changes, and...
Chest CT can provide diagnostic information on the size and function of the right ventricle.\textsuperscript{25} CT measurements of right and left ventricle dimensions in acute PE may help identify patients at risk for adverse events and predict early death.\textsuperscript{26,27} When CT 4-chamber views were reconstructed and measured, right ventricular enlargement was associated with a 30-day mortality rate of 15.6\%, compared with 7.7\% in those without right ventricular enlargement.

Clinicians must recognize that there are different generations of chest CT scanners and that the one being used is important in interpreting the imaging results. First-generation scanners lack the resolution to detect emboli reliably in subsegmental arteries. The latest generation of multi–detector row CT scanners permits the capture of the entire thorax, thinner imaging planes with 1-mm resolution, and visualization of subsegmental vessels.

**Pulmonary Angiography**

Pulmonary angiography was long considered the gold standard for diagnosis, but is rarely performed now because CT scanning can resolve most diagnostic dilemmas. However, pulmonary angiography is required when interventions are planned, such as suction catheter, embolectomy, mechanical clot fragmentation, or catheter-directed thrombolysis.

**Lung Scanning**

Pulmonary radionuclide perfusion scintigraphy is no longer the principal diagnostic imaging test when PE is suspected. It remains an initial imaging option for patients with renal insufficiency or contrast allergy, and for those who are pregnant.

**Plasma D-Dimer**

This blood screening test for PE is highly sensitive but lacks specificity. It is usually elevated in patients with advanced cancer. However, a negative D-dimer essentially rules out PE.\textsuperscript{28–33}

**Pulmonary Embolism Treatment**

The spectrum of PE ranges from small, clinically insignificant thrombi to life-threatening massive pulmonary embolism. PE can be classified based primarily on systemic arterial blood pressure and right ventricular function (Table 2). Appropriate treatment ranges from supportive care to invasive embolectomy. PE treatment algorithms have been designed with risk stratification, providing the key to appropriate

<table>
<thead>
<tr>
<th>Description</th>
<th>Clinical Presentation</th>
<th>Circulatory System Status</th>
<th>Anatomical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Massive</td>
<td>Dyspnea, syncope, cyanosis, chest pain absent</td>
<td>Systemic arterial hypotension which may progress to cardiogenic shock, right ventricular dysfunction</td>
<td>Thrombus frequently present bilaterally with greater than 50% obstruction of the pulmonary vasculature</td>
</tr>
<tr>
<td>Moderate to large (&quot;submassive&quot;)</td>
<td>Chest pain present, right ventricular hypokinesis, elevated troponin, NT-pro BNP, and BNP</td>
<td>Normal systemic arterial blood pressure with right ventricular dysfunction present</td>
<td>Thrombus obstructing one third or more of the pulmonary artery vasculature</td>
</tr>
<tr>
<td>Small to moderate</td>
<td>No elevations in troponin, NT-pro BNP, or BNP</td>
<td>Normal arterial blood pressure, normal right ventricular function</td>
<td>Small emboli in subsegmental branches</td>
</tr>
<tr>
<td>Pulmonary infarction</td>
<td>Fever, leukocytosis, elevated sedimentation rate, pleuritic chest pain, hemoptyysis, pleural rub, or evidence of lung consolidation</td>
<td>Normal right ventricular function</td>
<td>Small peripheral emboli lodged near the pleura</td>
</tr>
<tr>
<td>Nonthrombotic embolism</td>
<td>Occurring after catheter placement or removal, or drug administration</td>
<td>Respiratory failure, right ventricular dysfunction is rare</td>
<td>Most commonly air, fat, tumor fragments, amniotic fluid, drug precipitate, or contaminants</td>
</tr>
</tbody>
</table>

Abbreviation: NT-pro BNP, N-terminal-pro B-type natriuretic peptide.
treatment selection (Fig. 3). Elevations in cardiac biomarkers (troponin, NT-pro BNP, and BNP) require additional assessment of right ventricular function. Echocardiography is an important imaging tool to assist in risk stratification.

**Cardiac Biomarkers**

**Troponin:** Troponin is a component of the thin filaments in cardiac muscle. It is the protein to which calcium binds to regulate muscle contraction. Troponin has 3 subunits. Two of these, troponin I (TnI) and troponin T (TnT), are sensitive and specific indicators of myocardial damage. In acute PE, troponin levels correlate well with the extent of right ventricular dysfunction and reflect microscopic myocardial necrosis. The release of troponins in acute PE is less dramatic than in myocardial infarction. Peak plasma levels are quantitatively less, of shorter duration, and may require 6 to 12 hours to appear in some patients. Studies have shown that troponin release correlates with adverse outcomes in patients with PE. Elevations in TnT are associated with in-hospital death, prolonged hypotension, and cardiogenic shock. Elevation of TnT and TnI is associated with a higher rate of in-hospital complications and recurrent PE. Troponin elevation combined with right ventricular enlargement can be lethal, increasing the 30-day mortality rate to 38%.

**Brain Natriuretic Peptides:** BNP is a polypeptide hormone secreted by the ventricles of the heart in response to stretching of the myocytes. At BNP release, another N-terminal amino acid fragment, NT-pro BNP, is released. Because the natriuretic peptides counter the blood pressure–raising effects of the renin–angiotensin system, they are sensitive indicators of neurohormonal activation. Because elevated levels are associated with right ventricular dysfunction, BNP and NT-pro BNP are useful diagnostic and prognostic biomarkers for patients with PE. A lower cutoff level of less than 50 pg/mL identifies 95% of patients with a benign clinical course and is useful in ruling out an adverse in-hospital outcome. Measurement of BNP and NT-pro BNP with an imaging modality may identify low- and high-risk patients with acute PE.

**Echocardiography**

Transthoracic echocardiography is now a useful tool for risk-stratifying patients with acute PE. Abnormalities include right ventricle dilation, hypokinesis, abnormal motion of the interventricular septum, tricuspid regurgitation, and a congested inferior vena cava with a lack of inspiratory collapse. In PE patients with preserved systemic arterial pressure (90 mm Hg or higher), right ventricular hypokinesis on baseline echocardiography independently predicted a decrease in 30-day survival. In these patients, the 30-day mortality rate approached 17% and their risk for death almost doubled compared with those without right ventricular dysfunction. In multivariable analysis, cancer had the highest risk for 30-day mortality compared with other disease states, such as congestive heart failure or chronic lung disease. In a study of 126 patients with PE in which echocardiography was performed on the day of diagnosis, right ventricular dysfunction had a sixfold higher relative risk for in-hospital death. The presence of cancer doubled the risk for in-hospital death from PE, causing death in 27.7% of patients with right ventricular dysfunction. Therefore, echocardiography represents a convenient and safe imaging test to identify patients who appear stable but are at high risk for adverse outcomes.

**Anticoagulation**

Anticoagulation is the cornerstone of management in patients with PE and should be initiated as soon as...
the diagnosis is suspected. Although anticoagulant therapy does not dissolve existing thrombus, it prevents further propagation and allows time for the body’s endogenous fibrinolytic system to act. Patients with PE with normal systemic arterial blood pressure and normal right ventricular function experience excellent outcomes with anticoagulation alone. A wide array of anticoagulants is available for clinicians to prescribe (Table 3). When embolectomy or thrombolysis is considered, intravenous unfractionated heparin (UFH), a short-acting reversible agent, is the optimal choice. Heparin is administered as a bolus dose followed by a continuous infusion, typically using a weight-based nomogram to facilitate

![Table 3 Pharmacologic Options for Pulmonary Embolism Treatment](image)

<table>
<thead>
<tr>
<th>Agent</th>
<th>Trade Name</th>
<th>Route of Administration</th>
<th>Treatment Dose</th>
<th>FDA Indication</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unfractionated Heparin</td>
<td>None</td>
<td>IV bolus, followed by IV continuous infusion</td>
<td>80 units/kg, then 18 units/kg per hour</td>
<td>Treatment of venous thrombosis and its extension</td>
<td>Monitor with aPTT every 6 hours. Target aPTT 2.0–2.9 × control</td>
</tr>
<tr>
<td><strong>Low Molecular Weight Heparins</strong></td>
<td></td>
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<tr>
<td>Dalteparin</td>
<td>Fragmin</td>
<td>Subcutaneous</td>
<td>100 units/kg twice daily or 200 units/kg once daily</td>
<td>Treatment of DVT with or without PE</td>
<td>Dose adjustments required in renal dysfunction</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>Lovenox</td>
<td>Subcutaneous</td>
<td>1.0 mg/kg twice daily or 1.5 mg/kg once daily</td>
<td>Treatment of DVT with or without PE</td>
<td>Reduce dose to 1.0 mg/kg once daily with creatinine clearance &lt; 30 mL/min</td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>Arixtra</td>
<td>Subcutaneous</td>
<td>5.0 mg for patients weighing &lt; 50 kg; 7.5 mg for patients weighing &gt; 50 kg but &lt; 100 kg; 10 mg for patients weighing &gt; 100 kg</td>
<td>Treatment for acute PE</td>
<td>Contraindicated in renal dysfunction (creatinine clearance &lt; 30 mL/min) and patients weighing &lt; 50 kg</td>
</tr>
<tr>
<td>Tinzaparin</td>
<td>Innohep</td>
<td>Subcutaneous</td>
<td>175 units/kg once daily</td>
<td>Treatment of DVT with or without PE</td>
<td>Dose adjustments required in renal dysfunction</td>
</tr>
<tr>
<td><strong>Direct Thrombin Inhibitors</strong></td>
<td></td>
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<tr>
<td>Argatroban</td>
<td>Acova</td>
<td>IV</td>
<td>2 mcg/kg/min</td>
<td>Treatment of thrombosis in patients with heparin induced thrombocytopenia</td>
<td>Dose adjustments required in hepatic impairment</td>
</tr>
<tr>
<td>Lepirudin</td>
<td>Refludan</td>
<td>IV bolus, followed by IV continuous infusion</td>
<td>0.4 mg/kg, then 0.15 mg/kg per hour</td>
<td>Anticoagulation in patients with heparin induced thrombocytopenia</td>
<td>Dose adjustments required in renal impairment</td>
</tr>
<tr>
<td><strong>Thrombolytic agents</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alteplase, t-PA</td>
<td>Activase</td>
<td>IV</td>
<td>100 mg infused over 2 hours</td>
<td>Management of acute massive PE</td>
<td>Anticoagulant therapy should be held during 2 hours infusion</td>
</tr>
</tbody>
</table>

Abbreviations: aPTT, activated partial thromboplastin time; DVT, deep venous thrombosis; IV, intravenous; PE, pulmonary embolism; t-PA, tissue plasminogen activator.
Risk Factors for Pulmonary Embolism

Risk factors for bleeding and contraindications to anticoagulant therapy should be considered before initiating therapy. Patients who should not receive anticoagulation therapy include those with active (more than 2 units transfused in 24 hours) or chronic bleeding (measurable bleeding for greater than 24 hours); those with a recent history of central nervous system bleeding; those who have recently undergone major surgery, spinal anesthesia, or lumbar puncture; and those at high risk for falls.

Inserting an inferior vena caval filter should be considered when the risk for bleeding from anticoagulation is high. Several clinical scenarios also warrant consideration of filter placement, including development of new PE while on adequate anticoagulation, patient noncompliance with anticoagulation therapy, baseline pulmonary function severe enough to make recurrent PE life-threatening, and multiple PE and chronic pulmonary hypertension. Although the use of vena caval filters has risen dramatically, the efficacy data are limited.48 An 8-year follow-up of patients enrolled in PREPIC, the largest randomized trial of patients with DVT and/or PE, showed that vena caval filters reduced the risk for symptomatic PE, but increased the risk for DVT and had no effect on survival.51 In 2 small retrospective studies in patients with VTE, brain tumors, and brain metastases, filter placement was associated with thromboembolic complication rates of 45% and 40%, respectively.

Temporary filters are now available for when the contraindication to anticoagulation is time-limited. They can be inserted, left in place for 10 to 14 days and then retrieved, or left permanently if the contraindication persists. Unlike pharmacologic anticoagulation, a vena caval filter does not prevent continued thrombosis. When no contraindication persists, anticoagulation should be resumed as soon as possible after the filter is deployed.13

Thrombolysis

Although thrombolysis can be a lifesaving treatment, its use is still debated. Experts agree on its use in patients with hemodynamically unstable, massive PE who have a low risk for bleeding (Table 3). When compared with UFH alone, thrombolysis produces rapid improvement in right ventricular dysfunction and improvements in radiographic abnormalities in acute PE. However, in a meta-analysis of 11 trials involving unselected PE patients, no statistical difference in recurrent PE, death, or major bleeding was seen in patients who underwent thrombolysis and those who underwent anticoagulation with UFH. In the subanalysis of massive PE, thrombolysis reduced the risk for death or recurrent PE by 55%, but the risk for major bleeding doubled.13

The Management strategies and Prognosis of Pulmonary Embolism-3 Trial (MAPPET-3) suggests that thrombolysis should be administered to patients with submassive PE.56 MAPPET-3 is the largest randomized trial comparing thrombolytic therapy (alteplase, tissue plasminogen activator [t-PA]) plus UFH with UFH alone. MAPPET-3 enrolled 256 patients with submassive PE, defined as normal systemic arterial blood pressure but with right ventricular dysfunction or pulmonary hypertension. The primary end point was death or escalation of therapy, defined as the need for catecholamine infusion, open-label thrombolysis, endotracheal intubation, cardiopulmonary resuscitation, or emergency embolectomy. A statistically significant reduction occurred in the primary end point in the patients treated with t-PA plus heparin compared with those treated with UFH alone (10% vs. 25%, P = .006).

Major bleeding occurred in only 0.8% of patients treated with thrombolysis with no episodes of intracranial hemorrhage. These results sharply contrast
with events reported in routine practice. Registry data have shown major bleeding and intracranial hemorrhage occurring in 21.7% and 3.0% of patients, respectively. In a single-center study, major bleeding and intracranial hemorrhage occurred in 19.2% and 5.0% of patients, respectively. Cancer, along with pressure support, diabetes, and an elevated INR, were independent predictors of major hemorrhage. The risk for bleeding has led to a search for novel mechanical strategies and resurrected the use of older ones.

**Pulmonary Embolectomy**

Surgical or catheter-based embolectomy should be considered for patients with PE with contraindications to thrombolysis or if risk stratification indicates a high likelihood of adverse outcome. Historically, surgical embolectomy has been characterized by poor survival.

However, through the use of a well-organized, multidisciplinary team approach, surgical embolectomy has re-emerged as an effective strategy for treating patients with massive or moderate-sized PE who have contraindications to thrombolysis. CT imaging and modern risk stratification provide early diagnosis and identification of patients in whom hemodynamic compromise is likely, and allow for rapid transport to the operating room. The routine availability of a surgical team coupled with advances in surgical technique has improved the survival rate to 89%.

When contraindications to thrombolysis exist and surgical embolectomy is not available, percutaneous catheter thrombectomy may be capable of reversing PE-related right ventricular failure. Catheter-based approaches include mechanical clot fragmentation, aspiration, and rheolytic thrombectomy. Their use is limited to the main pulmonary arteries. Because of limitations in catheter size, percutaneous catheter embolectomy usually results in extraction of multiple tiny clot fragments rather than massive pulmonary arterial thrombus. Nonetheless, these procedures will provide modest angiographic improvement, restore normal blood pressure, and decrease hypoxemia. Advances in catheter design, specifically for use in the pulmonary arteries, are being investigated and may offer future benefit.

**Conclusions**

When massive PE strikes patients in the hospital, it typically receives widespread attention and analysis. The mundane task of ensuring adequate VTE prophylaxis in hospitalized cancer patients is frequently overlooked. However, the burden to health care facilities is significant.

Although the association of VTE and its complications in cancer have long been recognized, the lack of preventative strategies in this population is concerning. In an electronic DVT alert study that identified patients at high risk for VTE, the largest population of patients not undergoing a mechanical or pharmacologic prophylaxis strategy were medical patients with cancer. One oncology center reported that 6.1% of hospital-bed occupancy was caused by cancer-related VTE events. More recently, a study following up on outcomes of cancer patients with VTE found that the mean hospitalization for VTE-related events was 11.2 days with a mean cost of $20,065.

In summary, therapy for PE should be individually customized based on clinical status, thrombus burden, the presence of underlying cardiopulmonary disease, cardiac biomarker presentation, and the detection of right-side heart dysfunction through physical examination, EKG, or echocardiogram. Stable patients should be prescribed anticoagulation, preferably with LMWH, to prevent recurrent VTE. High-risk patients should be considered for more invasive treatments, either thrombolysis or embolectomy, as primary therapy to dissolve or remove the thrombus, in addition to heparin anticoagulation to prevent recurrent VTE.

**References**

Risk Factors for Pulmonary Embolism


