**Review Article**

# Management of Acute Pulmonary Embolism in Costa Rica and Spain

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## Abstract

Pulmonary thromboembolism is defined as the obstruction of the pulmonary artery or one of its branches due to the detachment of a thromboembolus of various etiologies, ultimately affecting the right chamber of the heart as a consequence of increased pressure. Risk factors are explained by the classic triad of Rudolf Virchow, which consists of vascular wall injury, a hypercoagulable state, and venous stasis. The most prevalent factor is stasis caused by immobilization in hospitalized patients, particularly those who have undergone surgical procedures. It is also important to emphasize that malignancy and tissue damage resulting from surgical interventions are the two main triggers of coagulation system activation. Clinical presentation is variable, ranging from asymptomatic cases or mild and nonspecific symptoms to sudden death. The classic triad of pulmonary embolism - dyspnea, pleuritic chest pain, and hemoptysis - is present in a low percentage of patients. However, dyspnea at rest or with exertion is the most common symptom, followed by pleuritic chest pain and cough, while tachypnea is the most prevalent clinical sign after findings suggestive of deep vein thrombosis. Pulmonary angiography with catheterization was formerly considered the gold standard for the diagnosis of pulmonary embolism and involves the insertion of a catheter into the right heart chambers for contrast injection. Currently, its use is limited to cases in which computed tomography pulmonary angiography or ventilation–perfusion scintigraphy is not feasible, or as a combined diagnostic and therapeutic modality aimed at thromboembolus removal. Several therapeutic strategies are available for the management of pulmonary embolism, including pharmacological and interventional approaches. Definitive treatment can be tailored according to the patient's hemodynamic status.

## Key words

Acute pulmonary embolism, Anticoagulation, Thrombolysis, Risk stratification, Catheter-directed therapy.

## Introduction

Pulmonary thromboembolism is defined as a rapidly developing disease that can be life-threatening, as the pulmonary circulation is compromised by the obstruction of one or more pulmonary arteries. This obstruction leads to right ventricular overload and may progress to shock and ultimately death [1].

Factors that raise suspicion of a potential risk for a patient to develop pulmonary embolism are of great importance, as they may precede appropriate patient management and the prevention of complications. In addition to traditional risk factors, predictive scoring systems are described, which are highly valuable in supporting the clinical approach and allowing for proper risk stratification [2].

The symptoms and signs of pulmonary embolism are considered nonspecific for diagnosis, indicating that the condition may go unrecognized if clinical suspicion is not raised. Therefore, it is essential to be aware of these clinical presentations and to incorporate complementary diagnostic studies to achieve an accurate diagnosis [3].

Long-term complications resulting from pulmonary embolism are highly relevant, as resolution of the acute event does not preclude the development of conditions such as chronic pulmonary hypertension, post-thrombotic syndrome, or recurrence within a few months [4].

Deaths caused by pulmonary thromboembolism often occur within the first hours after onset, posing a significant challenge in terms of timely intervention, as both the initiation of treatment and the implementation of diagnostic strategies may be delayed, potentially resulting in fatal outcomes. It is also important to consider the risk of recurrence, as one of the causes of mortality associated with pulmonary embolism is the repetition of the event in the same vascular territory as the initial episode [5].

## Methodology

For the present study, a systematic review with a descriptive qualitative approach was conducted. The sample was based on the selection of bibliographic sources that met the established inclusion and exclusion criteria, covering the period from 2019 to 2025. The study included adult patients from any hospital department except gynecology–obstetrics and pediatrics, published in English and Spanish, and limited to Costa Rica and Spain.

All information was obtained from databases such as PubMed, EBSCO, Dialnet, and the Virtual Health Library (BVS). Out of a total of 19,490 articles initially screened, only 11 studies met the criteria and were included in the final analysis. This review followed the PRIMA guidelines (**Figure – 1**).

### Definition

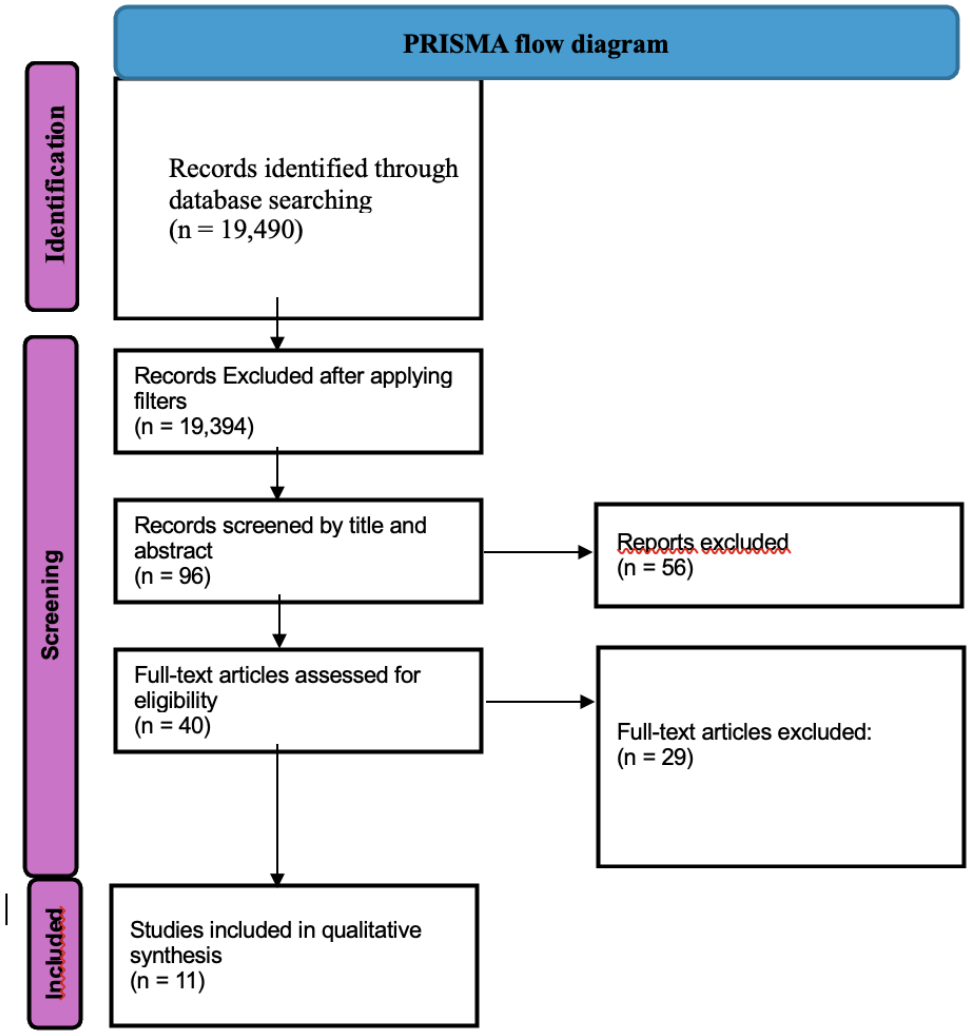
Pulmonary embolism is defined as the obstruction of the pulmonary artery or one of its branches due to the detachment of a thromboembolus originating from the venous system, usually from the deep veins. This process ultimately affects the right chambers of the heart due to increased pressure load [1].

### Thrombosis vs. Coagulation

Coagulation is the process involving the activation of a cascade of sequential events that results in clot formation, whereas thrombosis refers to the formation of a clot in situ. Thrombus formation begins with transient reflex vasoconstriction mediated by endothelin, followed by platelet adhesion leading to the formation of a platelet plug. This aggregation releases thromboplastin, which converts

prothrombin into thrombin. Simultaneously, fibrinogen is converted into fibrin, increasing platelet, erythrocyte, and leukocyte aggregation, ultimately forming the thrombus [6].

**Figure – 1:** PRISMA flow diagram.




**Morphology of Thrombi**

White thrombi (arterial thrombi) are observed at sites of endothelial injury and are composed primarily of platelet aggregates, presenting a coral-like appearance. In contrast, red thrombi (venous thrombi) occur in areas without endothelial damage and consist mainly of fibrin and erythrocytes. They have a cylindrical, red, friable, opaque appearance and typically occlude the entire vessel lumen [7].

**Embolism**

Emboli are defined as fragments that detach from a thrombus, travel through the bloodstream, and lodge in a different vascular territory. As they are part of a thrombus, emboli contain thrombin, and once deposited in another site, they can trigger a local thrombotic process. This explains why obstruction may occur either due to the embolus itself - if it is large enough to cause occlusion - or because its lodging initiates thrombus formation, ultimately leading to impaired perfusion [8].

Most emboli originate from the proximal veins of the lower extremities, such as the iliac, femoral, and popliteal veins. Embolization from calf vein thrombosis is rare, as these thrombi often resolve spontaneously; however, if untreated or unresolved, they may extend to proximal veins, where the risk of pulmonary embolization is

significantly higher. Pulmonary embolization from renal veins or upper extremity veins may occur but is uncommon [9].

## Classification

### Temporal Classification

When signs and symptoms occur immediately after vascular obstruction, the condition is classified as acute. If symptoms develop over days to weeks, it is considered subacute. When symptoms of pulmonary hypertension evolve slowly, the condition is classified as chronic, as seen in chronic thromboembolic pulmonary hypertension (CTEPH) [10].

### HemodynamicStability

- Hemodynamically unstable (massive or high-risk pulmonary embolism): Defined by hypotension with systolic blood pressure <90 mmHg, a drop >40 mmHg lasting at least 15 minutes, or the need for vasopressors or inotropic agents to correct hypotension. This may result from large thrombi or smaller thrombi in patients with underlying cardiovascular disease, emphasizing the patient's hemodynamic status [10].
- Hemodynamically stable: subdivided into intermediate-risk (submassive) and low-risk pulmonary embolism.
    - Intermediate-risk (submassive): presence of right ventricular dysfunction.
    - Low-risk: absence of right ventricular dysfunction.

Patients who do not meet criteria for instability may present with mild symptoms or may be asymptomatic. Hypotension may be borderline but does not require vasopressors or inotropes, and fluid therapy may be considered [10].

### Anatomical Location

Saddle pulmonary emboli are those lodged at the bifurcation of the main pulmonary artery and may extend into the right or left pulmonary arteries. The main pulmonary arteries are involved in approximately 3–6% of cases. Thrombi may extend beyond the main arteries to involve lobar, segmental, or subsegmental branches. Pulmonary embolism may be unilateral or bilateral [10].

### Presence or Absence of Symptoms

A symptomatic patient is one who presents with clinical manifestations leading to diagnostic confirmation. Conversely, asymptomatic pulmonary embolism is identified incidentally during imaging studies performed for other reasons [9].

## Pathophysiology

The thrombus may detach and migrate to the pulmonary circulation, generating several pathophysiological changes, including:

### Hemodynamic Changes

These alterations are caused by mechanical obstruction. Pulmonary arterial pressure increases proportionally to the degree of obstruction produced by the embolus. If this fraction exceeds normal limits, right ventricular dilation may occur. Reduced pulmonary blood flow and ventricular dilation decrease preload, while increased peripheral vascular resistance alters stroke volume and cardiac output, explaining the resulting hypotension. In cases of complete obstruction, cardiac output drops to zero, leading to circulatory collapse and sudden death [6, 9].

### Ventilation–Perfusion Mismatch

Perfusion is reduced or absent distal to the occlusion, resulting in increased ventilation–perfusion ratios and impaired carbon dioxide elimination. Hyperventilation occurs as a compensatory mechanism; however, persistent hypoperfusion disrupts surfactant production, leading to surfactant depletion, alveolar edema, alveolar collapse, atelectasis, and ultimately hypoxemia. Respiratory drive stimulation produces hypocapnia and alkalosis, whereas shock states may result in hypercapnia and acidosis [6, 9].

### Hypoxemia

The most frequent finding in acute pulmonary embolism is hypoxemia associated with low $PaCO_2$ levels. Several mechanisms contribute to hypoxemia:

1. Cardiopulmonary arteriovenous shunting, where abnormal pulmonary vessels form a capillary bypass to reduce pulmonary circulation pressure, and intracardiac right-to-left shunting may occur through a patent foramen ovale [6, 9, 11].
2. Ventilation–perfusion mismatch, compounded by histamine-induced bronchoconstriction and serotonin-mediated vasoconstriction, creating regions of good perfusion with poor ventilation and vice versa, ultimately causing atelectasis due to reduced surfactant [9, 11].
3. Reduced cardiac output, leading to systemic desaturation and worsening hypoxemia [11].
4. Impaired oxygen diffusion, when the alveolar–capillary membrane is affected, causing blood to preferentially flow through unaffected areas, thereby reducing effective oxygenation [11].

Together, these mechanisms result in increased right ventricular afterload due to mechanical obstruction and hormonally mediated vasospasm. The combined effect of increased afterload and tachycardia leads to right ventricular dilation, increased myocardial oxygen demand, reduced cardiac output, myocardial ischemia due to insufficient oxygen supply, and eventual heart failure [11].

**Risk Factors**

Risk factors for pulmonary embolism are explained by the classic triad described by Rudolf Virchow, consisting of endothelial injury, hypercoagulability, and venous stasis. Based on this framework, risk factors include immobilization or prolonged bed rest, central venous catheters, cancer, obesity, advanced age, smoking, arterial hypertension, hypercholesterolemia, diabetes mellitus, chronic obstructive pulmonary disease, chronic kidney disease, nephrotic syndrome, prolonged air travel, autoimmune diseases, genetic coagulation disorders, estrogen-containing contraceptives, pregnancy, hormone replacement therapy, surgery, and trauma [6, 9, 11, 12, 13].

The most prevalent factor is venous stasis due to immobilization in hospitalized patients, particularly those undergoing surgical procedures. Additionally, malignancy and tissue injury associated with surgery are the two main causes of coagulation system activation [6].

Special populations include patients with cancer, pregnant women, patients with stroke, hospitalized medical, surgical, or gynecological patients, individuals with nephrotic syndrome, spinal cord injury, total joint arthroplasty or joint replacement, and those with inherited thromboembolic disorders. Overall incidence is higher in men than in women; however, prevalence increases among women with advancing age. Regarding mortality, African American populations are the most affected, followed by White populations [9].

**Clinical Presentation**

The classic triad of pulmonary embolism consists of dyspnea, pleuritic chest pain, and hemoptysis; however, this combination is present in only a small percentage of patients. It is important to emphasize that dyspnea at rest or with exertion is the most common symptom, followed by pleuritic chest pain and cough, while tachypnea is the most prevalent physical sign after findings suggestive of deep vein thrombosis (DVT).

Other associated symptoms and signs include substernal chest pain, fever, tachycardia, presyncope and syncope, unexplained hypotension, cyanosis, jugular venous distension, accentuated second heart sound on auscultation, paradoxical bradycardia, atrial fibrillation, wheezing, and decreased breath sounds [6, 10, 12, 14, 15, 16].

Signs of deep vein thrombosis should also be assessed, including edema, tenderness, warmth, and erythema of the calf or thigh. Calf pain may vary in intensity and may worsen with ambulation or compression of muscle compartments. Pain elicited by compression against the bony plane is known as Olow's sign. Lowenberg's sign is considered positive when pain is reproduced by inflating a blood pressure cuff to 60–180 mmHg, while Homans' sign refers to pain induced by dorsiflexion of the foot, stretching the gastrocnemius–soleus complex. Pratt's sign and the presence of a palpable venous cord may also be observed. [6, 7, 10, 16].

**Diagnosis**

The diagnostic approach should begin with an assessment of the patient's hemodynamic stability, as this determines the need for aggressive management. In hemodynamically stable patients, clinical evaluation can be combined with pretest probability assessment, which involves the use of scoring systems to estimate disease likelihood and guide further diagnostic testing. In unstable patients, ultrasonographic methods may be used to justify immediate initiation of treatment [10, 16].

**Clinical Evaluation**

Clinical evaluation consists of a thorough medical history and physical examination aimed at identifying relevant patient information, risk factors, potential triggers, and symptoms suggestive of pulmonary embolism. Physical examination includes vital sign assessment, a complete systemic examination, and targeted evaluation for signs indicative of pulmonary embolism [10, 16].

**Pretest Probability Scores for Pulmonary Embolism**

Pretest probability tools guide clinical decision-making. Among these is the Pulmonary Embolism Rule-out Criteria (PERC), described in Annex 1. Patients who meet all eight criteria are considered to have a low probability of pulmonary embolism and do not require further testing. Patients who fail to meet these criteria may require additional diagnostic studies [16, 17].

The Wells score and its modified version (Annex 2) are interpreted as follows: using the traditional scale, probability is classified as low (<2), moderate (2–6), or high (>6). In the modified version, patients are categorized as "pulmonary embolism unlikely" (<4) or "pulmonary embolism likely" (>4) [16, 17].

The Geneva score (Annex 3) classifies probability as low (0–3), intermediate (4–10), or high (>11). In the simplified version, all variables are assigned one point, and probability is categorized as low (0–2) or high (>3) [16, 17].

**Laboratory Tests**

Laboratory tests are not diagnostic for pulmonary embolism but may be useful in excluding differential diagnoses or providing prognostic information once pulmonary embolism is confirmed. Commonly requested tests include a complete blood count and serum chemistry panel, with expected findings such as leukocytosis, elevated erythrocyte sedimentation rate, increased serum lactate, lactate dehydrogenase, and aspartate aminotransferase. Renal function tests, including serum creatinine and estimated glomerular filtration rate, are useful to ensure safe administration of contrast agents for imaging studies [16].

Arterial blood gas analysis may be normal or abnormal. When abnormal, findings typically include hypoxemia with an increased alveolar–arterial oxygen gradient, respiratory alkalosis, and hypocapnia. As previously mentioned, the presence of hypercapnia and respiratory and/or lactic acidosis should raise suspicion of shock - particularly obstructive shock - or respiratory arrest [16].

Elevated levels of B-type natriuretic peptide (BNP) or N-terminal pro-BNP, as well as troponin I or T, have limited diagnostic value but are useful for risk stratification and prognostic assessment. In pulmonary embolism, troponin

elevation typically resolves within approximately 40 hours, unlike acute myocardial injury, where elevations tend to persist longer [16].

D-dimer testing is an excellent rule-out tool for pulmonary embolism when values are within the normal range (<500 ng/mL), provided the clinical risk is low to moderate. In high-risk patients, D-dimer testing is less useful and often unnecessary. Elevated D-dimer levels are not specific for pulmonary embolism and may be observed in various acute or inflammatory conditions, such as recent surgery or trauma, pregnancy or postpartum state, rheumatologic disease, renal dysfunction, or sickle cell anemia; however, elevated values justify further advanced imaging. The standard cutoff is useful in patients under 50 years of age; in older patients, an age-adjusted cutoff (age × 10) is recommended [16].

**Imaging Studies**

Electrocardiography may show common but nonspecific abnormalities. Classic findings traditionally associated with pulmonary embolism - such as the S1Q3T3 pattern, right ventricular hypertrophy, or new right bundle branch block - are uncommon (approximately 10% of cases). The most frequent findings are sinus tachycardia and nonspecific ST-segment and T-wave changes (around 70% of abnormalities). Poor prognostic indicators include atrial arrhythmias, tachycardia or bradycardia, inferior Q waves (II, III, and aVF), and the less common patterns mentioned above [16].

Chest radiography is typically obtained to exclude alternative diagnoses or to assess suitability for ventilation–perfusion (V/Q) scanning. Findings are often nonspecific, such as atelectasis or pleural effusion. Classic signs - including Hampton's hump, Westermark's sign, and Palla's sign - are uncommon but, when present, should raise suspicion for pulmonary embolism [16].

Computed tomography pulmonary angiography (CTPA) is the gold standard for the diagnosis of pulmonary embolism, with sensitivity and specificity exceeding 90%. Contraindications include contrast allergy and renal insufficiency. In such cases, alternative modalities may be used, including venous ultrasound, ventilation–perfusion scanning, or magnetic resonance pulmonary angiography if available. When clinically feasible, CTPA may be delayed to allow for contrast allergy premedication or intravenous hydration in patients with renal impairment [16].

Ventilation–perfusion (V/Q) scintigraphy is used when CTPA is contraindicated, inconclusive, or when additional testing is required. A prior chest radiograph is necessary, and the patient must remain still for 30–60 minutes. Results are reported as normal, low, intermediate, or high probability and must be interpreted in conjunction with clinical probability scores [16].

**Other Diagnostic Modalities**

Alternative studies to CTPA - though less sensitive - include lower-extremity Doppler ultrasound, catheter-based pulmonary angiography, magnetic resonance pulmonary angiography (both operator-dependent), and echocardiography [16].

Compression ultrasonography of the lower extremities is not diagnostic for pulmonary embolism; however, evidence of DVT in the context of clinical suspicion may justify initiation of anticoagulant therapy. Whole-leg ultrasonography is recommended. If ultrasound is negative but clinical suspicion remains high, empirical anticoagulation and further imaging may be considered. If ultrasound is negative and clinical suspicion is low to moderate, anticoagulation is not required, though serial ultrasounds may be necessary until definitive imaging is obtained [16].

Catheter-based pulmonary angiography was the former gold standard for pulmonary embolism diagnosis and involves catheter insertion into the

right heart chambers for contrast injection. Currently, it is reserved for cases where CTPA or V/Q scanning is unavailable or as a combined diagnostic and therapeutic modality aimed at thrombus fragmentation or removal [16].

**Emerging Diagnostic Techniques**

Investigational imaging modalities include dual-energy computed tomography, single-photon emission computed tomography (SPECT), and multiorgan ultrasound. Dual-energy CT reduces contrast exposure compared with standard CTPA and increases sensitivity by generating contrast-enhanced perfusion maps. SPECT does not require contrast administration, involves lower radiation exposure, and improves detection of small emboli; it is as sensitive as CTPA and more sensitive than conventional V/Q scintigraphy [16].

**Risk Stratification Scores**

Once pulmonary embolism is diagnosed, risk stratification is essential to determine prognosis. Tools such as the Pulmonary Embolism Severity Index (PESI) and its simplified version are commonly used. Due to greater clinical practicality, the simplified PESI is preferred, as the full version includes numerous variables that limit routine use. Interpretation classifies patients as low risk (score 0) or high risk (score ≥1). Annex 4 outlines the clinical variables included in this index and their respective scores [17, 18].

**Treatment**

**Pharmacological Management of Acute Pulmonary Embolism**

Initial treatment of acute pulmonary embolism should include medical stabilization and symptom relief, resolution of the vascular obstruction, and prevention of recurrence. Most of these objectives are achieved through anticoagulant therapy, which prevents thrombus progression while allowing the endogenous fibrinolytic system to resolve the obstruction and promote collateral circulation [19, 20, 21].

Anticoagulation is the treatment of choice for both the acute phase (first 10 days) and long-term management, provided the patient is hemodynamically stable and has no contraindications. When indicated, anticoagulation should be initiated as early as possible. Several anticoagulant options are available. The classic regimen includes vitamin K antagonists, which require an initial overlap with parenteral anticoagulation, such as low-molecular-weight heparin (LMWH). This overlap should be maintained for a minimum of 5 days and until two consecutive international normalized ratio (INR) measurements are within the therapeutic range (2.0–3.0) [20–25].

A particular scenario is subsegmental pulmonary embolism. Although anticoagulation is recommended in most cases, uncertainty remains as to whether this represents a false-positive finding or a more benign entity due to its low mortality. However, the main concern is a higher risk of recurrence at 90 days. Selected patients with involvement of a single subsegmental artery, absence of concomitant DVT, adequate cardiopulmonary reserve, and no additional risk factors may be considered for management without anticoagulation on an individualized basis [26].

Vitamin K antagonists, such as warfarin and acenocoumarol, act by inhibiting the vitamin K–dependent synthesis of coagulation factors II, VII, IX, and X. These agents interact with multiple drugs and foods and therefore require strict monitoring. They are preferred in patients with antiphospholipid syndrome, presence of lupus anticoagulant, and severe renal failure [20, 26].

Another option is direct oral anticoagulants (DOACs), which inhibit factor Xa - except for dabigatran, which directly inhibits thrombin. DOACs demonstrate comparable efficacy to conventional therapy, with the added advantages of not requiring routine monitoring and being associated with a lower risk of major bleeding. For these reasons, some authors propose DOACs as first-line therapy. Contraindications include severe renal failure, chronic liver disease,

pregnancy, and lactation. Rivaroxaban and apixaban can be used as monotherapy, whereas dabigatran and edoxaban require a 5–10-day overlap with parenteral anticoagulation [20–23, 25, 26].

Therapeutic dosing regimens include rivaroxaban 15 mg twice daily for the first 3 weeks followed by 10 mg once daily; apixaban 10 mg twice daily for 7 days followed by 5 mg twice daily; edoxaban 60 mg once daily; and dabigatran 150 mg twice daily [22].

For parenteral anticoagulation, LMWH or fondaparinux is preferred over unfractionated heparin (UFH), with no demonstrated superiority among LMWH agents. Therapeutic doses include enoxaparin 1 mg/kg every 12 hours, tinzaparin 175 IU/kg once daily, dalteparin 100 IU/kg every 12 hours, and nadroparin 86 IU/kg every 12 hours. These agents exert their effect by binding to antithrombin and inducing a conformational change. Fondaparinux is a selective factor Xa inhibitor and represents a safe alternative in patients who develop heparin-induced thrombocytopenia [20, 23].

UFH acts by binding to antithrombin and is preferred in patients with potential need for fibrinolytic therapy, severe renal failure, or morbid obesity. Dosing consists of an initial bolus of 80 IU/kg or 5,000 IU regardless of weight, followed by continuous infusion at 18 IU/kg/hour, targeting an activated partial thromboplastin time (aPTT) of 46–70 seconds. When administered prior to fibrinolytic therapy, UFH should be discontinued during administration of urokinase or streptokinase but may be continued during recombinant tissue plasminogen activator infusion. The main complication of both heparin formulations is heparin-induced thrombocytopenia [20, 23].

In patients receiving LMWH or fondaparinux who are candidates for systemic fibrinolysis, initiation of UFH infusion should be delayed until 12 hours after the last twice-daily LMWH dose or 24 hours after the last once-daily LMWH or fondaparinux dose [23].

Supplemental oxygen should be considered in patients with oxygen saturation below 90–92%, and invasive mechanical ventilation should be evaluated if necessary. Part of the increased pulmonary vascular resistance is due to vasoconstriction rather than mechanical obstruction by the embolus. Supplemental oxygen improves pulmonary vascular resistance by acting as a potent selective pulmonary vasodilator [20, 23].

In a study comparing two groups - one receiving LMWH plus supplemental oxygen at 7 L/min via face mask (and the same flow via nasal cannula during meals) and the other receiving anticoagulation alone - transthoracic echocardiography was performed at days 2 and 7, along with cardiac biomarkers at 24 hours after diagnosis. The group receiving supplemental oxygen demonstrated mild normalization of right ventricular size and a reduction in the right-to-left ventricular diameter ratio [19].

The study concluded that supplemental oxygen led to echocardiographic improvement, reflecting protection of right ventricular function during endogenous thrombolysis and reversal of hypoxic vasoconstriction, thereby preventing pulmonary toxicity, ischemia, and systemic vasoconstriction with target-organ hypoperfusion [19].

Patients with hemodynamic instability may benefit from moderate fluid administration; however, volumes should not exceed 500 mL of normal saline. In cases of cardiogenic shock, norepinephrine is the inotropic agent of choice [21, 23].

Systemic fibrinolysis is the treatment of choice for patients with hemodynamic instability, as it is the only therapy capable of rapidly lysing the thrombus and preventing or reversing right ventricular failure. Nevertheless, its use is

discouraged in hemodynamically stable patients due to the risk of major intra- and extracranial bleeding. Two regimens are described: an accelerated 2-hour regimen and a prolonged regimen, with preference given to the accelerated approach [20, 21, 23–26].

Fibrinolytic agents and dosing for prolonged and accelerated regimens include:

- Streptokinase: 250,000 IU over 30 minutes followed by 100,000 IU/hour for 12–24 hours, or 1.5 million IU over 2 hours.
- Urokinase: 4,400 IU/kg over 10 minutes followed by 4,400 IU/kg/hour for 12–24 hours, or 3 million IU over 2 hours.
- Recombinant tissue plasminogen activator: 100 mg over 2 hours (full dose) or 0.6 mg/kg over 15 minutes up to a maximum of 50 mg (reduced dose).

**Interventional Management of Acute Pulmonary Embolism**

Surgical embolectomy or catheter-directed percutaneous intervention is indicated in patients with hemodynamic instability who have contraindications to systemic fibrinolysis or in those who fail fibrinolytic therapy. The choice of technique depends on institutional expertise and availability [20, 23–26].

Mechanical aspiration thrombectomy via percutaneous catheter has been proposed for intermediate–high-risk patients, which contrasts with guideline recommendations favoring anticoagulation alone in this group. Additionally, fibrinolytic therapy has not shown mortality benefit in this population due to hemodynamic consequences and major bleeding risk. In one study, mechanical thrombectomy was performed in both hemodynamically stable and unstable patients via femoral access using an 8-F Indigo CAT8 XTORQ catheter [27].

The study concluded that this technique is safe in intermediate–high-risk patients, demonstrating improvement in right ventricular function and respiratory parameters, including oxygen saturation and $PaO_2/FiO_2$ ratio, within the first 24 hours. A lower complication rate was observed, with major bleeding occurring only in patients who underwent fibrinolysis due to hemodynamic instability [27, 28].

Another proposed technique combines low-dose catheter-directed fibrinolysis with percutaneous aspiration thrombectomy. Using a jugular approach, an 8-F angled pigtail catheter is introduced to administer a local bolus of 100,000–250,000 IU of urokinase, followed by continuous aspiration thrombectomy with an Indigo CAT8 catheter. The primary response observed was a reduction in pulmonary artery systolic pressure, followed by hemodynamic stabilization and improvement in right ventricular function. No major bleeding complications were reported; minor complications included arrhythmias, minor bleeding, hematomas, and hematuria, all self-limited [28].

The study concluded that the combination of both techniques results in significant reduction of pulmonary hypertension and improvement in right ventricular function, achieving a high survival rate and supporting its role as a safe and effective first-line treatment in hemodynamically unstable acute pulmonary embolism [28].

Inferior vena cava (IVC) filters are indicated in patients with contraindications to anticoagulation, recurrent thromboembolism despite adequate anticoagulation, or as primary prophylaxis in high-risk DVT. Their purpose is to prevent embolization from deep veins. Placement is typically infrarenal and percutaneous; however, suprarenal placement is preferred when thrombus involves the renal veins. Retrievable filters are favored over permanent devices. Immediate complications include insertion-site thrombosis, bleeding, infection, and malposition. Long-term complications include recurrent DVT, post-thrombotic syndrome, filter occlusion, device migration, and IVC perforation, supporting filter

removal as soon as adequate anticoagulation is achieved [20, 21, 23–26].

IVC filter placement is discouraged in patients with symptomatic acute pulmonary embolism, concomitant DVT, and poor cardiopulmonary reserve, as studies have shown no significant reduction in thrombotic recurrence during the first 3 months of follow-up in this setting [26].

**Maintenance Management of Acute Pulmonary Embolism**

This phase spans from day 11 through the first 3–6 months. All patients should receive a minimum of 3 months of anticoagulation, with duration individualized according to recurrence risk, bleeding risk, and patient preferences. In patients with a major transient and resolved risk factor, anticoagulation may be discontinued after 3 months. Patients with persistent risk factors, antiphospholipid syndrome, two or more episodes of idiopathic DVT, or idiopathic pulmonary embolism in men should receive indefinite anticoagulation [20, 22, 26].

Additional tools that may guide treatment duration include clinical characteristics, D-dimer testing, and thrombophilia studies, the latter having limited utility. Relevant subgroups include women with idiopathic pulmonary embolism, patients with embolism secondary to a minor transient resolved risk factor, patients wishing to discontinue anticoagulation, and those in whom the risk–benefit balance of indefinite therapy is uncertain [26].

Reduced-dose DOACs, such as apixaban 2.5 mg twice daily and rivaroxaban 10 mg once daily, have been shown to be effective and safe in preventing recurrence during the extended or chronic phase (after the first 6 months) [20].

Pulmonary Embolism Rule-out Criteria (PERC) is as per **Table – 1**. Wells score is as per **Table – 2**. Ginebra Score is as per **Table – 3**. Simplified Pulmonary Embolism Severity Index (sPESI) is as per **Table - 4**.

**Table – 1:** Pulmonary Embolism Rule-out Criteria (PERC).

| |
|---|
| Age < 50 years |
| Heart rate < 100 beats per minute |
| Oxygen saturation > 95% |
| Absence of hemoptysis |
| No estrogen use |
| No previous history of deep vein thrombosis (DVT) or pulmonary embolism (PE) |
| No clinical signs of deep vein thrombosis |
| No surgery or trauma requiring hospitalization within the previous 4 weeks |

Source: Adapted from Thompson T, Kabrhel C, Pena C. Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism. 2024 [16].

**Table – 2:** Wells Score.

| **Parameters** | **Score** |
|---|---|
| Clinical signs of deep vein thrombosis | 3 |
| An alternative diagnosis less likely than pulmonary embolism | 3 |
| Heart rate > 100 beats per minute | 1.5 |
| Immobilization for more than 3 days or surgery within the previous 4 weeks | 1.5 |
| Previous history of deep vein thrombosis or pulmonary embolism | 1.5 |
| Hemoptysis | 1 |
| Malignancy | 1 |

Source: Adapted from Thompson T, Kabrhel C, Pena C. Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism. 2024 [16].

**Table – 3:** Ginebra Score.

| | Variable | Points |
|---|---|---|
| **Risk factors** | Age >65 years | 1 |
| | History of prior DVT/PE | 3 |
| | Surgery or lower-limb fracture within the last 4 weeks | 2 |
| | Active malignancy or malignancy cured within the last year | 2 |
| **Symptoms** | Unilateral lower-limb pain | 3 |
| | Hemoptysis | 2 |
| **Signs** | Heart rate 75–94 bpm | 3 |
| | Heart rate >95 bpm | 5 |
| | Pain on deep venous palpation of the lower limb and unilateral edema | 4 |

Source: Adapted from Thompson T, Kabrhel C, Pena C. Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism. 2024 [16].

**Table – 4:** Simplified Pulmonary Embolism Severity Index (sPESI).

| Clinical characteristics | Score |
|---|---|
| Age >80 years | 1 |
| History of cancer | 1 |
| Chronic cardiopulmonary disease | 1 |
| Heart rate >110 bpm | 1 |
| Systolic blood pressure <100 mmHg | 1 |
| Arterial oxygen saturation <90% | 1 |

Source: Adapted from Weinberg A, Rali P. Treatment, prognosis, and follow-up of acute pulmonary embolism in adults. 2024 [18].

## Conclusion

Acute pulmonary embolism is defined as obstruction of the pulmonary artery or its branches due to embolization of a thrombus of varying etiology, ultimately leading to increased pressure and involvement of the right cardiac chambers.

Its clinical presentation is often nonspecific, allowing the condition to go unrecognized or be confused with other disorders with overlapping symptoms; therefore, maintaining a high index of suspicion is essential for timely diagnosis and management.

The cornerstone of pharmacological treatment is anticoagulation, preferably with direct oral anticoagulants. However, multiple formulations exist that must be tailored to individual patient characteristics, emphasizing the need for a comprehensive and personalized therapeutic approach.

Catheter-directed interventional management combined with fibrinolytic therapy has demonstrated sufficient safety and efficacy to be considered a first-line option in hemodynamically unstable patients, potentially replacing standalone systemic thrombolysis.

The main post-acute complications are related to treatment itself and recurrence. Maintenance therapy with a minimum of 3 months of anticoagulation - primarily with reduced-dose direct oral anticoagulants - combined with healthy lifestyle measures markedly reduces these complications.

## Ethical Considerations

This study is a systematic review of previously published literature. Therefore, ethical approval from an institutional review board was not required.

**Informed Consent**

Informed consent was not required, as no individual patient data were collected or analyzed.

**Conflict of Interest**

The authors declare no conflicts of interest related to this manuscript.

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