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Review on Pulmonary Embolism Diagnosis and Management Approach

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ABSTRACT

A frequent illness, pulmonary embolism can cause considerable morbidity and mortality. Certain inherited and acquired risk factors predispose vulnerable individuals, and the clinical situation can arouse suspicion. Studies involving people who had non-alcoholic fatty liver disease were sought after in the Medline, Pubmed, Embase, NCBI, and Cochrane databases. Analysis was done on incidence, etiology, and treatment options. Because PE presents differently in each patient, it is important to take that into account. The accuracy of the diagnosis decreases with patient age. The diagnosis is difficult because of comorbidities like bronchopneumonia, Chronic Obstructive Pulmonary Disease (COPD), asthma, or chronic fibrosing pulmonary processes The clinical diagnosis of pulmonary embolism is extremely ambiguous because none of the symptoms or signs related to pulmonary embolism are distinctive and can all be caused by other cardiorespiratory illnesses. Patients who have an abnormal perfusion lung scan require more sophisticated care. It can be difficult to diagnose conditions when they have comorbid conditions like bronchopneumonia, chronic obstructive pulmonary disease (COPD), asthma, or chronic fibrosing pulmonary processes. The diagnosis of PE can be made quickly in patients who have DVT, though. Thrombosis of the renal, iliac, and inferior vena cava veins, as well as DVT, were the main causes of up to 85% of PE cases.

Keywords: *Pulmonary embolism (PE), Pulmonary embolism response team, Deep vein thrombosis (DVT), Venous thromboembolism (VTE)*

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INTRODUCTION

A common condition that can lead to death or disability is pulmonary embolism (PE). It is the third leading cause of death from cardiovascular disease, behind myocardial infarction and cerebrovascular stroke. Patients present with a wide range of symptoms and indications, making detection difficult. Certain inherited and acquired risk factors predispose vulnerable individuals, and the clinical situation can arouse suspicion. The best laboratory screening test is D-dimer concentration in blood, while chest CT has become the most widely used imaging test. Before hemodynamic decompensation and the development of cardiogenic shock, treatment requires early and accurate risk classification. The core of therapy is anticoagulation (Pineda *et al*., 2001).

Even if the blood pressure is normal at presentation, high-risk individuals may require escalation of therapy with thrombolysis or embolectomy if they have right ventricular dysfunction on echocardiography and higher than average troponin concentrations. Patients should have prophylactic measures prescribed by their doctors to avoid PE when they are admitted to medical wards or when they have surgery. Patients with a high risk of thromboembolism should maintain prophylaxis for about a month after leaving the hospital (Goldacre *et al*., 2000).

PE is a severe and potentially fatal Deep Venous Thrombosis consequence (DVT). Because 30% of persons with DVT develop symptoms of PE, it is essential to prevent, diagnose, and treat

the condition. Including asymptomatic episodes increases the likelihood of PE in 50–60% of DVT patients. DVT and PE are both symptoms of the same situation, thromboembolic disease. In a population of 10 million, 20,000 new thromboembolic illnesses may be expected each year. PE will occur in 10,000 of these patients, with 6,000 experiencing symptoms and 900 dying during the first trimester (Smulders, 2001).

MATERIALS AND METHODS

PubMed database was used for article selection, and the following keys were used in the Mesh (("Pulmonary embolism "[Mesh]) AND ("signs and symptoms" [Mesh]) OR ("Management" [Mesh])).

Regarding the inclusion criteria, the management and features of pulmonary embolism rosea were taken into consideration when choosing which articles to include. All other articles that did not use one of these subjects as their primary endpoint were excluded.

Out of 1,202 articles indexed in the previous two decades, about 90 publications were selected as the most clinically pertinent, and their full texts were assessed. After careful review, 31 of the 90 were decided to be included. Using the reference lists from the acknowledged and linked studies, additional studies and publications were located. To aid practicing physicians in assessing pulmonary embolism in the most straightforward and practical way possible, expert consensus recommendations and commentary were added where appropriate .

Epidemiology

Despite advances in detection and treatment over the past 30 years, pulmonary embolism (PE), a moderately common acute cardiovascular illness with high early fatality rates, has remained largely unchanged. Due to pulmonary bed obstruction, PE can result in acute right ventricular (RV) failure, a condition that may be fatal. Early diagnosis is essential because the majority of patients pass away within the first few hours of presenting. Emergency responses are frequently quite successful, and RV failure may be reversible. The initial course of treatment, depending on the degree of the PE, concentrates on reestablishing adequate blood flow through the pulmonary bed and preventing PE recurrence .

The best way to select an appropriate treatment plan is through risk stratification. This involves assessing variables like hemodynamic impact—the most reliable predictor of short-term prognosis—the morphological extent of PE, the patient's cardiovascular and pulmonary systems, the degree of neurohumoral adaptation, and potential drug side effects. Although there is a lack of precise epidemiological data, it is estimated that 124 people will develop venous thrombosis for every 100,000 people, and the incidence of PE will be between 60 and 70 per 100,000. (Oger, 2000). The annual incidence rates of PE and venous thrombosis range from 0.5 to 1.0 per 1000 people, according to European guidelines for the diagnosis and treatment of PE (Torbicki *et al*., 2008). The accurate estimates, however, are probably much higher given that up to 40% to 50% of people with deep vein thrombosis (DVT) may experience silent PE development (Meignan *et al*., 2000). In addition, postmortem studies have shown that PE was found in 30%–45% of patients prior to death (Pineda *et al*., 2001).

Acute PE is the third most prevalent type of cardiovascular disease, behind coronary artery disease and stroke. While clinical data suggest that most instances of PE occur between the ages of 60 and 70, autopsy data reveal that the highest incidence occurs between the ages of 70 and 80. Untreated, acute PE has a large mortality rate (as high as 30%), whereas diagnosed and treated PE has a fatality rate of 8%. Up to 10% of people with acute PE die abruptly. Two of three patients who succumb to PE die two hours after the presentation (Goldhaber *et al*., 1999).

Risk factors

Pulmonary embolism has many risk factors. Together with other presentational characteristics, the mentioned risk factors aid in establishing the clinical impression or phenomenology of pulmonary embolism presence or absence.

In the past, there were high rates of morbidity and mortality associated with pulmonary embolism. The ability to identify "disease" in what was previously believed to be a normal lung function—the filtering of small clots—implies that we can now identify even the smallest pulmonary emboli. The mortality rate for otherwise healthy individuals with pulmonary embolism and normal physiology is close to zero in the outpatient setting. (Newman & Schriger, 2011). Compared to the frequency of pulmonary embolisms diagnosed and treated in this category, this is less fatal. Numerous studies show that tiny pulmonary emboli are temporary and common, so the diagnosis of pulmonary embolism in the modern era should not "chill the marrow of clinicians." (Newman & Schriger, 2011).

The diagnosis of pulmonary embolism can, without a doubt, be devastating and tragic, but doctors have trouble locating pulmonary emboli of different sizes. At some point along the spectrum, trying to diagnose every pulmonary embolus—even those that the data indicates are "normal"—will start to cause more harm than good. Figuring out where it is is difficult.

Examples of surgery include major gastrointestinal tract surgery, lower limb surgery, pelvic or abdominal cancer surgery, multiple trauma, and spinal cord injury with paresis .

Chronic and acute illnesses like inflammatory bowel disease, myocardial infarction, active rheumatic disease, nephrotic syndrome, acute respiratory failure, and chronic lung disease are examples of risk factors. Some factors associated with malignancy include myeloproliferative neoplasms, active malignancy, and cancer therapy. Hormonal factors include pregnancy or the early postpartum period, the oral contraceptive pill, hormone replacement therapy, and known thrombophilia. A body mass index greater than 30 kg/m2, varicose veins, venous stasis, a history of deep vein thrombosis or pulmonary embolism, and prolonged iimmobilization/travel are additional risk factors (Goergen *et al*., 2015).

Symptoms and signs

These are numerous and non-specific, and they might not be accurate predictors of the degree of pulmonary vascular blockage even if venography has shown leg vein thrombosis in 70–80% of pulmonary embolism patients at the time of clinical presentation (Hull *et al*., 1983). These thrombi typically cause no symptoms. Indeed, fewer than 20% of individuals with pulmonary embolism had venous thrombosis, according to clinical data (Hull *et al*., 1983). Tachypnea, tachycardia, cyanosis, and altered mental and cognitive states are all signs of a massive pulmonary embolism. The patient could have

hypotension or show signs of pulsus paradoxus. The jugular venous pressure is frequently increased with a noticeable A wave, which can occasionally be challenging to see on a clinical examination. A gallop rhythm, a loud pulmonary component of the second heart sound, and a pronounced right ventricular impulse are frequently seen during a non-specific cardiac examination. A systolic murmur that might either be ejection- or pansystolic-type is often present.

The results of a physical examination of the chest may be normal or show vague anomalies. Evidence of airway blockage is relatively uncommon. Tachycardia and tachypnea may be the only physical symptoms in patients with submassive pulmonary embolism, or there may also be simultaneous cardiac or respiratory manifestations of massive embolism. Reduced chest movement is a symptom of patients' pulmonary infarction or congestive atelectasis. There could be rales, atelectasis, pulmonary consolidation symptoms, and a pleural friction rub.

Fever happens whether there is a pulmonary infarction ; when it does, it often only reaches a low level (101°F). Very infrequently, rigors may be present, with a temperature increase of more than 103°F (Hull *et al*., 1985).

Diagnosis

Pulmonary embolism can present in a variety of ways depending on the size and location of the affected artery, the quantity of emboli, and the patient's underlying condition. Most pulmonary emboli are silent clinically (The urokinase pulmonary embolism trial, 1973).

When clinical manifestations emerge, the patient may exhibit a variety of symptoms. They are as follows: 1) transient dyspnea and tachypnea with no other clinical signs; 2) the syndrome of pulmonary infarction or congestive atelectasis (also known as ischemic pneumonitis, or incomplete infarction), characterized by pleuritic chest discomfort, cough, hemoptysis, pleural effusion, and pulmonary infiltrates on chest X-ray examination; 3) symptoms of right-sided heart failure including severe dyspnea and tachypnea; 4) cardiovascular collapse with hypotension, syncope, and coma; and 5) a variety of less common and less specific clinical features, such as confusion, pyrexia, wheezing, resistant heart failure, and unexplained arrhythmias, promised cardiorespiratory system or the obstruction of a peripheral pulmonary vessel by an embolus, resulting in pulmonary infarction or congestive atelectasis.

Pain

Two types of chest pain are experienced by pulmonary embolism patients. The most common and recognizable type of pain is pleuritic pain, which is brought on by inflammation of the pleura overlying areas of pulmonary infarction or congestive atelectasis. Pulmonary infarction is typically induced by the obstruction of relatively small-sized pulmonary branches, which act as end arteries when occluded acutely. A pulmonary infarction is typically not associated with obstruction of larger, more proximal arteries because the bronchial circulation provides adequate blood flow. Contrarily, a proximal embolus in a large or medium-sized artery may break up, obstruct smaller arteries, and result in pulmonary infarction hours or even days after the acute pulmonary embolic event. Recurrent pulmonary embolism may be mistakenly diagnosed as the cause if it occurs while the patient is receiving treatment.

Hemoptysis

Hemoptysis is a relatively uncommon symptom of pulmonary embolism despite being frequently thought of as one of its hallmarks (The urokinase pulmonary embolism trial, 1973). Its presence suggests that congestive atelectasis or pulmonary infarction, which resulted in alveolar bleeding, has occurred. Hemoptysis can happen alone or in conjunction with pleurisy. It typically starts hours or even days after the original embolic event, similar to pleurisy. Blood-stained sputum, frank blood clots, and conspicuous hemorrhage are all possible types of bleeding. Anticoagulant medication may potentially make hemoptysis worse; however, this rarely results in a severe clinical issue (Hull *et al*., 1983).

Syncope

Sometimes, the earliest and most obvious sign of a pulmonary embolism is syncope. A major pulmonary embolism typically accompanies it, and its root cause is a decrease in cardiac output, which briefly lowers blood pressure and impairs cerebral blood flow. Syncope may occasionally result from submassive pulmonary embolism because of induced cardiac tachyarrhythmias.

Treatment

Anticoagulation is the cornerstone of pulmonary embolism treatment. A large pulmonary embolism may necessitate thrombolytic treatment. One point of contention is whether or not to treat subsegmental pulmonary embolism (SSPE).

Thrombolysis

If a patient's systolic blood pressure exceeds 90 mmHg, the American College of Chest Physicians (ACCP) recommends systemic thrombolysis (Kearon *et al*., 2016). Fibrinolysis is reasonable for patients with a massive acute pulmonary embolism and a manageable risk of bleeding complications, according to the American Heart Association (Jaff *et al*., 2011). Fibrinolysis "may be considered for... submassive acute pulmonary embolism (with)... hemodynamic instability, worsening respiratory insufficiency, severe right ventricular dysfunction, or major myocardial necrosis and low risk of bleeding complications" in patients with a systolic blood pressure of 90 mmHg or bradycardia of 40 beats/minute. The Therapeutic Guidelines advise heparin infusion and alteplase for patients with significant pulmonary emboli, who are typically treated in hospitals (Cardiovascular Working Group, 2017).

Anticoagulation

When used as the first line of treatment for VTE, LMWH decreases morbidity and thrombus size without changing mortality (Robertson & Jones, 2017). The Therapeutic Guidelines (Cardiovascular Working Group, 2017). Acute pulmonary embolism is advised to be treated with enoxaparin 1.5 U/kg daily or 1 U/kg twice daily, as well as dalteparin 200 U/kg, up to 18,000 U daily, or 100 U/kg, up to 9000 U twice daily. The preferred dosage is twice daily if there is a high risk of bleeding or thrombus extension (e.g., older age, obesity, malignancy). In the event that the creatinine clearance is lower than 30 mL/min, the dose must be changed.

The INR should be maintained between 2-3 after starting warfarin. In Australia, rivaroxaban is currently approved and

subsidized for use in the treatment of pulmonary embolism in place of LMWH and warfarin (EINSTEIN–PE Investigators *et al*., 2012). High-risk individuals, such as those with recurrent unprovoked pulmonary embolism and antiphospholipid syndrome, were not included in the clinical trials (EINSTEIN–PE Investigators *et al*., 2012; Yoo *et al*., 2016). Rivaroxaban is taken for three weeks in doses of 15 mg twice daily, then 20 mg once daily. Treatment lasts for six months, but if a temporary major risk factor is present, it may be reduced to three months, or it may last indefinitely if there are persistent major risk factors (e.g., cancer, recurrent unprovoked pulmonary embolism) (EINSTEIN–PE Investigators *et al*., 2012; Ghaffar *et al*., 2021).

CONCLUSION

The clinical diagnosis of pulmonary embolism is extremely ambiguous because none of the symptoms or signs related to pulmonary embolism are distinctive and can all be caused by other cardiorespiratory illnesses . Therefore, objective testing is necessary to confirm or rule out a pulmonary embolism diagnosis. A diagnostic strategy for managing clinically suspected pulmonary embolism is suggested based on the currently available information. Patients who have an abnormal perfusion lung scan require more sophisticated care. Considering the risk of PE is crucial because the variety in presentation makes it challenging to diagnose PE. As a patient gets older, the diagnosis' accuracy declines. It can be difficult to diagnose conditions when they have comorbid conditions like bronchopneumonia, chronic obstructive pulmonary disease (COPD), asthma, or chronic fibrosing pulmonary processes. The diagnosis of PE can be made quickly in patients who have DVT, though. Thrombosis of the renal, iliac, and inferior vena cava veins, as well as DVT, were the main causes of up to 85% of PE cases. Normal recognition of the upper limbs as a major PE source is uncommon (Gholizadeh *et al*., 2021).

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REFERENCES

Cardiovascular Working Group. (2017). *Treatment of deep vein thrombosis and pulmonary embolism*. Melbourne: Therapeutic Guidelines Limited.

EINSTEIN–PE Investigators, Büller, H. R., Prins, M. H., Lensin, A. W., Decousus, H., Jacobson, B. F., Minar, E., Chlumsky, J., Verhamme, P., Wells, P., et al. (2012). Oral rivaroxaban for the treatment of symptomatic pulmonary embolism. *The New England Journal of Medicine*, *366*(14), 1287-1297.

Ghaffar, F. A., Redzuan, A. M., & Makmor-Bakry, M. (2021). Effectiveness of Sildenafil in Pulmonary Hypertension Secondary to Valvular Heart Disease: A Systematic Review and Meta-Analysis. *Archives of Pharmacy Practice*, *12*(3), 55-65.

Gholizadeh, B., Zadeh, F. J., Nabavi, S. S., Moradi-Joo, E., & Baghaei, S. (2021). The Relationship between Quality of Life and Mental Health in Patients with Heart Failure. *Journal: Entomology and Applied Science Letters*, (3), 60-66.

Goergen, S., Tran, H., Jong, I., & Zallman, M. (2015). Suspected pulmonary embolism. *Sydney: The Royal Australian and New Zealand College of Radiologists*.

Goldacre, M. J., Roberts, S., Yeates, D., & Griffith, M. (2000). Hospital admission and mortality rates for venous thromboembolism in Oxford region, UK, 1975-98. *Lancet (London, England)*, *355*(9219), 1968-1969.

Goldhaber, S. Z., Visani, L., & De Rosa, M. (1999). Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). *Lancet (London, England)*, *353*(9162), 1386-1389.

Hull, R. D., Hirsh, J., Carter, C. J., Jay, R. M., Dodd, P. E., Ockelford, P. A., Coates, G., Gill, G. J., Turpie, A. G., Doyle, D. J., et al. (1983). Pulmonary angiography, ventilation lung scanning, and venography for clinically suspected pulmonary embolism with abnormal perfusion lung scan. *Annals of Internal Medicine*, *98*(6), 891-899.

Jaff, M. R., McMurtry, M. S., Archer, S. L., Cushman, M., Goldenberg, N., Goldhaber, S. Z., Jenkins, J. S., Kline, J. A., Michaels, A. D., Thistlethwaite, P., et al. (2011). Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American Heart Association. *Circulation*, *123*(16), 1788-1830.

Kearon, C., Akl, E. A., Ornelas, J., Blaivas, A., Jimenez, D., Bounameaux, H., Huisman, M., King, C. S., Morris, T. A., Sood, N., et al. (2016). Antithrombotic Therapy for VTE Disease: CHEST Guideline and Expert Panel Report. *Chest*, *149*(2), 315-352.

Meignan, M., Rosso, J., Gauthier, H., Brunengo, F., Claudel, S., Sagnard, L., d'Azemar, P., Simonneau, G., & Charbonnier, B. (2000). Systematic lung scans reveal a high frequency of silent pulmonary embolism in patients with proximal deep venous thrombosis. *Archives of Internal Medicine*, *160*(2), 159-164.

Newman, D. H., & Schriger, D. L. (2011). Rethinking testing for pulmonary embolism: less is more. *Annals of Emergency Medicine*, *57*(6), 622-627.

Oger E. (2000). Incidence of venous thromboembolism: a community-based study in Western France. EPI-GETBP Study Group. Groupe d'Etude de la Thrombose de Bretagne Occidentale. *Thrombosis and Haemostasis*, *83*(5), 657-660.

Pineda, L. A., Hathwar, V. S., & Grant, B. J. (2001). Clinical suspicion of fatal pulmonary embolism. *Chest*, *120*(3), 791-795.

Robertson, L., & Jones, L. E. (2017). Fixed dose subcutaneous low molecular weight heparins versus adjusted dose unfractionated heparin for the initial treatment of venous thromboembolism. *The Cochrane Database of Systematic Reviews*, *2*(2), CD001100.

Smulders Y. M. (2001). Contribution of pulmonary vasoconstriction to haemodynamic instability after acute pulmonary embolism. Implications for treatment?. *The Netherlands Journal of Medicine*, *58*(6), 241-247.

The urokinase pulmonary embolism trial. A national cooperative study. (1973). *Circulation*, *47*(2 Suppl), II1–II108.

Torbicki, A., Perrier, A., Konstantinides, S., Agnelli, G., Galiè, N., Pruszczyk, P., Bengel, F., Brady, A. J., Ferreira, D., Janssens, U., et al. (2008). Guidelines on the diagnosis and management of acute pulmonary embolism: the Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). *European Heart Journal*, *29*(18), 2276-2315.

Yoo, H. H., Queluz, T. H., & El Dib, R. (2016). Anticoagulant treatment for subsegmental pulmonary embolism. *The Cochrane Database of Systematic Reviews*, (1), CD010222.