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Pulmonary Embolism With Deep Vein Thrombosis Pasca Negleted Injury Genue Sinistra

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ABSTRACT
Pulmonary embolism is an infarction of lung tissue due to blockage of the pulmonary artery due to an embolic event. Pulmonary Embolism and Deep Vein Thrombosis have the same pathological process. Pulmonary embolism usually originates from a thrombus dislodged from the deep venous system of the lower extremities. In this case study, a 43-years-old woman with present palpitations, dyspnea, dizziness, suddenly syncope, palpation genue sinistra erytema, tenderness, painful, pitting edema, superficial collateral veins without sign of infection. She after fell from a motorbike and taken to Sangkal Putung for a massage therapy. After this, she just stay in bed (imobilisation) more than 5 week. In physical examination, Heart rate 148 beats/minute. Blood preasure of 100/70 mmHg. Decreased ventilation on oxygen saturation 92% with reservoir mask oxygen. In palpation genue sinistra erytema, tenderness, painful, pitting edema, superficial collateral veins without secondary infection. Chest x-rays present of atelectasis in lobus pulmonum dextra. An electrocardiogram present sinus tachycardia, Q wave followed by an inversion of the T wave in lead III accompanied by an S wave in lead I, P pulmonary, New right bundle branch block, Right ventricular strain with inversion T waves in leads V2 to V3. In Pulmonary angiogram examination there is occlusion of the pulmonary artery branch. From clinical diagnosis and physical examination Deep Vein Thrombosis are applied in the form of Well score 75% and Pulmonary embolism probability high risk with geneva score > 60%.

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INTRODUCTION

Pulmonary embolism is an infarction of lung tissue due to blockage of the pulmonary artery due to an embolic event. Pulmonary embolism is a cardiovascular emergency that occurs quite often. Pulmonary embolism is an infarction of lung tissue due to blockage of the pulmonary artery due to an embolic event. Some of the main causes of pulmonary embolism are venous thromboembolism, but other causes such as air embolism, fat embolism, amniotic fluid, tumor fragments, and sepsis are still possible. Pulmonary Embolism and Deep Vein Thrombosis have the same pathological process. Pulmonary embolism usually originates from a dislodged thrombus from the deep venous system of the lower extremities.

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CASE PRESENTATION

Pulmonary embolism is an infarction of lung tissue due to blockage of the pulmonary artery due to an embolic event. Pulmonary Embolism and Deep Vein Thrombosis have the same pathological process. Pulmonary embolism usually originates from a thrombus dislodged from the deep venous system of the lower extremities. In this case study, a 43-years-old woman with present palpitations, dyspnea, dizziness, suddenly syncope, palpation genue sinistra erytema, tenderness, painful, pitting edema, superficial collateral veins without sign of infection. She after fell from a motorbike and taken to Sangkal Putung for a massage therapy. After this, she just stay in bed (imobilisation) more than 5 week. In physical examination, Heart rate 148 beats/minute. Blood preasure of 100/70 mmHg. Decreased ventilation on oxygen saturation 92% with reservoir mask oxygen. In palpation genue sinistra erytema, tenderness, painful, pitting edema, superficial collateral veins without secondary infection. Chest x-rays present of atelectasis in lobus pulmonum dextra. An electrocardiogram present sinus tachycardia, Q wave followed by an inversion of the T wave in lead III accompanied by an S wave in lead I, P pulmonary, New right bundle branch block, Right ventricular strain with inversion T waves in leads V2 to V3. In Pulmonary angiogram examination there is occlusion of the pulmonary artery branch. From clinical diagnosis and physical examination Deep Vein Thrombosis are applied in the form of Well score 75% and Pulmonary embolism probability high risk with geneva score > 60%.

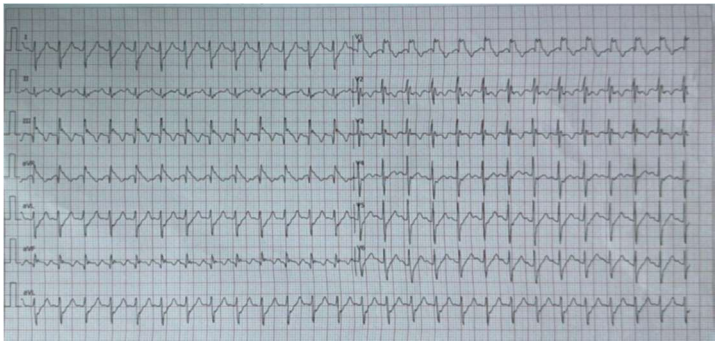

Figure 1. Electrocardiogram (EKG) shows sinus tachycardia, Q waves followed by T wave inversion in lead III accompanied by S waves in lead I, P pulmonary, New right bundle branch block, Right ventricular strain with T wave inversion in leads V2 to V3.

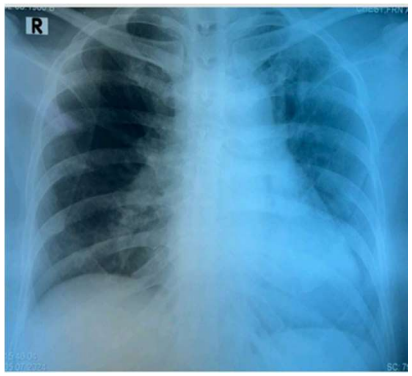

Figure 2. Chest X-ray shows atelectasis in the right pulmonary lobe.

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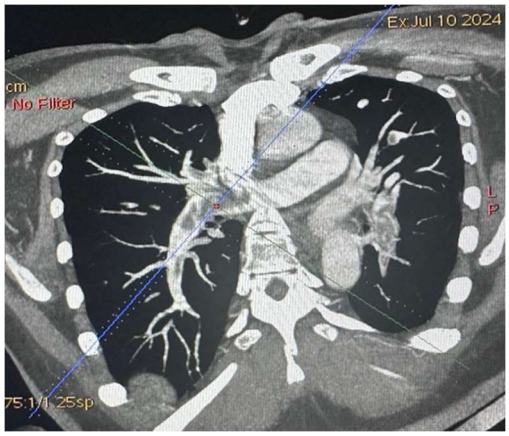

Figure 3. There is occlusion of the pulmonary artery branch visible on the CT scan.

RESULTS AND DISCUSSION

Pulmonary embolism is an infarction of lung tissue due to blockage of the pulmonary artery due to an embolic event. Pulmonary embolism is a cardiovascular emergency that occurs quite often. Pulmonary embolism is Lung tissue infarction occurs due to blockage of the pulmonary artery due to an embolic event. Some of the main causes of pulmonary embolism are venous thromboembolism, but other causes such as air embolism, fat embolism, amniotic fluid, tumor fragments, and sepsis are still possible. Pulmonary Embolism and Deep Vein Thrombosis have the same pathological process. Pulmonary embolism usually originates from a dislodged thrombus from the deep venous system of the lower extremities. According to the American Heart Association, there are several predisposing factors that can increase the likelihood of pulmonary embolism.

Table 1. Predisposition factors for pulmonary embolism

High Risk Factor	Moderate	Low Risk Factor
Fracture	Heart failure	Bed rest for more than 3 days
Hip or knee replacement	Hormone therapy	Laparotomy surgery
General surgery	Malignancy	Obesity
Spinal cord injury	Paralytic stroke	Varicose veins
Major trauma	Postpartum	Antepartum
	Pulmonary embolism	
	Thrombophilia	

Pathophysiology

In 1856, Rudolf Virchow made a postulate which stated that there were three factors that could cause intravascular coagulation, namely:

a. Local trauma to the blood vessel wall, resulting in damage to the vascular endothelium. Usually caused by previous thrombophlebitis, trauma, or surgery.
b. Blood hypercoagulability caused by various treatments, such as: oral contraceptives, hormone therapy, steroid therapy, malignancy, nephrotic syndrome, thrombocytopenia

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due to the use of heparin, protein C deficiency, protein S, antithrombin III, and DIC conditions.

c. Venous stasis, usually caused by prolonged immobilization or bed rest, incompetent venous valves due to previous thromboembolic processes, side effects of anesthesia, congestive heart failure, and cor pulmonale.

Emboli will increase resistance and pressure in the pulmonary arteries which will then release vasoconstrictor compounds, platelet aggregation and mast cells. State of arterial vasoconstriction pulmonary hypertension and hypoxemia will then cause pulmonary arterial hypertension, resulting in increased right ventricular pressure. Furthermore, dilatation and dysfunction of the right ventricle will cause compression of the intraventricular septum to the left side and regurgitation of the tricuspid valve. This can interfere with the ventricular filling process. With reduced left ventricular filling, systemic cardiac output will decrease and reduce coronary perfusion. Myocardial infarction occurs as a result of decreased coronary flow which can cause cardiogenic shock. If not treated quickly, it can cause circulatory failure and death.

In patients who successfully overcome an acute embolic episode, activation of the sympathetic system occurs. Inotropic and chronotropic stimulation increases pulmonary artery pressure which can help to restore pulmonary blood flow and improve left ventricular filling, so that systemic blood pressure becomes stable again. However, this inotropic and chronotropic compensation is not able to maintain right ventricular function for the long term. So there will be an increase in oxygen demand in the right ventricular myocardial muscle accompanied by a decrease in the right ventricular coronary perfusion gradient. As a result, ischemia and failure of right ventricular function occur.

If there is no previous cardioembolic disease, obstruction of less than 20% will cause only minimal hemodynamic disturbances with nonspecific clinical symptoms. When obstruction reaches 30-40%, there will be an increase in right ventricular pressure, but systemic cardiac output can still be maintained with inotropic and chronotropic compensation which increases heart rate and myocardial contractility. When obstruction exceeds 50-60% of the pulmonary artery, compensation will begin to fail. Cardiac output is reduced and right atrial pressure will increase, causing real hemodynamic failure. Meanwhile, respiratory insufficiency in pulmonary embolism is caused by low cardiac output resulting in desaturation of venous blood entering the pulmonary circulation.

Ventilation-perfusion imbalance will cause symptoms of shortness of breath and hypoxemia. In pulmonary emboli that are located more distally, hemodynamic disturbances may not be found. However, symptoms of hemoptysis, pleurisy and mild pleural effusion can be found due to rupture of blood vessels around the alveolar.

Sign and Symptoms

Most of the clinical signs and symptoms presented by pulmonary embolism are nonspecific and can be manifestations of other diseases, such as myocardial infarction and pneumonia. Pulmonary embolism can be asymptomatic to life-threatening with signs and symptoms of severe dyspnea, syncope and cyanosis.

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Table 2. Sign and Symptoms presented by pulmonary embolism

Symptoms	Frekuensi (%)
Dyspnea	73
Pleuritic pain	66
Cough	37
Swelling of the lower limbs	33
Coughing up blood	13
Wheezing	6
Sign	Frekuensi (%)
Respiratory rate more than 20 times per minute	70
Ronchi	51
Heart frequency is more than 100 beats per minute	30
Heart sounds 3 and 4 (gallop)	26
Cyanosis	11
Temperature more than 38.5°C	7

Diagnosis of Pulmonary Embolism and Deep Vein Thrombosis

History and physical examination, these two things are important modalities in establishing a diagnosis of pulmonary embolism. History and physical examination are applied in the form of scoring such as the Well and Geneva scores. Many studies have linked the occurrence of pulmonary embolism with the Well and Geneva scores. The higher the score (≥5), the higher the incidence of pulmonary embolism.

Table 3. Wells Scoring System

Variable	Score
Clinical signs and symptoms of deep vein thrombosis	3.0
Other differential diagnoses have a low probability compared with pulmonary embolism	3.0
Pulse more than 100 times per minute	1.5
Immobilization or surgery in the last 4 weeks	1.5
Previous history of DVT or pulmonary embolism	1.5
Hemoptysis	1.0
Cancer (received treatment in the last 6 months or received palliative treatment)	1.0

Based on the Wells scoring system, the possibility of pulmonary embolism is as follows:

a. Score 0 – 1: low probability
b. If points 2 – 6: moderate possibility
c. If points are more than 6: high probability

Table 4. Geneva Scoring System

Variable	Score
Age over 65 years	1
History of pulmonary embolism or DVT	3
Surgery or fracture in the last 1 month	2
Active malignancy	2
Pain at the lower point is unilateral	3
Hemoptysis	2
Pain on palpation of deep veins on lower shivering accompanied by unilateral edema	4

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Variable	Score
Pulse 75 to 94 beats per minute	3
Pulse more than 95 times per minute	5

Based on the Geneva scoring system, the possibility of pulmonary embolism is as follows:

a. Score 0 – 3: low probability, less than 8%
b. Score 4 – 10: medium probability, approximately 28%
c. Score more than 10: high probability, approximately 74%

Electrocardiography examination

An electrocardiogram (ECG) examination is less specific if carried out on patients with mild to moderate pulmonary embolism, because it can give a normal picture. But in sufferers of severe pulmonary embolism, the following may be seen:

a. A narrow Q wave followed by an inversion of the T wave in lead III accompanied by an S wave in lead I indicates a change in heart position due to dilatation of the right atrium and ventricle. You can also find axis deviation to the right
b. P pulmonary
c. Right bundle the new branch block
d. Right ventricular strain with T wave inversion in leads V1 to V4
e. Supraventricular arrhythmia or sinus tachycardia

D-dimer and fibrinogen examination

D-dimer is a product of fibrinolysis. The higher the D-dimer level, the greater the possibility of pulmonary embolism, however relying solely on D-dimer as a marker for pulmonary embolism is very difficult. This is because during pregnancy, infection and malignancy the D-dimer value will increase. Fibrinogen levels in acute conditions will increase and in chronic conditions they do not increase. The ratio between D-dimer and fibrinogen can be used as a more specific marker of pulmonary embolism.

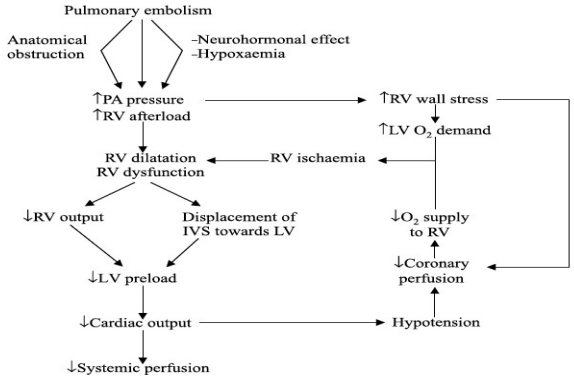

Figure 5. Relationship between pulmonary embolism and hemodynamics
Source: Kostadima

Chest X-ray Examination

Chest radiographs usually show abnormalities, although they are unclear, non-specific and do not confirm the diagnosis. Pulmonary embolism appears in the form of atelectasis or

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infiltrates. Other features may include consolidation, changes in the position of the diaphragm, decreased pulmonary vascular features, and pulmonary edema.

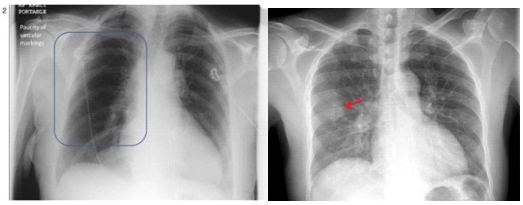

Figure 6. Picture of pulmonary embolism, a. Westermark Sign, collapse of vascularity distal to the embolism b. Hampton's Hump, a wedge-like opaque image.

Computed Tomography Pulmonary Angiogram

Computer Tomography Pulmonary Angiogram (CTPA) is a test that can diagnose pulmonary embolism. This examination has a sensitivity of 86% and a specificity of 96%. Now this examination can be used to rule out the diagnosis of pulmonary embolism in low and moderate risk patients.

Ventilation Perfusion Scanning Examination

Ventilation-Perfusion Scanning can provide useful information that can be interpreted quickly. Combined Ventilation-Perfusion Scanning and clinical assessment can provide better diagnostic accuracy. The likelihood of being positive or negative varies, but generally depends on the size, number and distribution of perfusion defects associated with the chest x-ray and ventilation abnormalities.

Pulmonary angiogram examination is the gold standard for confirming pulmonary embolism. This examination is invasive and has high risks, such as allergic reactions to contrast, pulmonary artery perforation, arrhythmia, bronchospasm, right ventricular perforation, and congestive heart failure. So the role of this examination has been replaced by spiral CT scans which have similar accuracy. Findings that can usually be found in pulmonary embolism are filling defects and abrupt cutoff of blood vessels.

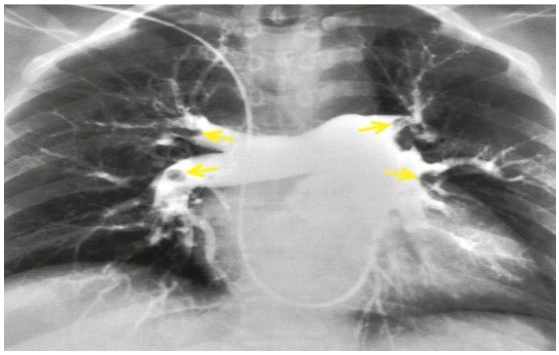

Figure 7. Angiography of pulmonary embolism. Arrows indicate multiple filling disorders due to thromboembolism.

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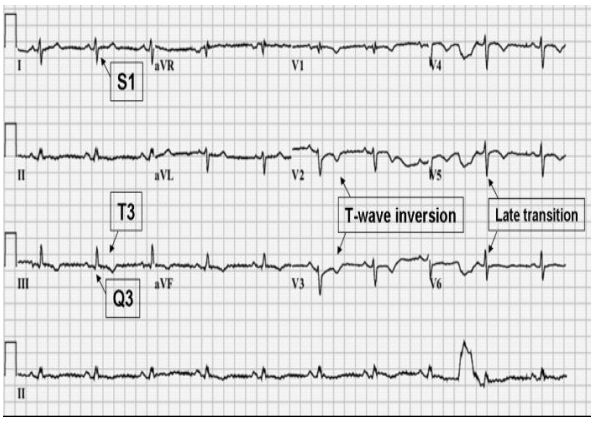

Figure 8. ECG in pulmonary embolism
Source: Todd

Echocardiography examination

Transthoracic or transesophageal echocardiography examination is of limited use for the diagnosis of pulmonary embolism. Based on echocardiography, changes in the size and function of the right ventricle and acute right heart tricuspid regurgitation indicate a strain. Appropriate clinical assessment accompanied by right ventricular changes may indicate acute pulmonary embolism. Examination for diagnosis must be adjusted to the level of clinical emergency of the patient based on the patient's condition, whether the hemodynamic state is stable or unstable.

Cardiac biomarker examination

Cardiac biomarker examination can be used to estimate the prognosis in patients with pulmonary embolism. Based on research conducted by Konstantinides, increased levels of troponin T and I biomarkers indicate a worse prognosis compared to patients who do not experience increased levels of troponin T and I. Elevated levels of these biomarkers increase the risk of mortality up to 3.5 times. Recent research states that the heart-type fatty acid binding protein (H-FABP) marker is the best marker for detecting pulmonary embolism when compared with the troponin biomarker. Several markers that can be used to diagnose acute pulmonary embolism include:

Table 5. Markers of Pulmonary Embolism

Clinical markers	Found shock and hypotension
Markers of right ventricular dysfunction	Right ventricular dilatation and hypokinesis on echocardiography. Right ventricular dilatation on spiral computed tomography Increased levels of brain natriuretic peptide or N-terminal proBNP Increased pressure in the right heart on catheterization
Markers of myocardial infarction	Troponin T or I examination shows positive

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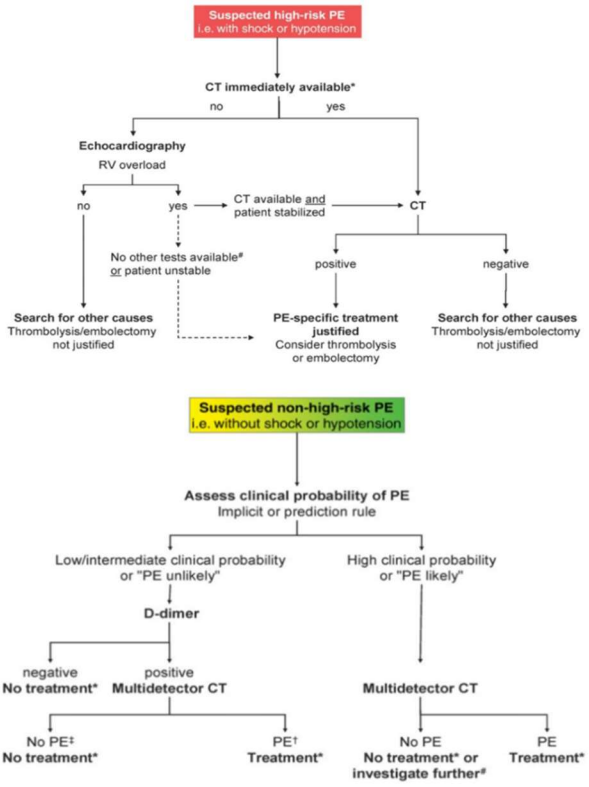
Differential Diagnosis

Some of the differential diagnoses of pulmonary embolism are pneumonia, bronchitis, bronchial asthma, acute exacerbation of chronic obstructive pulmonary disease, myocardial infarction, pulmonary edema, anxiety, aortic dissection, pericardial tamponade, lung cancer, primary pulmonary hypertension, rib fracture, pneumothorax, costochondritis, and musculoskeletal pain.

Management of Pulmonary Embolism

If a case with a probability of pulmonary embolism is found, it is necessary to differentiate cases with a high probability and a low probability because the management approach can be different. The following is an algorithm for the diagnosis and management approach in patients with a high suspicion of pulmonary embolism.



Gambar 2. Pendekatan diagnosis emboli paru

Figure 9. approach to pulmonary embolism diagnosis

Management of pulmonary embolism is respiratory and hemodynamic support, thrombolysis, embolectomy, anticoagulation. In pulmonary embolism with right heart failure there will be a decrease in systemic cardiac output, so supportive assistance is needed. If hypoxemia occurs, oxygen administration by nasal cannula is recommended. Mechanical ventilation with positive pressure should be avoided because it can reduce venous return to the heart and worsen right heart failure. Some thrombolytic agents that have been accepted as suitable regimens for pulmonary embolism are:

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a. Streptokinase: 250,000 units in 30 minutes, followed by 100,000 units/hour for 12-24 hours.
b. Urokinase: 4,400 units in 10 minutes, followed by 4,400 units/kg/hour for 12-24 hours.
c. Recombinant tissue plasminogen activator (rtPA): 100 mg in 2 hours or 0.6 mg/kg in 15 minutes. The maximum dose of rtPA administered is 50 mg.

A person's response to thrombolytic agents can be assessed via echocardiography within the first 36 hours after administration of the thrombolytic agent. There should be an improvement in the echocardiography image. Thrombolysis has the best effect if given within the first 48 hours after onset. But thrombolysis can still be given up to 6-14 days after onset. Some contraindications for administering fibrinolytic therapy are:

Table 6. Contraindications for Fibrinolytic Therapy

Absolute Contraindication	Relative Contraindication
Hemorrhagic stroke	Transient ischemic attack (TIA) within the last 6 months
Ischemic stroke within the last 6 months	Use of oral anticoagulant therapy
Damage to the central nervous system or neoplasm, Major trauma, surgery or head trauma within the last 3 weeks	Pregnancy or condition 1 week post partum
Gastrointestinal bleeding	Systolic blood pressure >180 mmHg
	Advanced stage liver disease
	Endocarditis

Apart from that, embolectomy can be performed if thrombolysis therapy cannot be carried out or fails. The percutaneous embolectomy technique using a catheter can only be used if the blocked part is a main artery, because the embolectomy technique on small arterial branches has a higher risk of perforation and damage to the blood vessel structure.

Anticoagulants also have an important role in the management of pulmonary embolism. Based on research, administering unfractionated heparin can prevent death and prevent recurrence of pulmonary embolism with bleeding complications that can still be treated. Some anticoagulants that have a rapid onset are unfractionated heparin, low-molecular-weight heparin (LMWH) heparin, or subcutaneous fondaparinux. Heparin is currently the standard initial treatment for patients with venous thromboembolism because it has the function of dissolving the thrombus and preventing recurrent embolism. Heparin dose: intravenous bolus of 5000–10,000 units followed by 1,000–1,200 units/hour. Treatment until the target PTT (partial thromboplastin time) reaches 1.5–2 times the normal value. Oral vitamin K antagonists are usually given after administration of heparin. The intravenous dose of unfractionated heparin is 80 units/kg bolus followed by a maintenance dose of 18 units/kg/hour. The aPTT should be checked every 4-6 hours after bolus injection and the dose of unfractionated heparin should be adjusted based on the aPTT results. The following is a dose adjustment of unfractionated heparin based on the results of the aPTT examination.

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Table 7. Dose Adjustment of Unfractionated Heparin Based on aPTT Value

aPTT value	Dose
<35 seconds (<1.2 times control)	80 units/kg bolus, increase dose by 4 units/kg/hour
35 – 45 seconds (1.2 – 1.5 times control)	40 units/kg bolus, increase dose by 2 units/kg/hour
46 – 70 seconds (1.5 – 2.3 times control)	No change
71 – 90 seconds (2.3 – 3.0 times control)	Reduce the infusion dose by 2 units/kg/hour
>90 seconds (>3.0 times control)	Stop infusion for 1 hour, then reduce the dose by 3 units/kg/hour

If the patient does not have a high risk of bleeding and has good kidney function, then administering subcutaneous LMWH or fondaparinux is recommended rather than administering unfractionated heparin at the following dosage:

Table 8. Dosage of LMWH and Fondaparinux

	Dose	Interval
Enoxaparin	1.0 mg/kg or 1.5mg/kg	Every 12 hours Every 24 hours
Tinzaparin	175 unit/kg	Every 24 hours
Fondaparinux	5mg(BW<50kg) 7.5 mg (BW 50 – 100 kg) 10mg (BW>100 kg)	Every 24 hours

Long-term and prophylactic management for patients suffering from pulmonary embolism is by administering vitamin K antagonists for at least 3 months. The dose of vitamin K antagonist should be adjusted to achieve a target INR of 2.0 – 3.0. If the patient has other conditions such as cancer, then LMWH prophylaxis must be extended for at least 3 – 6 months.

CONCLUSION

Pulmonary embolism is an infarction of lung tissue due to blockage of the pulmonary artery due to an embolic event. Pulmonary Embolism and Deep Vein Thrombosis have the same pathological process. Pulmonary embolism usually originates from a thrombus dislodged from the deep venous system of the lower extremities. Very important For diagnosed pulmonary emboli by clinical diagnosis with well score and geneva score, imaging examination such as electrocardiogram present sinus tachycardia, Q wave followed by an inversion of the T wave in lead III accompanied by an S wave in lead I, P pulmonary, new right bundle branch block, right ventricular strain with inversion T waves in leads V2 to V3. In Pulmonary angiogram examination there is occlusion of the pulmonary artery branch. This is can be help for management terapy and make differential diagnosis of pulmonary embolism are pneumonia, bronchitis, bronchial asthma, acute exacerbation of chronic obstructive pulmonary disease,

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