JIKW

pISSN 1978-2071 | eISSN 2580-5967
Jurnal Ilmiah Kedokteran Wijaya Kusuma 13(2):94-110, September 2024

ARTICLE INFO

AUTHOR'S AFFILIATIONS
Faculty of Medicine, Universitas Muhammadiyah Surabaya, East Java, Indonesia[1]
Department of Cardiology and Vascular Medicine, RSUD Dr. Soegiri Lamongan, Indonesia[2]

CORRESPONDING AUTHOR
Irma Kartikasari
Faculty of Medicine, Universitas Muhammadiyah Surabaya, East Java, Indonesia
E-mail:
irma.cardio@gmail.com

Article history

Received	08-07-2024
Revised	31-08-2024
Accepted	15-09-2024
Available online	30-09-2024

Please cite this article in APA 7th edition style as:
Kartikasari, I., Akil, S. N. H. (2024). Management of High-Risk Pulmonary Embolism in the Post-partum Period: A Case Report. *Jurnal Ilmiah Kedokteran Wijaya Kusuma*, 13 (1), 94-110

https://dx.doi.org/10.30742/jikw.v13i2.3246

Pulmonary Embolism in the Post-partum Period: A Case Report

*Irma Kartikasari[1,2], Syafarinah Nur Hidayah Akil[1]**

Abstract

Background: One well-known hypercoagulable state that raises the risk of venous thromboembolism is pregnancy and it is more common in cesarean delivery. Pulmonary embolisms are difficult to identify based solely on postpartum symptoms. Most of the symptoms are non-specific, therefore they are typically underdiagnosed throughout pregnancy and the postpartum period. **Objective:** This work presented information on the treatment of high-risk pulmonary embolism in the postpartum phase. **Case Presentation:** This paper reported the case of a 33-year-old female, admitted to the emergency room for shortness of breath. Her personal history showed that she had just given birth to her sixth child by caesarian section a month ago and had a history of hysterectomy due to severe infection. **Result:** She suffered a high-risk pulmonary embolism in the post-partum period. It takes a strong index of suspicion to diagnose and treat patients in a timely manner. **Conclusion:** Echocardiography and Multislice Computerized Tomography are the best diagnostic methods. Venous thromboembolism prophylaxis should be taken into account, particularly in patients who have risk factors.

Keywords: pulmonary embolism, pregnancy, post-partum period

Case Report

INTRODUCTION

Acute pulmonary embolism is one potentially dangerous clinical manifestation of venous thromboembolism (VTE). While not common in women of childbearing age, pregnant women have a five-fold increased risk of developing it compared to non-pregnant women of the same age. (Bělohlávek et al., 2013; Tan et al., 2021). The increased risk of VTE is due to hypercoagulability during pregnancy (Ho et al., 2014; Turetz et al., 2018). This condition is caused by many factors, and is believed to occur due to a combination of physical, hormonal, and hematological changes that occur during pregnancy. Some women have additional risk factors besides the pregnancy itself, placing them at higher risk of developing VTE (Heit et al., 2005; Song et al., 2022).

In terms of time, the highest risk of VTE occurs during the third trimester of pregnancy and in the 6-week period after delivery. The risk increases up to 60 folds higher at 3 months after delivery, when compared to women who are not pregnant (Alsheef et al., 2020; Samuelsson et al., 2007). It has been reported that pulmonary embolism occurs between 0.45-1 in 1000 pregnancies (Samuelsson et al., 2007). Surgery has been known to be a risk factor for VTE, and the incidence of pulmonary

Register with CC BY NC 4.0 license. Copyright © 2024, the author (s)

embolism in women delivering by cesarean section is said to be higher than normal (vaginal) delivery (Morris et al., 2010; Tadesse et al., 2020; Wu et al., 2022).

As most pulmonary embolism symptoms and indicators are ambiguous, pulmonary embolism is frequently misdiagnosed or left untreated throughout pregnancy and the postpartum period. Clinical suspicion is very important in the diagnosis of VTE. Every pregnant woman needs to be assessed whether she has risk factors for VTE from the time of antenatal care because, in every individual who successfully goes through an episode of VTE, there is an increased risk of developing cardiovascular complications later on; therefore, consideration of thromboembolic prophylaxis needs to be done in individuals who have these risk factors (Alsheef et al., 2020; Konstantinides et al., 2020). Pulmonary embolism has a varied course of the disease, depending on the severity, time and accuracy of diagnosis, and the type of treatment given. Although early detection and successful care of acute pulmonary embolism have been thoroughly reviewed in multiple guidelines, there is still controversy about the management of high-risk pulmonary embolism in pregnant women and the postpartum period.

In this case report, we describe a 33-year-old woman who underwent a caesarean section to deliver her sixth child and developed a high-risk acute pulmonary embolism during the postpartum period. Furthermore, this paper provides data on the management of high-risk pulmonary embolism during the postpartum period.

CASE PRESENTATION

Medical History

The case describes a 33-year-old housewife (Madura race), admitted to the emergency room of Dr. Soetomo Hospital Surabaya, with the main complaint of sudden shortness of breath 8 hours ago. She was referred from a private hospital with a diagnosis of cardiogenic shock and decompensation cordis. There was body weakness but no complaints of chest pain, nausea, or vomiting.

A month before the current admission, she had just given birth to her sixth child by caesarean section at a private hospital in Sidoarjo. After three days of treatment, the patient was referred to Dr. Soetomo General Hospital with a diagnosis of P6005 post caesarian section + contraceptive surgery *metode operasi wanita* (MOW) (day 3) + severe preeclampsia + hypoalbumin + gestational diabetes mellitus (DM) + suspected pulmonary edema and peripartum cardiomyopathy. She was intubated at the RES due to unstable condition. The patient was consulted to the Cardiology Department and undergone echocardiography, showing results: Ejection Fraction 62%, normal cardiac chamber dimensions, and no abnormalities of the valves. Therefore, there were no spesific therapy given.

Then, the patient was treated at intensive observation room (ROI) with a diagnosis of P6005 post caesarian section + MOW (4th day) + severe preeclampsia + DM + suspected sepsis and paralytic ileus dd obstructive + hypoalbumin. The patient was also consulted to the Digestive Surgery Department and diagnosed with suspicion of intestinal adhesions dd Ogilvie Syndrome + partial bowel obstruction + severe sepsis + septic shock + multiple organ dysfunction syndrome (MODS). After 3 days of treatment at ROI, the patient's condition did not improve, the infection was not resolved, the abdomen was still distended, then it was decided that the next day a laparotomic hysterectomy would be planned. At the time of laparotomy, there was great dilatation of the intestine, with 1000 ml of cloudy peritoneal fluid. The uterus is enlarged 24/26 weeks, there was dehiscence and there is necrotic tissue in the previous suture site, and the underlying tissue was fragile. Then, a hysterectomy and necrootomy were performed by an obstetrician, and a digestive surgeon performed decompression and washing of the abdominal cavity.

Treatment on the 8th day (4 days after the hysterectomy) the patient was declared stable and moved to the Obstetrical Room. After 1 day in the obstetric room, the patient complained of severe shortness of breath and high fever, then it was decided to be treated in the ICU and put on a ventilator. The patient was consulted to the Pulmonary Department and diagnosed with late onset Hospital Acquired Pneumonia (HAP) and given antibiotics according to culture results. After receiving treatment

in the intensive care unit (ICU) for four days, the patient was deemed stable and moved to the gynecological intensive care unit, where she stayed for thirteen days before being released to her home in good condition and without any complaints.

The length of stay at Dr. Soetomo General Hospital since admission was 23 days, and most of the time the patient was lying down. During the 4 days at home the patient admitted that she had no complaints at all, and was active as usual. Exactly on the 5th day at home, the patient suddenly felt shortness of breath at 03.00 in the morning.

The past medical history showed no history of hypertension nor DM. The patient has given birth 6 times, 5 times spontaneously vaginally, and the last child was by caesarean section (1 month ago). All children were born alive. However, one child died due to illness when the baby was 2 months old. The antenatal history showed that the patient was routinely monitored by the midwife and had a history of hypertension during pregnancy.

Clinical Findings

On admission, the physical examination showed weak general condition and somnolent. The vital sign showed blood pressure of 85/palpation, pulse 140x/minute regular, weak and deficient, dopamine installed 5 microgram/kgBW/minute, respiratory rate 32x/minute, SO2 97% (using a 10 lpm O_2 reservoir mask), and axillary temperature 36.5˚C.

There were no anomalies found during the head and neck examination. On cardiac examination, there was ictus cordis in the left intercostal space IV midclavicular line, single S_1S_2 heart sound, no extrasystoles, gallops, or murmurs were found. On lung examination, there were no crackles or wheezing sounds in both lung fields. Abdominal examination revealed no abnormalities. Examination of the extremities found the acral to be dry, cold, pale, with capillary refill time (CRT) > 2 seconds. Examination of both upper and lower limbs found no abnormalities.

Diagnostic Assessment

Laboratory examination result can be seen in **Table 1**. The electrocardiography (ECG) examination showed sinus tachycardia rhythm, 140 x/minute, a prominent S wave in lead I with inversion of Q and T waves in lead III (S1Q3T3 sign), and inverted T waves in V1-V3 (**Figure 1 Left**). Examination of the chest x-ray showed a normal heart picture and elevation of the right hemidiaphragm (**Figure 1 Right**).

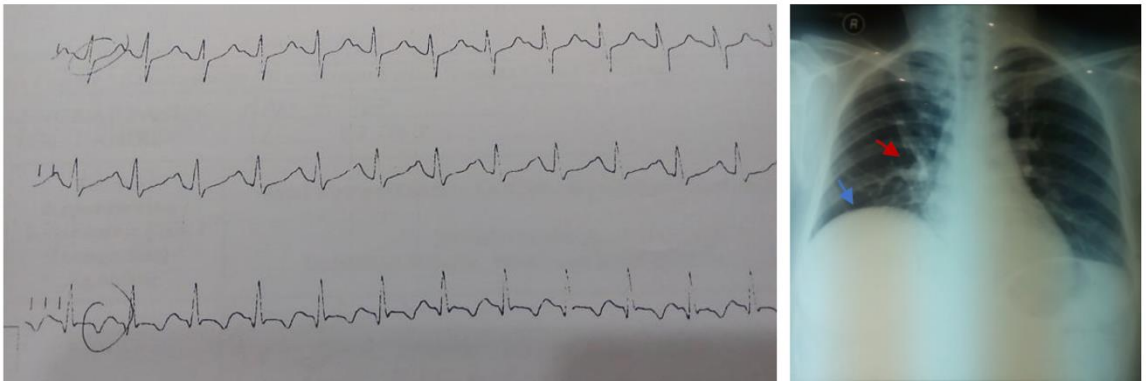

Figure 1. Left: The ECG on admission showing sign of S1Q3T3. Right: Chest x-ray with elevation of the right hemidiaphragm (blue arrow), and fibrosis in the right paracardial fibrosis (red arrow)

Table 1. The laboratory results

Laboratory findings	On admission	ICCU	Cardiology Ward		Outpatient care
	Day 1	Day 2	Day 6	Day 10	3 months
Complete blood count:					
Hemoglobin (g/dL)	11,9	9,2	11,4		
Leukocytes	10600/µL	12010	9,26		
Platelets	201000/µL	158000	282000		

Register with CC BY NC 4.0 license. Copyright © 2024, the author (s)

Hematocrit (%)	34	28,9	34,7		
Serum electrolites:					
Sodium (mmol/L)	141	138	138		
Potassium (mmol/L)	4,6	3,9			
Chloride (mmol/L)	104	107			
Renal function test:					
BUN (mg/dl)	10	7			
Serum creatinine (mg/dl)	1,5	0,69			0,78
Liver function test:					
SGOT (U/L)	30	265			17
SGPT (U/L)	14	108			21
Blood gas analysis:					
pH	7,38	7,42			
pCO2 (mmHg)	29	37			
pO2 (mmHg)	89	220			
HCO3- (mmol/l)	17,2	24			
BE (mmol/l)	-7,9	-0,5			
SO_2		100%			
Albumin (g/dl)	3,67	3,4			
INR			1,9	2,49	2,95
Random blood sugar (mg/dL)	314				143
HbA1c					6,9
Lipid profile:					
Total cholesterol					202
Triglycerides					267
HDL					39
LDL					119

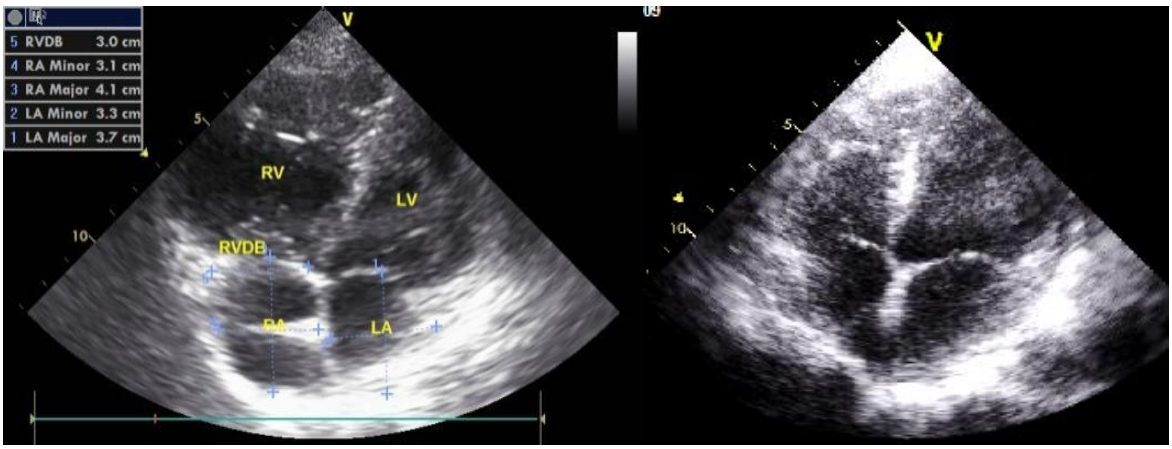

Figure 2. Left: Echocardiography in the Emergency Room (Apical 4C view) showed right ventricular dilatation is seen, and the interventricular septum is shifted to the left giving a left ventricular D-shape image; ***Right***: Echocardiography evaluation on the 8th day of treatment (Apical 4C view) showed right ventricular dilatation is reduced and left ventricular D-shape is not visible.

The results of echocardiographic examination showed that the valves showed mild tricuspid regurgitation (TR) (32 m/sec), moderate PR, the dimensions of the heart chambers showed dilated right ventricle (RV), D shape, with pulmonary hypertension (PH) (estimated pulmonary arterial systolic pressure (PASP) 47 mmHg), no thrombus or intracardiac vegetations. The left ventricular systolic function is normal with an ejection fraction (EF) of 68%. Right ventricular systolic function decreased with a tricuspid annular plane systolic excursion (TAPSE) of 1.1 cm. Segmental analysis of the right

ventricle shows hypokinetic at the base, and normokinetic at the apical. Normokinetic left ventricular segmental analysis. There is concentric left ventricular remodeling **(Figure 2 Left).**

On the 8th day of treatment, an echocardiography evaluation was carried out. The echocardiography showed an improved right ventricular function with normal right ventricular dimensions (RVDB 2,3 cm), no D shape, normal right ventricular systolic function (TAPSE 1.9cm), normal left ventricular systolic function EF by Teich 62%, Biplane 63%, left and right ventricular segmental analysis normokinetic **(Figure 2 Right).**

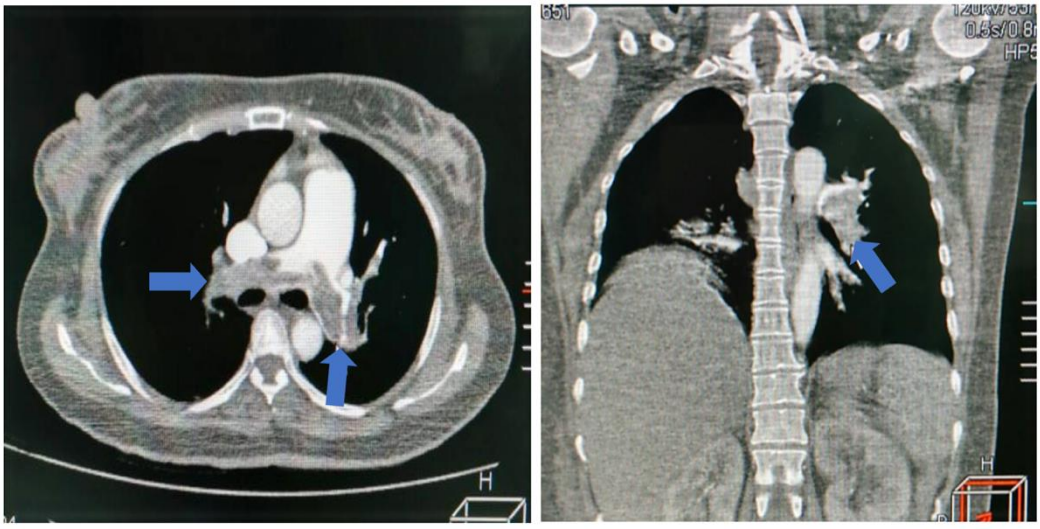

Figure 3. CT Pulmonary Angiography (CTPA) in Emergency Room. Left: Axial view, thrombus (filling defect) was seen in the right and left pulmonary arteries (blue arrow); Right: Coronal view, a thrombus (filling defect) was seen in the left pulmonary artery (blue arrow).

CT Pulmonary Angiography (CTPA) was done in the emergency room and showed a partial thrombus in the right pulmonary artery for ± 7.05cm and in the left pulmonary artery for ± 7.7cm, and fibrosis in the superior segment of the inferior lobe of the right lung **(Figure 3).**

Treatment

The patient received an infusion of 0.9% NaCl 14 drops/minute, with an O2 reservoir mask attached while in the emergency room. Then the patient was transferred to the ICCU with a diagnosis of high risk acute pulmonary embolism and hyperglycemia DM. While in the ICCU the patient was given thrombolytic therapy with Streptokinase drip 250,000 units/30 minutes, followed by Streptokinase pump 100,000 units/hour, for 12 hours, correction of hyperglycemia with insulin rapid resuscitation 3 x 4 units IV, and 0.9% NaCl infusion followed by 14 drops /minute.

A day after admission, there were decreased shortness of breath and improved hemodynamics with blood pressure 110/70, pulse 120 x/minute, respiratory rate 26x/minute, SaO2 98%, acral warm dry, red, and CRT < 2 seconds. Therapy at initial admission was continued, with the addition of Dissolf tab 3x1 per oral, Ceftriaxon injection 2 x1 gr IV, Ranitidine injection 2 x 1 ampoule IV. A basal INR examination was performed, the result was 1.54, then 8 hours after the streptokinase was finished, it was continued with 4 mg warfarin at night, in addition to controlling blood sugar levels, rapid acting insulin injections were given 3 x 6 sub-cutaneous units 5 minutes before eating, and 8 nocturnal subcutaneous unit.

On the second day of treatment at the ICCU, a laboratory examination was carried out **(Table 1),** The results showed that there were signs of infection, impaired liver function, improvement of kidney function, and normal blood gas analysis. On the third day of treatment in the ICCU due to the fever and signs of infection, the antibiotics were replaced with injections of Cefo-Sulbactam 3 x 1 gram IV, Systenol 4 x 1 tab, oxygen was replaced with 4 liters of nasal O2 per minutes, and Dissolf was

Register with CC BY NC 4.0 license. Copyright © 2024, the author (s)

terminated. On the fifth day of treatment, the patient's condition was declared stable, and the patient was transferred to the Cardiology Ward, with a diagnosis of high-risk acute pulmonary embolism (post-thrombolytic) + type II DM + secondary infection + increased liver function. On the sixth day of treatment, a laboratory evaluation was carried out (**Table 1**). On the seventh day of treatment, additional therapy was given with Enoxaparin 2x0.6cc sub-cutaneously, and other therapies including warfarin were continued.

On the 8th day of treatment, an echocardiography evaluation was carried out showing results of improved right ventricular function with normal right ventricular dimensions (RVDB 2.3cm), no D shape, normal right ventricular systolic function (TAPSE 1.9cm), normal left ventricular systolic function EF by Teich 62%, Biplane 63%, left and right ventricular segmental analysis normokinetic **(Figure 2-Right).** On the 10th day, an INR examination was carried out with a result of 2.49 (target INR reached), Enoxaparin was stopped, other therapies were continued. On Day 11, the patient's condition was stable, there were no complaints with normal vital signs. The patient was sent home with therapy: Warfarin tablets 1x 4mg at night, Ranitidine tablets 2x1 tablet, Rapid-acting insulin injections 3x8 units subcutaneously 5 minutes before eating, and long-acting insulin 1x 10 units subcutaneously at night, and the patient is planned for control to the Cardiac Polyclinic and Diabet Polyclinic.

Follow-up and Outcome

During outpatient care, the patient underwent another laboratory evaluation, with result showing high level of cholesterol and triglycerides (**Table 1).** Treatment given at the Cardiology and Diabetes Outpatient Clinic were: Warfarin alternating 1x4mg a night, and 1x3 mg a night, Simvastatin 1x20mg a night, and rapid-acting insulin injection 3x8 units subcutaneously 5 minutes before eating , and long acting insulin 1x 10 units subcutaneously at night.

Approximately 3 months after the patient had an acute pulmonary embolism, a CTPA examination was performed, and the results were good, no pulmonary embolism was found **(Figure 4).** The C and S Protein were also examined with decreasing results.

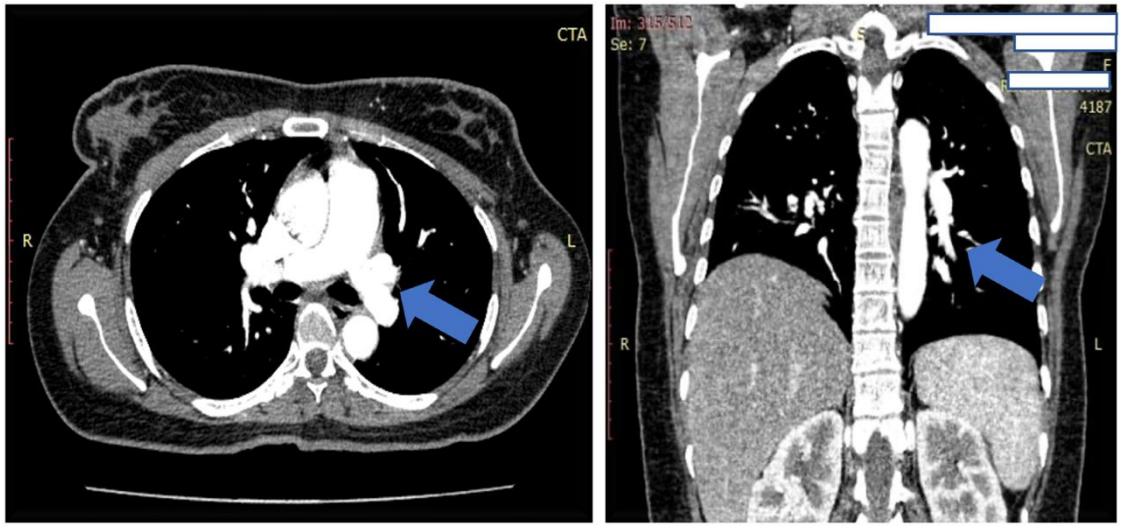

Figure 4. CT Pulmonary Angiography (CTPA) 3 months after initial anticoagulant therapy. Left: Axial view, the right and left pulmonary arteries were completely filled with contrast, no filling defects were seen. (blue arrow); Right: Coronal view, the left pulmonary artery can be seen completely filled with contrast, no filling defect is seen.

DISCUSSION

Epidemiology

When compared to non-pregnant women of the same age, pregnant women are five times more likely to have VTE. The highest risk occurs during the third trimester of pregnancy, and in the 6 week period

after delivery, then increases up to 60 times higher at 3 months after delivery, when compared to women who are not pregnant (Heit et al., 2005; Turetz et al., 2018; Wu et al., 2022). It has been reported that pulmonary embolism occurs between 0.45-1 in 1000 pregnancies (Samuelsson et al., 2007). The incidence of pulmonary embolism in women giving birth by caesarean section is said to be higher than normal delivery. VTE accounts for 1.1 deaths/100,000 births, or about 10% of maternal and child mortality. Approximately 80% of VTE that occur during pregnancy are DVT and 20% are pulmonary embolism (Simpson et al., 2001; Turetz et al., 2018; White, 2003). In this case report, the acute pulmonary embolism occurred in the patient in the post-partum period, a month after the patient delivered her 6th child by caesarean section.

Pathophysiology of VTE Related to Pregnancy

Virchow's triad—hypercoagulability, venous stasis and turbulence, and endothelial damage and dysfunction—are the syndrome that is associated with pregnancy (Tlamcani et al., 2018; Turetz et al., 2018). Hypercoagulability in pregnancy occurs due to changes in coagulation proteins. Protein S activity and antigen-free S protein decrease due to increased estrogen and possibly due to other mechanisms related to hormonal changes during pregnancy. The second and third trimesters also see an increase in resistance to protein C. Numerous coagulation factors experience a 20–2000% rise, including fibrinogen, factors II, VII, VIII, and X. Furthermore, there is an increase in the von Willebrand factor (Bělohlávek et al., 2013; Ho et al., 2014).

Thrombin activatable fibrinolytic inhibitors (TAFI), PAI-1, PAI-2, and fibrinolytic inhibitors activity increased in PE. The end result of these changes is an increased tendency for thrombus formation, extension, and thrombus stability. The normalization of the various coagulation parameters above varies depending on the influencing factors, but it is said that within 8 weeks post-partum, everything will return to normal (Chauhan & Tadi, 2023). Thrombophilia can exacerbate changes in coagulation proteins, thereby increasing the risk of VTE (Anderson & Spencer, 2003; Senst et al., 2023). Because of the dilation of the lower extremity veins during pregnancy, which is brought on by the uterus's expansion and the iliac arteries' enlargement, the stasis in the veins also increases (Tlamcani et al., 2018).

Risk factors and prevention of VTE in pregnancy and the post-partum period

Every woman during early pregnancy should have an assessment of the risk factors for TEV occurring, and this assessment can be repeated if there is a change in the patient's status during pregnancy, and will give birth (Alsheef et al., 2020; Anderson & Spencer, 2003; Morris et al., 2010; Tadesse et al., 2020). The presence of multiple risk factors that have a synergistic effect places the patient at a higher probability of developing VTE during pregnancy or the post-partum period. It is still unknown as of right now which risk factors are more directly linked to the development of pulmonary embolism during pregnancy or the postpartum phase. **Figure 5** shows VTE risk factor associated with pregnancy (Ewins & Ní Ainle, 2019; Raia-Barjat et al., 2022)**.**

Risk factor assessment can be divided into 5 categories **(Table 2).** Based on the assessment, a strategy for giving thromboprophylaxis will be determined in the antenatal, intra-partum, and post-partum periods. Thromboprophylaxis can be given non-pharmacologically, mechanically, and pharmacologically.

Because the patient in this case report had three recognized risk factors—preeclampsia, gestational diabetes, and multiparity (pregnant with her 6th child)—she was actually placed in the intermediate risk group for VTE at the time of pregnancy based on the prenatal criteria. In this group, non-pharmacological and pharmacological administration of thrombophylaxis is recommended, as well as consideration of mechanical thromboprophylaxis (Alsheef et al., 2020). After giving birth, according to postnatal criteria, moderate risk factors for VTE were obtained, due to a history of delivery by SC with emergency indications, namely because the baby could not immediately descend (arrest of descent) because of suspicion of a large baby. In addition, the patient also experienced post partum

Register with CC BY NC 4.0 license. Copyright © 2024, the author (s)

infection and sepsis which required the patient to undergo another operation, which are laparotomy and hysterectomy. A few days after surgery the patient also experienced late onset of antepartum hemorrhage, and during the course of treatment while at the hospital, the patient was on bed rest for quite a long time (aproximately 1 month). Due to the presence of the various risk factors mentioned above, they have a synergistic effect, and may place the patient at a higher risk for developing VTE in the future. However, during the antenatal and postnatal periods, the patient did not receive thromboprophylaxis pharmacologically, non-pharmacologically, or mechanically.

Figure 5. Known risk factors for VTE associated with pregnancy

Table 2. Pregnancy-associated VTE risk factor assessment criteria

Assessment criteria	Antenatal criteria	Postnatal criteria
All risk	All pregnant women have an increased risk of VTE	All women have an increased risk of VTE after delivery
Anticoagulant therapy	Anticoagulant therapy since before pregnancy	Anticoagulant therapy during the antenatal period
High risk	One or more antenatal high-risk factors	One or more postnatal high-risk factors
Moderate Risk	• 3 or more of known risk factors • If hospitalized, 2 or more risk factors	• SC for emergency indications • 3 or more of known risk factors
Low risk	0-2 known risk factors during antenatal care	1-2 known risk factors known postnatally

Establishing the diagnosis of pulmonary embolism in pregnancy

The diagnosis of VTE during pregnancy and the post-partum period is difficult and challenging. Pregnancy-associated pulmonary embolism is frequently "underdiagnosed" despite the wide range of diagnostic techniques available because most symptoms are non-specific and some can occur during a normal pregnancy. A pulmonary embolism may be suspected if there are symptoms such as tachycardia, chest pain, coughing, unexplained loss of consciousness, and/or hemoptysis. High risk pulmonary embolism is characterized by hypoxemia, hemodynamic instability, syncope episodes, and/or cyanosis (Konstantinides et al., 2020; Righini et al., 2017).

Table 3. Pregnancy-associated VTE risk factor assessment (*Source: Righini et al., 2014; Touhami et al., 2018)*

Well score			**Revised Geneva score**		
Variable	Points	**Patient**	**Variable**	Points	**Patient**
Predisposing factor			**Presdiposing factor**		
Previous DVT or PE	1.5		Age >65	1	
Recent surgery/ immobilization	1.5	**1.5**	Previous DVT or PE	1	
Cancer	1		Surgery or fracture within 1 month	1	**1**
Symptoms			Active malignancy	1	
Haemoptysis	1		**Symptoms**		
Clinical signs			Unilateral lower limb pain	1	
Heart rate > 100/min	1.5	**1.5**	Hemoptysis	1	
Clinical signs of DVT	3	**1.5**	**Clinical signs**		
Clinical judgement			Heart rate		
Alternative diagnosis less than PE	3		75-94/min	1	
			≥ 95/min	2	**2**
			Pain on lower limb deep vein at palpation and unilateral edema	1	
Clinical probability	**Total**		**Clinical probability**	**Total**	
Low	0-1		Low	0-1	
Intermediate	2-6	**4.5**	Intermediate	2-4	**3**
High	≥ 7		High	≥ 5	

Register with CC BY NC 4.0 license. Copyright © 2024, the author (s)

A combination of certain specialized diagnostic tests and a clinical likelihood pre-test should be used to diagnose pulmonary embolism. Validated probability pre-tests that are thought to be equally accurate at predicting the likelihood of pulmonary embolism are the Wells Score and the Geneva Rules. (Righini et al., 2014; Touhami et al., 2018). **Table 3** shows the clinical prediction score for PE (Well score and Revised Geneva score) and the result of the patient. The patient had a Well score of 4.5 and a Revised Geneva score of 3, therefore it was concluded that the patient might have had a pulmonary embolism (intermediate).

There are two types of pulmonary embolism: high-risk and low-risk. This stratification is critical for establishing the diagnostic and therapeutic techniques to be used based on the patient's clinical presentation at the time of initial admission to hospital. Pulmonary embolism is classified as high risk if the patient presents in a condition of shock or chronic arterial hypotension (Bělohlávek et al., 2013; Konstantinides et al., 2020). Suspicion of the presence of a high-risk pulmonary embolism is a condition that can be life-threatening quickly, while shock and hypotension themselves can occur in various diseases other than pulmonary embolism. High-risk pulmonary embolism usually has a fairly high probability pretest value. Since transthoracic echocardiography can identify if acute pulmonary hypertension and acute right ventricular dysfunction—symptoms of acute pulmonary embolism—are present or absent, it is the most effective initial diagnostic test in this case. When a very unstable patient's echocardiogram shows right ventricular dysfunction, such information is enough to determine whether to start reperfusion therapy right away. This decision can be strengthened if the echocardiography shows a thrombus in the right heart chamber (very rare). As soon as the patient is stabilized with supportive treatment and the requisite facilities are available, CT angiography should be performed to confirm the diagnosis (Bikdeli et al., 2018; Dabbouseh et al., 2019; Miranda-Bacallado et al., 2019; Oh & Park, 2023). **Figure 6** shows the diagnostic scheme of a suspected high-risk PE

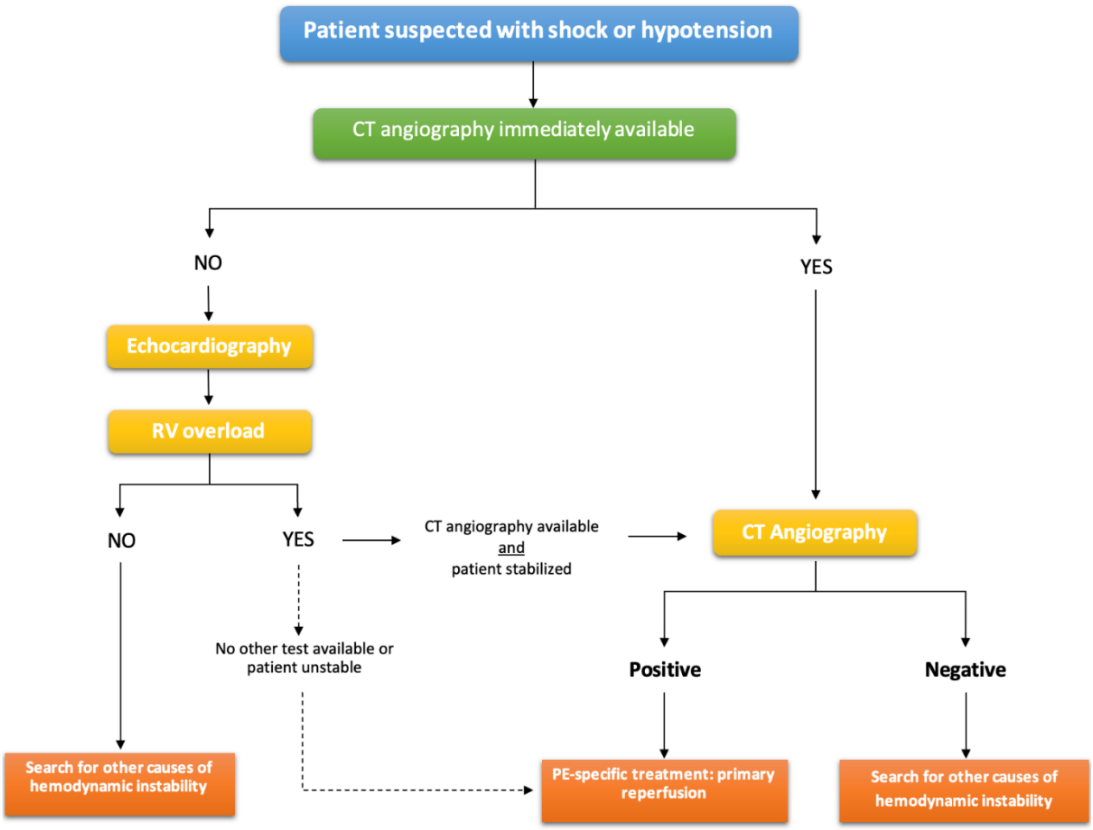

Figure 6. Diagnostic scheme of a patient suspected of having a high-risk pulmonary embolism
***Source**: Konstantinides et al., 2020*

The patient came for the first time in an unstable hemodynamic state, with a blood pressure of 85/palpation, then was stabilized by giving an O_2 reservoir mask, and infusion of 0.9% NaCl fluid 14 drops/minute. After the condition allows, a CT scan of the chest with contrast was done immediately. Prior to the CT scan, an echocardiographic examination was performed on the patient and found signs of right ventricular overload, with normal left ventricular function. On CT scan of the chest with contrast, thrombus was found in the right and left pulmonary arteries, therefore the diagnosis of high-risk pulmonary embolism was established, and then specific therapy for pulmonary embolism was carried out, namely primary reperfusion through thrombolytics using Streptokinase.

Investigations in acute pulmonary embolism

Electrocardiogram

The ECG in the form of S1Q3T3 signs (prominent S wave in lead I, Q wave and inversion of T wave in lead III) is a sign of acute cor pulmonale (acute increase in right ventricular pressure and volume due to pulmonary hypertension) and can also be found strain on the right ventricle. This ECG abnormality occurs in 15% to 25% of PE patients. Additional ECG findings observed during the initial phase of the illness include right bundle branch block, right-shifted QRS axis, ST segment elevation in V1 and aVR, low QRS amplitude in all leads, premature atrial contractions, sinus tachycardia, atrial fibrillation/flutter, and T-wave inversion in leads V1–V4. PE is frequently linked with ECG abnormalities, however these abnormalities are neither sensitive nor specific for detecting the condition (Bikdeli et al., 2018; Dabbouseh et al., 2019). The result of ECG in this patient showed a prominent S wave in lead I, with inverted Q and T waves in lead III (S1Q3T3 sign), and there are inverted T waves in V1-V3.

Laboratory examination

Laboratory tests such as cardiac enzymes, blood gas analysis, brain natriuretic peptide, are usually only used to evaluate possible alternative diagnoses other than pulmonary embolism. Laboratory tests are of little use in terms of establishing the diagnosis of TEV in pregnancy. For example, the D-Dimer examination is not recommended for examination in pregnancy conditions, because it is known that D-Dimer levels in normal pregnant women increase according to gestational age, and after delivery, besides that D-Dimer levels also increase in twin pregnancies, after undergoing caesarean section, in post-partum hemorrhage, and in preeclamptic conditions. Blood gas analysis is also non-specific, because in 58% of cases of pulmonary embolism in pregnancy, 58% of cases have normal blood gas analysis results (Righini et al., 2014). In this case, the patient's blood gas analysis results showed low pO_2 and pCO_2, even with oxygen mask reservoirs of 10 liters/minute. These features of hypoxaemia and hypocapnia suggest the presence of a PE, although this is not always the case.

Thorax x-ray

Normal chest radiographs are found in 18-20% of patients with acute pulmonary embolism. The most common abnormalities included cardiomegaly, lung parenchymal infiltrates, pulmonary congestion, pleural effusion, and atelectasis. In addition, rarer findings include elevation of the hemidiaphragm, pulmonary artery enlargement, and focal oligemia (Al Dandan et al., 2020; Zubairi et al., 2007). In this patient, elevation of the right hemidiaphragm was found during the acute phase **(Figure 1)**. There was still an elevation of the right hemidiaphragm at the evaluation three months following therapy, raising the possibility of right paracardial base fibrosis.

Echocardiography

Right ventricular dilatation, right ventricular dysfunction, and occasionally normal apical contractility (McConnell's sign) are echocardiographic characteristics associated with acute pulmonary embolism. The right ventricle is hypokinetic in more than 90% of individuals with massive pulmonary embolism, as indicated by indirect indicators such as tricuspid regurgitation and right ventricular dilatation/dysfunction, which can be identified on both TTE and TEE and have a pulmonary embolism

Register with CC BY NC 4.0 license. Copyright © 2024, the author (s)

specificity of roughly 90% and sensitivity of about 50%. Signs of pulmonary hypertension can also be seen from the displacement and flattening of the interventricular septum which gives a D shape on a short axis section during both systole and diastole, tricuspid regurgitation with a velocity of more than 2.7 m/s. The presence of right ventricular dilatation and dysfunction is a poor prognostic sign and an indicator for thrombolytic therapy in pulmonary embolism (Bělohlávek et al., 2013; Bikdeli et al., 2018; Dabbouseh et al., 2019; Oh & Park, 2023).

In this case report, the results of echocardiography showed dilatation and dysfunction of the right ventricle, with the presence of McConnell's sign, and a flattened interventricular septum (D shape). In addition, mild tricuspid regurgitation and moderate pulmonary regurgitation were also found, with normal left ventricular function **(Figure 2)**.

Computed tomographic pulmonary angiography (CTPA)

With the invention of multi-detector computed tomographic (MDCT) angiography, which offers superior arterial opacification and great spatial and temporal resolution, CTPA has emerged as the go-to technique for diagnosing patients who may have had a pulmonary embolism. The pulmonary arteries can be adequately seen with CTPA, at least at the segmental level. The Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) II assay for MDCT, which predominantly used four detectors, demonstrated 83% sensitivity and 96% specificity. Furthermore, PIOPED II showed how clinical likelihood influences MDCT predictive value (Dalen, 2006; Stein et al., 2006; Worsley & Alavi, 1995).

Pulmonary angiography

Pulmonary angiography has been the "gold standard" for diagnosing or ruling out pulmonary embolism for the past few decades, but it is no longer performed frequently because CTPA, which is now extensively used, offers nearly the same diagnostic accuracy using a less intrusive technique. Pulmonary angiography is used more frequently in diagnostic delivery. When treating an acute pulmonary embolism, percutaneous catheter-directed guided therapy is more frequently guided by pulmonary angiography. Digital subtraction angiography (DSA) yields better image quality for the peripheral pulmonary arteries in patients who are able to hold their breath while using less contrast media than conventional cineangiography. However, movement artifacts make it difficult to see the main pulmonary artery branches (Konstantinides et al., 2020).

Management of high-risk pulmonary embolism

Management of ana cute pulmonary embolism consisted of three things, the use of anticoagulant, reperfusion of the pulmonary artery, and cardiopulmonary support (Martinez Licha et al., 2020). Patients with high-risk acute pulmonary embolism present with a clinical presentation of shock or hypotension, and have a high mortality rate while in hospital, especially within the first few hours in hospital. Hemodynamic and respiratory supportive therapy should be administered pending definitive therapy. Aggressive fluid therapy is not recommended, but giving fluid challange (500 mL) is said to help increase the cardiac index. The use of vasopressors is often necessary, but keep in mind that if the cardiac index increases excessively above the physiological threshold, it can exacerbate the ventilation–perfusion mismatch. Hypoxaemia and hypocapnia are common in patients with pulmonary embolism, and most can be treated with oxygen, but in some severe cases, mechanical ventilation is sometimes required. Administration of UFH should be given as an initial anticoagulation option, whereas LMWH or fondaparinux is not recommended for pulmonary embolism accompanied by hypotension and shock. The definitive treatment for pulmonary embolism is primary reperfusion, and systemic thrombolytics are the treatment of choice for primary reperfusion in patients with acute high-risk pulmonary embolism. Thrombolytic therapy in acute pulmonary embolism can restore pulmonary perfusion faster than administration of unfractionated heparin (UFH), and quickly resolve obstruction in the lungs so as to reduce pressure and resistance in the pulmonary arteries, and result in improved

right ventricular function. Recombinant tissue plasminogen activator (rtPA), urokinase, and streptokinase are among the options for thrombolytic drugs. Streptokinase can be administered accelerated over 2 hours (1.5 million IU), and is preferable to prolonged infusions over 12-24 hours (250,000 IU loading dose in 30 minutes, followed by 100,000 IU/hour). Thrombolytics are said to be still effective up to 14 days after the onset of pulmonary embolism (Alsheef et al., 2020; Konstantinides et al., 2020).

Whenever possible, surgical embolectomy is advised for patients who are contraindicated for thrombolytics or who have had systemic thrombolytic therapy but do not see an improvement in their hemodynamic condition. Percutaneous catheter-directed therapy is an alternative that should be considered if the required resources and therapies are available. Patients who have a documented recurrent pulmonary embolism despite receiving appropriate anticoagulation or who have an acute pulmonary embolism and are completely contraindicated to anticoagulation should have a venous filter inserted (Konstantinides et al., 2020).

Anticoagulants should be administered either in the acute phase or laterAnticoagulation is advised during the acute phase of pulmonary embolism in order to avoid both early death and repeated symptoms as well as a deadly pulmonary embolism. Anticoagulant therapy for pulmonary embolisms must be administered for a minimum of three months. In the acute phase period, parenteral heparin such as UFH, LMWH or fondaparinux can be given for 5-10 days. Parenteral heparin must be overlapped with a vitamin K antagonist (VKA) at the initiation of VKA administration, an alternative to VKA is new oral anticoagulants (NOAC), but what has been proven safe in breastfeeding mothers is VKA compared to NOAC. Before deciding to discontinue anticoagulation, the risk of further thrombosis must be evaluated (Alsheef et al., 2020; Chen et al., 2020; Martinez Licha et al., 2020).

CHEST's latest antithrombotic administration guidelines (2016) suggest that patients with DVT of the proximal lower extremities and a pulmonary embolism following surgery should be given for at least 3 months or more. However, the duration to be given can be adjusted according to the condition of each patient and the clinician's considerations. Grants can be given over a period shorter than 3 months, a longer period but limited in time (e.g. 6, 12, or 24 months), or an extended period indefinitely (can be for life). Extending the duration of anticoagulant therapy for more than 3 months must be accompanied by an evaluation of the benefits and risks of giving therapy, which is carried out periodically (Barnes, 2016; Kearon et al., 2016).

Evaluation for the possibility of thrombophilia should be performed after discontinuation of anticoagulant therapy, only if the results of such tests may influence further management. Protein S and C deficiencies can be caused by secondary conditions such as cancer, vitamin K deficiency, or liver illness, or main conditions such as genetic causes. Low levels of protein C and S have been linked to severe infections, inflammatory diseases, kidney illness, cancer, some chemotherapy treatments, HIV, pregnancy, the moments following a thrombotic episode, and the administration of VKA. If the results of protein C and S are decreased in a patient, it must be repeated at another time before a definite diagnosis is made. If a deficiency is suspected due to acquired/secondary factors, it is advisable to repeat the examination when the underlying conditions or conditions that can reduce protein C and S have been resolved. Once the diagnosis of primary protein C and S deficiency (inherited deficiency) is established, clinicians should be aware that these patients are prone to recurrent TEV when exposed to conditions that can precipitate TEV such as surgery, chemotherapy, and oral contraceptives (Alsheef et al., 2020; Martinez Licha et al., 2020; Tadesse et al., 2020).

In this case report, the management of the patient while at the ER was IV fluids NaCL 0.9% 14 drops/ minute, Dopamine 5 micro/kgBB/minute, and oxygen mask reservoir 10 liters/minute. After being transferred to the ICCU, systemic thrombolytics with streptokinase were given intravenously. The therapy was continued with the administration of oral VKA anticoagulants, enoxaparin, while the patient was in the room together with the VKA. When the target INR was reached, the patient was sent home with VKA as the anticoagulant of choice. After giving anticoagulants for 3 months, CTPA was re-evaluated with good results where no pulmonary embolism was seen. The patients were also

Register with CC BY NC 4.0 license. Copyright © 2024, the author (s)

examined for protein C and S and obtained decreased results. However, this decrease in protein C and S levels could occur secondary to the fact that the examination was carried out while the patient was still undergoing therapy with VKA, with an INR that was on target. After considering the benefits and risks of therapy, it was decided to extend anticoagulant therapy for 6 months and will be re-evaluated later to consider whether anticoagulant administration needs to be given longer.

CONCLUSION

A pulmonary embolism can be fatal, and the postpartum phase carries the biggest risk. The patient in this case repot is an example with history of multiparity and previous surgical procedures, which put her at a higher risk of developing PE. The importance of early and definite diagnosis will lead to a better management of the disease. The clinical signs were confirmed with chest MSCT and echocardiography. The management given were systemic thrombolytics in a prolonged infusion and continued with administration of VKA anticoagulants. The patient's clinical improvement during outpatient care, the patient is motivated to always move actively (mobilize) according to ability. The patient is currently able to carry out daily activities, although he has not been able to do heavy work.

REFERENCES

Al Dandan, O., Hassan, A., AbuAlola, H., Alzaki, A., Alwaheed, A., Alalwan, M., Al Shammari, M., AlShamlan, N., & Alsaif, H. S. (2020). Clinical and imaging profiles of pulmonary embolism: A single-institution experience. *International Journal of Emergency Medicine*, *13*(1), 47. https://doi.org/10.1186/s12245-020-00303-y

Alsheef, M. A., Alabbad, A. M., Albassam, R. A., Alarfaj, R. M., Zaidi, A. R. Z., Al-Arfaj, O., & Abu-Shaheen, A. (2020). Pregnancy and Venous Thromboembolism: Risk Factors, Trends, Management, and Mortality. *BioMed Research International*, *2020*, 4071892. https://doi.org/10.1155/2020/4071892

Anderson, F. A., & Spencer, F. A. (2003). Risk Factors for Venous Thromboembolism. *Circulation*, *107*(23_suppl_1), I–9. https://doi.org/10.1161/01.CIR.0000078469.07362.E6

Barnes, G. D. (2016). *CHEST Guideline for Antithrombotic Therapy in VTE - American College of Cardiology*. American College of Cardiology. https://www.acc.org/latest-in-cardiology/ten-points-to-remember/2016/03/02/15/45/antithrombotic-therapy-for-vte-disease

Bělohlávek, J., Dytrych, V., & Linhart, A. (2013). Pulmonary embolism, part I: Epidemiology, risk factors and risk stratification, pathophysiology, clinical presentation, diagnosis and nonthrombotic pulmonary embolism. *Experimental & Clinical Cardiology*, *18*(2), 129–138.

Bikdeli, B., Lobo, J. L., Jiménez, D., Green, P., Fernández-Capitán, C., Bura-Riviere, A., Otero, R., DiTullio, M. R., Galindo, S., Ellis, M., Parikh, S. A., & Monreal, M. (2018). Early Use of Echocardiography in Patients With Acute Pulmonary Embolism: Findings From the RIETE Registry. *Journal of the American Heart Association: Cardiovascular and Cerebrovascular Disease*, *7*(17), e009042. https://doi.org/10.1161/JAHA.118.009042

Chauhan, G., & Tadi, P. (2023). Physiology, Postpartum Changes. In *StatPearls*. StatPearls Publishing. http://www.ncbi.nlm.nih.gov/books/NBK555904/

Chen, A., Stecker, E., & A. Warden, B. (2020). Direct Oral Anticoagulant Use: A Practical Guide to Common Clinical Challenges. *Journal of the American Heart Association*, *9*(13), e017559. https://doi.org/10.1161/JAHA.120.017559

Dabbouseh, N. M., Patel, J. J., & Bergl, P. A. (2019). Role of echocardiography in managing acute pulmonary embolism. *Heart (British Cardiac Society)*, *105*(23), 1785–1792. https://doi.org/10.1136/heartjnl-2019-314776

Dalen, J. E. (2006). New PIOPED Recommendations for the Diagnosis of Pulmonary Embolism. *The American Journal of Medicine*, *119*(12), 1001–1002. https://doi.org/10.1016/j.amjmed.2006.06.034

Ewins, K., & Ní Ainle, F. (2019). VTE risk assessment in pregnancy. *Research and Practice in Thrombosis and Haemostasis*, *4*(2), 183–192. https://doi.org/10.1002/rth2.12290

Heit, J. A., Kobbervig, C. E., James, A. H., Petterson, T. M., Bailey, K. R., & Melton, L. J. (2005). Trends in the Incidence of Venous Thromboembolism during Pregnancy or Postpartum: A 30-Year Population-Based Study. *Annals of Internal Medicine*, *143*(10), 697–706. https://doi.org/10.7326/0003-4819-143-10-200511150-00006

Ho, Y.-K., Wang, C.-P., Wu, Y.-L., Lee, T.-H., Ying, T.-H., & Lee, M.-S. (2014). Pulmonary embolism after cesarean section and successful treatment with early application of extracorporeal membrane oxygenation system and anticoagulant agents. *Taiwanese Journal of Obstetrics and Gynecology*, *53*(2), 273–275. https://doi.org/10.1016/j.tjog.2013.04.041

Kearon, C., Akl, E. A., Ornelas, J., Blaivas, A., Jimenez, D., Bounameaux, H., Huisman, M., Cs, K., Ta, M., N, S., Sm, S., Jre, V., P, W., Sc, W., & L, M. (2016). Antithrombotic Therapy for VTE Disease: CHEST Guideline and Expert Panel Report. *Chest*, *149*(2). https://doi.org/10.1016/j.chest.2015.11.026

Konstantinides, S. V., Meyer, G., Becattini, C., Bueno, H., Geersing, G.-J., Harjola, V.-P., Huisman, M. V., Humbert, M., Jennings, C. S., Jiménez, D., Kucher, N., Lang, I. M., Lankeit, M., Lorusso, R., Mazzolai, L., Meneveau, N., Ní Áinle, F., Prandoni, P., Pruszczyk, P., … ESC Scientific Document Group. (2020). 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS): The Task Force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC). *European Heart Journal*, *41*(4), 543–603. https://doi.org/10.1093/eurheartj/ehz405

Martinez Licha, C. R., McCurdy, C. M., Maldonado, S. M., & Lee, L. S. (2020). Current Management of Acute Pulmonary Embolism. *Annals of Thoracic and Cardiovascular Surgery*, *26*(2), 65–71. https://doi.org/10.5761/atcs.ra.19-00158

Miranda-Bacallado, J., Izquierdo-Gómez, M. M., García-Niebla, J., Jiménez, J. J., Iribarren, J. L., Laynez-Cerdeña, I., & Lacalzada-Almeida, J. (2019). Role of echocardiography in a patient with suspected acute pulmonary embolism: A case report. *Journal of Medical Case Reports*, *13*(1), 37. https://doi.org/10.1186/s13256-019-1994-y

Morris, J. M., Algert, C. S., & Roberts, C. L. (2010). Incidence and risk factors for pulmonary embolism in the postpartum period. *Journal of Thrombosis and Haemostasis*, *8*(5), 998–1003. https://doi.org/10.1111/j.1538-7836.2010.03794.x

Oh, J. K., & Park, J.-H. (2023). Role of echocardiography in acute pulmonary embolism. *The Korean Journal of Internal Medicine*. https://doi.org/10.3904/kjim.2022.273

Raia-Barjat, T., Edebiri, O., & Chauleur, C. (2022). Venous Thromboembolism Risk Score and Pregnancy. *Frontiers in Cardiovascular Medicine*, *9*, 863612. https://doi.org/10.3389/fcvm.2022.863612

Righini, M., Es, J., Exter, P., Roy, P.-M., Verschuren, F., Ghuysen, A., Rutschmann, O., Sanchez, O., Jaffrelot, M., Trinh-Duc, A., Le Gall, C., Moustafa, F., Principe, A., Houten, A., Wolde, M.,

Register with CC BY NC 4.0 license. Copyright © 2024, the author (s)

Douma, R., Hazelaar, G., Erkens, P., Kralingen, K., & Le Gal, G. (2014). Age-Adjusted D-Dimer Cutoff Levels to Rule Out Pulmonary Embolism. *JAMA : The Journal of the American Medical Association*, *311*, 1117–1124. https://doi.org/10.1001/jama.2014.2135

Righini, M., Robert-Ebadi, H., & Le Gal, G. (2017). Diagnosis of acute pulmonary embolism. *Journal of Thrombosis and Haemostasis*, *15*(7), 1251–1261. https://doi.org/10.1111/jth.13694

Samuelsson, E., Hellgren, M., & Högberg, U. (2007). Pregnancy-related deaths due to pulmonary embolism in Sweden. *Acta Obstetricia et Gynecologica Scandinavica*, *86*(4), 435–443. https://doi.org/10.1080/00016340701207500

Senst, B., Tadi, P., Basit, H., & Jan, A. (2023). Hypercoagulability. In *StatPearls*. StatPearls Publishing. http://www.ncbi.nlm.nih.gov/books/NBK538251/

Simpson, E. L., Lawrenson, R. A., Nightingale, A. L., & Farmer, R. D. T. (2001). Venous thromboembolism in pregnancy and the puerperium: Incidence and additional risk factors from a London perinatal database. *British Journal of Obstetrics and Gynaecology*, *108*(1), 56–60. https://doi.org/10.1016/S0306-5456(00)00004-8

Song, B., Sun, Y., Liu, D., & Li, G. (2022). *Acute pulmonary embolism right after caesarean section despite left ventricle dilatation: A case report and literature review* [Preprint]. In Review. https://doi.org/10.21203/rs.3.rs-1515928/v1

Stein, P. D., Fowler, S. E., Goodman, L. R., Gottschalk, A., Hales, C. A., Hull, R. D., Leeper, K. V., Popovich, J., Quinn, D. A., Sos, T. A., Sostman, H. D., Tapson, V. F., Wakefield, T. W., Weg, J. G., & Woodard, P. K. (2006). Multidetector Computed Tomography for Acute Pulmonary Embolism. *New England Journal of Medicine*, *354*(22), 2317–2327. https://doi.org/10.1056/NEJMoa052367

Tadesse, T. A., Kedir, H. M., Fentie, A. M., & Abiye, A. A. (2020). Venous Thromboembolism Risk and Thromboprophylaxis Assessment in Surgical Patients Based on Caprini Risk Assessment Model. *Risk Management and Healthcare Policy*, *13*, 2545–2552. https://doi.org/10.2147/RMHP.S272852

Tan, T. C., Goh, C. M. Y., Tan, S. S. X., Tan, L. K., Yang, Y., & Lee, L. H. (2021). Epidemiology of pregnancy-associated pulmonary embolism in South Asian multi-ethnic country: Mortality trends over the last four decades. *Journal of Obstetrics and Gynaecology Research*, *47*(1), 174–183. https://doi.org/10.1111/jog.14450

Tlamcani, I., Mouh, N. E., Amrani, K. E., & Hassani, M. A. (2018). Pregnancy and Hemostasis: From Physiology to Pathological States. *Clinical Research in Hematology*, *1*(1).

Touhami, O., Marzouk, S. B., Bennasr, L., Touaibia, M., Souli, I., Felfel, M. A., Kehila, M., Channoufi, M. B., & Magherbi, H. E. (2018). Are the Wells Score and the Revised Geneva Score valuable for the diagnosis of pulmonary embolism in pregnancy? *European Journal of Obstetrics & Gynecology and Reproductive Biology*, *221*, 166–171. https://doi.org/10.1016/j.ejogrb.2017.12.049

Turetz, M., Sideris, A. T., Friedman, O. A., Triphathi, N., & Horowitz, J. M. (2018). Epidemiology, Pathophysiology, and Natural History of Pulmonary Embolism. *Seminars in Interventional Radiology*, *35*(2), 92–98. https://doi.org/10.1055/s-0038-1642036

White, R. H. (2003). The Epidemiology of Venous Thromboembolism. *Circulation*, *107*(23_suppl_1), I–4. https://doi.org/10.1161/01.CIR.0000078468.11849.66

Worsley, D. F., & Alavi, A. (1995). Comprehensive Analysis of the Results of the PIOPED Study. *The Journal of Nuclear Medicine*, *36*(12).

Wu, Y.-Y., Shan, T.-T., & Pan, X.-T. (2022). Pulmonary Embolism After in vitro Fertilization and Cesarean Section: Two Case Reports and Brief Review of the Literature. *International Journal of Women's Health*, *14*, 1489–1497. https://doi.org/10.2147/IJWH.S366355

Zubairi, A. B. S., Husain, S. J., Irfan, M., Fatima, K., Zubairi, M. A., & Islam, M. (2007). Chest radiographs in acute pulmonary embolism. *Journal of Ayub Medical College, Abbottabad: JAMC*, *19*(1), 29–31.

Register with CC BY NC 4.0 license. Copyright © 2024, the author (s)