Acute pulmonary embolism right after caesarean section despite left ventricle dilatation: A case report and literature review

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Case Report

Keywords: acute pulmonary embolism, postpartum, dilated left ventricular, catheter-directed angiography, patent foramen ovale□case report

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Abstract

Background

Pulmonary embolism is a lethal complication during pregnancy and puerperium. Compared to vaginal delivery, caesarean section has almost twice the pulmonary risk. The diagnosing of pulmonary embolism can be challenging because of the overlapping symptoms and signs in maternity, and also the clinician should balance the benefits and risks of radioactive examinations.

Case presentation:

A 31-year-old nulliparous female underwent caesarean section for a separated symphysis pubis. Right after the operation, the patient developed abrupt dyspnea and dull pain whitin the left back region, and her cardiopulmonary function deteriorated rapidly. Venous ultrasound confirmed left common iliac vein occlusion, and transthoracic echocardiogram revealed a relatively normal right heart and dilated left ventricle with patent foramen ovale. Pulmonary angiography confirmed the diagnosing of acute embolism. Then, catheter-directed thrombus fragmentation and thrombolysis were performed. The patient recovered quickly, and the echocardiogram results were normal a month later.

Conclusions

Dilated left ventricular echocardiography cannot rule out postpartum acute pulmonary embolism. In some extreme emergency scenarios, when acute PE is the most likely diagnosis (a life-threatening condition), catheter-directed angiography can be performed instead of other radioscopies.

Background

Pulmonary embolism (PE) is a fatal cardiovascular complication contributing to maternal mortality in developed countries[1]. Hypercoagulability caused by physiological changes in pregnancy can protect women from haemorrhage but can also lead to maternal death from thromboembolic disease[2]. Compared to traditional vaginal delivery, caesarean section has almost double the risk for pulmonary embolism[3]. The diagnosis of PE can be challenging because the patients can be asymptomatic or can have signs and symptoms in pregnancy that overlap with those of other complications (cardiomyopathy, amniotic fluid embolism). Currently, the preliminary diagnosis of acute PE in postpartum women is established by bedside ultrasonography and the evaluation of the pulmonary artery and right heart size and function[4]. In suspected PE during pregnancy and up to 6 weeks postpartum, computed tomography pulmonary angiography (CTPA) and V/Q(ventilation/perfusion) scans suggested to additional confirm the PE diagnosis[5]. However, left heart dilation is rarely reported and may lead to misdiagnosis. In some emergency situations, fast diagnosis of PE can save time and prevent maternal death. Herein, we present a case of severe acute PE caused by iliac vein thrombosis right after caesarean delivery with left ventricle

dilation, which was quickly diagnosed by catheter-directed pulmonary angiography, and local thrombolysis was successfully conducted.

Case Presentation

In July 2021, a 31-year-old nulliparous female at 39 weeks 4 days was brought to the obstetrical unit of this hospital for occasional contractions for one day without vaginal bleeding. The patient was pregnant for the first time and without abnormalities throughout the gestation. The results of all antenatal screening tests were normal. On examination, she looked well. Her vital signs were ordinary. Her abdomen was gravid, soft, and not tender. Blood tests, chest X-ray and cardiogram were normal. Ultrasonographic examination with a foetal survey revealed that the placenta was positioned posteriorly and that the measurements of the foetus coincided with gestational age. Suspecting relative macrosomia and separation of the symphysis pubis, we scheduled the patient for caesarean section.

She underwent spinal anaesthesia at L3-4 with 2 ml of 0.5% lidocaine and 15 μg of fentanyl. The surgery was successful, and a healthy infant (3880 g) was delivered. Oxytocin was given after the operation to promote uterine contractions. Approximately 5 min after removal of the placenta, she abruptly developed dyspnoea and dull pain in the left back region. Her heart rate increased to 120/min, her blood pressure fell to 100/60 mmHg, and her peripheral oxygen saturation was 85%. Mask ventilation with oxygen was immediately started. Her peripheral oxygen saturation returned to 95% after the procedure, but her heart rate and blood pressure still did not change. She was transferred to the intensive care unit for unstable cardiopulmonary conditions.

The patient was safely transferred to the ICU. She was conscious and complained about continuous, dull left back pain and shortness of breath. Physical examination results were as follows: body temperature: 37°C; heart rate: 125/min; respiratory rate: 28/min; blood pressure: 90/60mmHg; and no obvious abnormality on pulmonary auscultation, but the heart sounds were markedly enhanced, the abdomen was soft, and also the incision showed no obvious bleeding. Her legs showed symmetrical, slight swelling without varicose veins. Laboratory examinations: Blood gas analysis revealed a pH of 7.39, PCO2 of 33 mmHg, PO2 of 71 mmHg，Lac of 1.9 mmol/L, HCO3 of 20 mmol/L and a surplus pulse O2(SpO2) maintained at 92-95% with oxygen delivered by mask at 6 L/min. The white blood cell count was 12.00×10^9/L, haemoglobin was 117 g/L and platelets were 137×10^9/L. The coagulation function test showed that the D-dimer level was 4.359 mg/L and the fibrinogen degradation product level was 27.6 μg/ml. Other coagulation parameters were within normal ranges. Cardiac troponin I was 3.02 ng/ml, and NT-BNP was 963 pg/ml. Chest X-ray showed that the texture of the bilateral lungs was increased and blurred, with multiple patchy, striped shadows and enlarged cardiac shadows (Figure 1). Electrocardiogram revealed nodal tachycardia, slightly elevated ST segments in leads V1-V3 and ST segment depression in lead V4 (Figure 2). Transthoracic echocardiogram (TTE) (Figure 3) showed a dilated, hypocontractile left ventricle (LV), Ejection Fractions (EF) 45%, patent foramen ovale with left-to-right shunt with a beam width of 3 mm, and normal right ventricle (RV)，RV to LV ratio(RV:LV) was 0.47，RV end-diastolic diameter 25mm, LV end-diastolic diameter 53mm. Venous ultrasound

of the bilateral lower extremities showed no thrombosis, but iliac vein ultrasound revealed that the left common iliac vein blood stream was slow and predicated blood clots (Figure 4). The patient's condition continuing to deteriorate, her SpO2 decreased to 85%, her heart rate increased to 130 bpm, and her blood pressure fell, which required pharmacological support with dobutamine. A mask with an oxygen bag at 10 L/min was used to maintain SpO2 at 90-92%.

The reason for the hypotension and hypoxemia was unclear. When women are pregnant or have recently given birth, they sometimes present the syndrome of chest pain and dyspnea combined with sudden deterioration of cardiopulmonary and circulatory failure. The diagnoses that should be considered are pulmonary embolism, myocardial infarction, aortic dissection and peripartum cardiomyopathy. The elevated troponin level and atypical ECG results (slightly elevated ST segment) made the diagnosis of myocardial infarction less likely, while her normal pulmonary auscultation made heart failure cause by myocardial infarction looked impossible. Aortic dissection was excluded based on her healthy history, and the characteristics of pain in her chest were different from the classic symptom of aortic dissection, which is always described as severe, ripping, tearing, stabbing or sharp. In addition, echocardiography did not reveal enlarged aortic diameters[6]. Amniotic fluid embolism was excluded by cleaning the abdominal incision, and ongoing coagulopathy was not seen. Peripartum and Takotsubo cardiomyopathy is rare but still needs to be excluded by further investigation. Although uncommon, TTE results of a dilated left ventricle made the diagnosis of pulmonary embolism seem less likely, given the symptom of abrupt dyspnoea and dull pain in her chest combined with the evidence of elevated D-dimer and blood clots located in left common iliac vein. In the end, the diagnosis of acute pulmonary embolism was the primary diagnosis.

Clinical diagnosis

Postpartum acute pulmonary embolism from iliac vein thrombosis. Patent foramen ovale.

Treatment

Pulmonary embolism was presumed to be the primary diagnosis given the abrupt hypoxemia and circulatory collapse without any signs of bleeding or other perinatal complications. Low-molecular-weight heparin calcium 4000 IU was subcutaneously administered for initial treatment. As PE is an immediately life-threatening situation, further examination, including CTA and V/Q scans, should be performed to confirm the diagnosis. However, cardiopulmonary fragility impedes the removal of patients from the ICU for radiological imaging and could increase the risks of cardio-pulmonary function collapse and even cardiac arrest. After consulting with cardiologists and interventional radiologists, we conducted inferior vena cava angiography to confirm the PE diagnosis and facilitate subsequent therapy. The procedure revealed bilateral pulmonary thrombosis, which indicated the diagnosis of pulmonary embolism and excluded other diagnoses. Then, catheter-directed thrombus fragmentation and thrombolysis were performed, and angiography confirmed the reopening of a bilateral pulmonary thrombosis (Figure 4). An inferior vena cava (IVC) filter was implanted for iliac vein thrombosis, which was confirmed by ultrasonic testing. The patient continued to receive anticoagulant therapy: subcutaneous injections of low-

molecular-weight heparin calcium 8000 IU/d followed by oral rivaroxaban tablets 10 mg/d. A month later, the cardiac ultrasound was normal. The inferior vena cava (IVC) filter was removed three months later and continued to do well at follow-up.

Discussion And Conclusion

Venous thromboembolism (VTE), which includes of deep vein thrombosis (DVT) and pulmonary embolism (PE), is one of the most common cardiovascular diseases and is a leading cause of maternal death[7]. VTE accounted for 3% of maternal deaths in developing countries, while the proportion is estimated to be 14% in developed countries[8]. In a recent multicenter analysis of pregnancy-related hospitalizations, the rate of pregnancy-related acute PE was 19.36 per 100,000 hospitalizations, which rate was low but had not decreased over the past decade. Although advanced therapies are used, the rate of in-hospital mortality from acute PE during pregnancy and puerperium has not improved[9]. Pregnancy is a hypercoagulable state that protects women from delivery-related bleeding, but the pathophysiological changes include increased clotting factors and decreased fibrinolysis[10]. Additionally, venous flow velocity in the lower limbs is reduced because of physiological vasodilation, compression of the vena cava by the gravid uterus, and compression of the left iliac vein by the right iliac artery[11]. Alterations in haemostasis and vein blood flow with pregnancy all increase the risk of thrombosis in gestation and the perinatal period. Caesarean section (CS) is associated with higher maternal and perinatal mortality and morbidity than vaginal delivery[12]. The incidence of maternal VTE after caesarean delivery is approximately four times greater than that after traditional vaginal delivery. The risk of CS-associated VTE seems independent of other risk factors[13]. Besides haemostatic modifications induced by pregnancy, the CS procedure itself may lead to greater activation of coagulation[14].

The differential diagnosis of VTE in pregnant and postpartum women must be made carefully. The symptoms and signs of VTE often overlap with the physiological changes of pregnancy, including tachycardia, dyspnea and lower extremity oedema, which might lead to misdiagnosis. The majority of published prediction models for the diagnosis of pulmonary embolism have not been validated in pregnant women[15]. D-dimer is not helpful for diagnosing PE in pregnant/postpartum women, as D-dimer levels increase throughout pregnancy[16]. Duplex ultrasonography (DUS) is the standard protocol for the diagnosis of DVT in symptomatic pregnant woman. It is widely available and carries a low risk for both mother and fetus. The Use of bilateral lower extremity venous compression ultrasonography (CUS) before chest imaging has been advocated. However, the sites of DVT in pregnant women are common in left lower extremity veins but also exist in iliac veins owing to compression of the left iliac vein by the gravid uterus, that is sort of completely different from the DVT within the general population, which is usually located within the calf[17]. The deeper, intrapelvic-located iliofemoral veins always hinder the gravid uterus, making it difficult to acquire clear images during the compression manoeuvre of venous duplex ultrasonography. CUS of the symptomatic leg along the length of the femoral vein to the level of the calf vein combined with Doppler imaging of the iliac veins was confirmed to reliably exclude clinically important DVT[18]. The diagnosis of symptomatic DVT by DUS not only establishes the diagnosis of DVT but also circumvents further radiological examinations if PE is clinically suspected. Additional

diagnostic methods to assess PE via chest imaging CTPA and/or lung perfusion scintigraphy (V/Q scan) are required[5].

Acute PE always leads to RV pressure overload and dysfunction, which can be detected by echocardiography, but there are no standard echocardiographic parameters that provide reliable information on RV size or function. Therefore, a negative RV result cannot exclude PE[19]. Furthermore, TTE shows no significant abnormalities of PE in a large proportion of patients with confirmed acute PE[20]. Studies of acute PE detected by TTE always focus on image of right heart size and function[21]. LV dilated and hypokinesis in echocardiography often help in the differential diagnosis of severe global or regional LV dysfunction[22]. Dilated LV is rare in pulmonary embolism and can mislead the clinician to diagnose cardiomyopathies. The haemodynamic changes of pregnancy include blood volume increases, cardiac output increases and oxygen consumption[23]. The detrimental effects of acute PE on the myocardium may impair LV blood flow output, which causes LV pressure overload. Cardiovascular changes in pregnancy include haemodynamic, neurohumoral, renin/angiotensin, RBC changes and cardiac structural changes[23]. Left ventricular wall thickness and left ventricular mass increase throughout pregnancy [24]. Plasma volume increases may result in a relatively deteriorated cardiac workload that has been damaged by acute PE. We extrapolated the LV dilation from decreased myocardial contractility and systemic afterload overload. Although the results of echocardiography were not consistent with the usual results of acute PE, we accepted the diagnosis of acute PE because DUS showed left iliac vein thrombus and clinical symptoms of acute PE. In this case, TTE also found patent foramen ovale(PFO) with a left-to-right shunt. PFO is a highly prevalent condition in the adult population[25, 26]. Although most individuals with PFO are asymptomatic, a PFO can serve as a pathway for a transient right-to-left gradient during early ventricular systole and with the Valsalva manoeuver, which depends on the pressure in the right atrium exceeding that in the left atrium[26]. The left-to-right shunting in this case indicated that the pressure in the left atrium exceeded that in the right, which is rarely reported and indirectly reflects an overloaded afterload and left heart dilation.

Many cardiac emergencies, including myocardial infarction, peripartum cardiomyopathy, acute myocarditis, dilated cardiomyopathy and Takotsubo cardiomyopathy, ought to be considered, as all of them are life-threatening illnesses that could afflict patients with symptoms of left heart failure, such as onset dyspnoea and hypotension, which mimic acute PE. Most peripartum cardiomyopathies are diagnosed by individual history, cardiac biomarkers, cardiography, and echocardiography[27]. Practitioners should endeavour to better understand these rare conditions to provide timely high-quality service to pregnant women who suffer life-threatening situations.

The backbone of treatment for acute PE in pregnancy is anticoagulation, which includes the primary selection of low-molecular-weight heparin (LMWH) and also the second alternative of unfractionated heparin (UFH)[5]. High-risk hemodynamically unstable PE patients should receive advanced therapies, including systemic and catheter-based thrombolysis, as well as surgical embolectomy. In addition, extracorporeal membrane oxygenation can be a rescue treatment for life-threatening situations[28].

Inferior vena cava (IVC) filers during pregnancy can prevent additional venous clots causing pulmonary embolism, with few complications[29].

In conclusion, acute PE is a life-threatening scenario in the perinatal period. TTE is a standard imaging protocol for confirming the diagnosis of PE and evaluating heart function. However, negative results, even left ventricular dilation, do not fully rule out the PE diagnosis, as physiological changes in pregnant women may further influence cardiac structure and function. In some emergency situations, patients have haemodynamic instability and a risk of cardiac arrest. Catheter-directed angiography, as the gold standard, can be performed at first, when the patient's symptoms and other results point to PE as the suspected diagnosis.

Abbreviations

PE
Pulmonary Embolism
VTE
Venous Thromboembolism
CS
Caesarean Section
CTPA
Computed Tomography Pulmonary Angiography
V/Q
Ventilation/Perfusion
TTE
Transthoracic Echocardiogram
LV
Left Ventricle
RV
Right Ventricle
SpO2
Surplus Pulse O2
ECG
Electrocardiogram
IVC
Inferior Vena Cava
DUS
Duplex Ultrasonography
CUS
Compression Ultrasonography
PFO
Patent Foramen Ovale

LMWH
Low Molecular Weight Heparin
UFH
Unfractionated Heparin

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the declaration of Helsinki.

Consent to publish

Written informed consent for publication was obtained from the patient.

Availability of data and materials

The data and materials are available on request from the corresponding author.

Competing interests

The authors declare that they have no competing interests.

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Author's contributions

BS managed the patient, analyzed the case report and was a major contributor in writing the manuscript. YS managed the patient and contributed to writing the manuscript. DDL analyzed the case report and contributed in writing the manuscript. GGL analyzed the case report and contributed reviewing the manuscript. All authors read and approved the final manuscript.

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Figures

Figure 1

Chest X-ray showed that the texture of the bilateral lungs was increased and blurred, with multiple patchy, striped shadows and an enlarged cardiac shadow

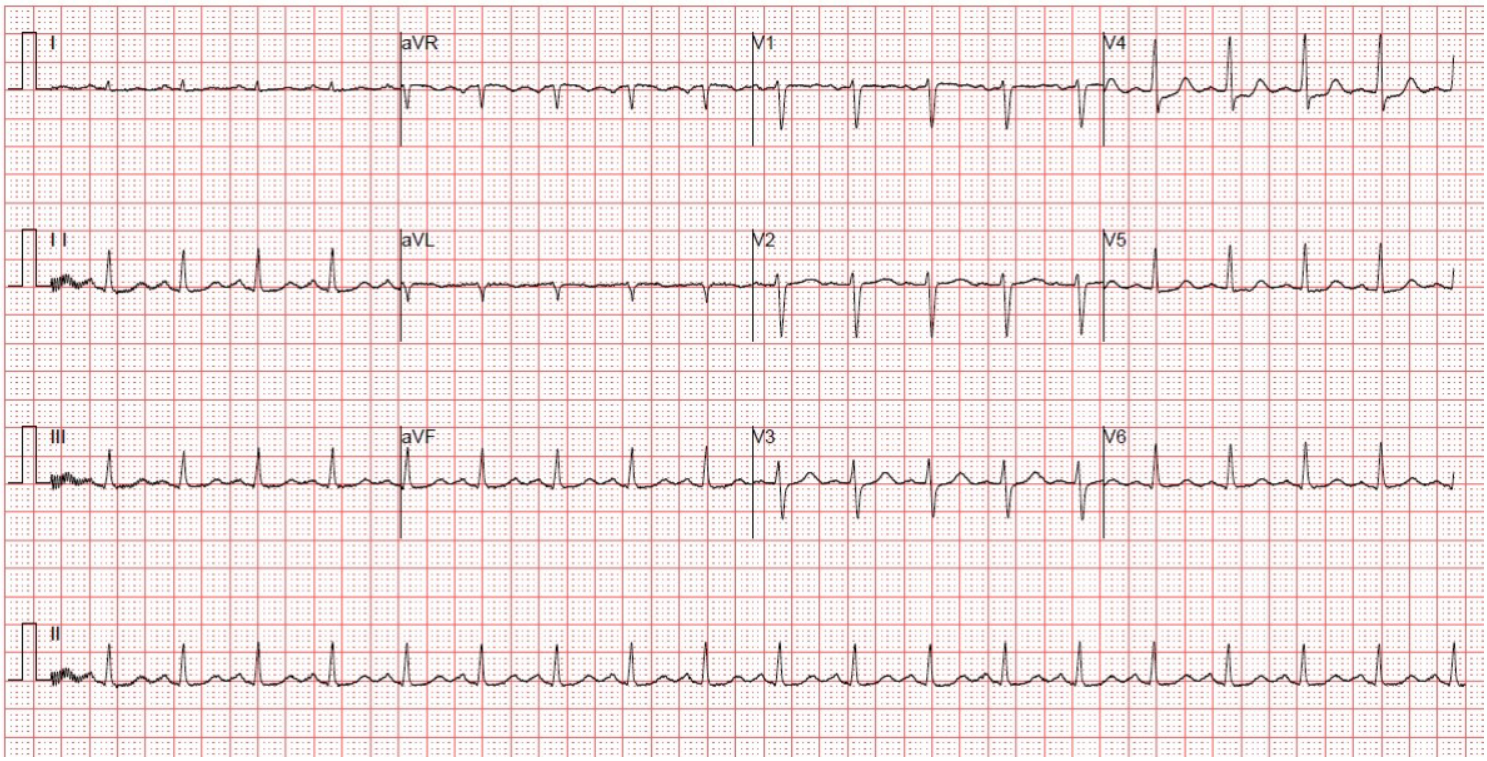

Figure 2

Electrocardiogram revealed nodal tachycardia, slightly elevated ST segments in leads V1-V3 and ST segment depression in lead V4

Figure 3

A the left common iliac vein blood stream was slow and predicated blood clots

B Dilated left ventricle (LV), patent foramen ovale with a left-to -right shunt which a beam width of 3mm, the right ventricle (RV) was normal.

C Hypocontractile left ventricle (LV), EF 45%

D Normal TTE results a month later

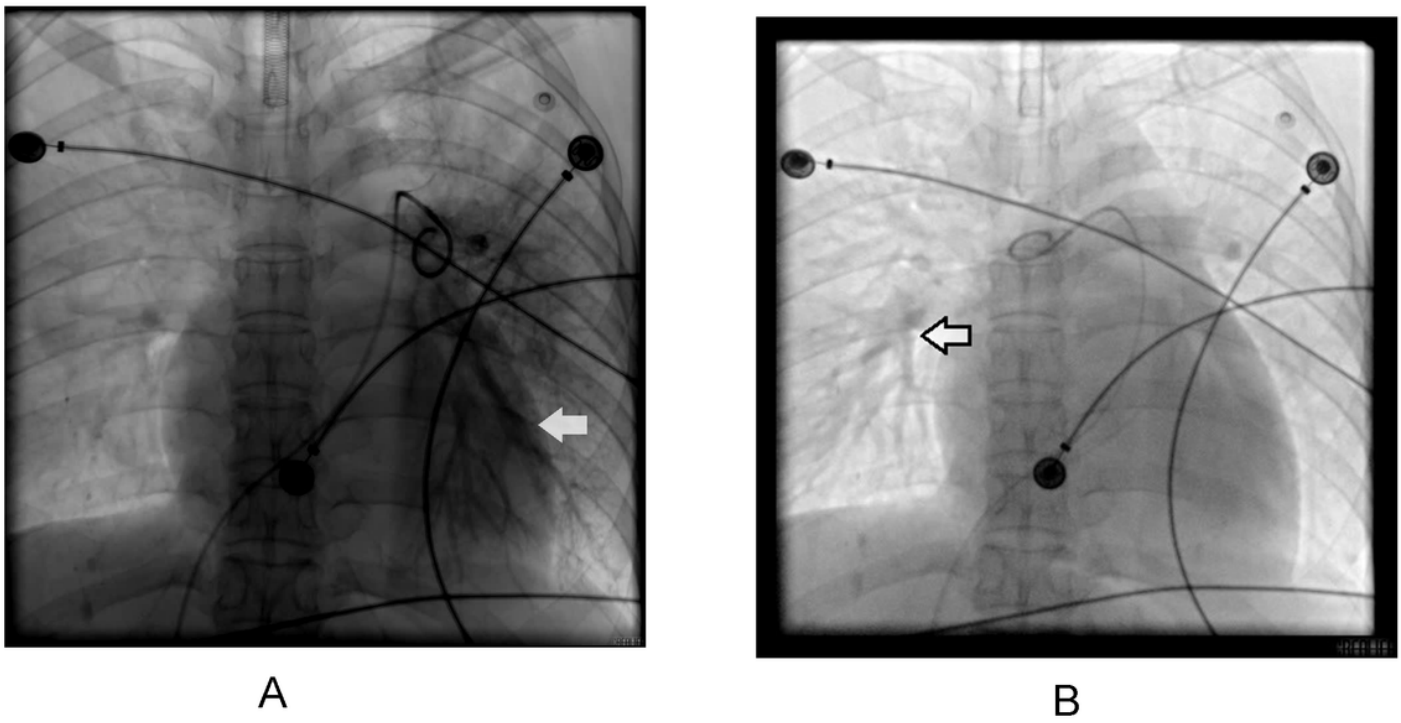

Figure 4

A Arrow shows the Inferior lobe of the left pulmonary artery. The upper and middle lobe of the left pulmonary artery were not developed.

B Arrow shows the partially inferior lobe of the right pulmonary artery. The upper and middle lobes of the right pulmonary artery were not developed.