RESEARCH PAPER

Pulmonary Embolism in a Young Woman: A Case Report Highlighting Oral Contraceptive Use as a Risk Factor

Dr. Jay Shankar[1], Dr. Saifa M. Latheef[2], Dr. Shubham Gupta[3*], Dr. Pankaj Sood[4]
[1-4]Department of Emergency Medicine, School of Medical Sciences and Research, Sharda Hospital, Greater Noida, Uttar Pradesh
[1]Consultant
[2]Associate Professor and Clinical HOD
[3,4]PGY-3
***Corresponding Author:** Dr. Shubham Gupta (PGY-3), Department of Emergency Medicine, School of Medical Sciences and Research, Sharda Hospital, Greater Noida, Uttar Pradesh
Email: shubhamgupta.dr@gmail.com
Received: 25th June 2026; **Revised:** 28th July 2026; **Accepted:** 10th October 2026; **Available Online:** 15th October 2026

ABSTRACT

Background:
Pulmonary embolism (PE) in young, otherwise healthy patients is an uncommon diagnosis that can be easily missed due to its non-specific clinical presentation.

Case Presentation:

A 20-year-old woman presented to the emergency department with acute-onset chest pain, dyspnoea, palpitations, dizziness, and diaphoresis of 8 hours duration. Her only identifiable risk factor was the recent initiation of a combined oral contraceptive pill (OCP, Femilon) three months prior. On examination, she was tachycardic (145/min) and tachypnoeic (24/min) but maintained a normal blood pressure (102/76 mmHg) and room air oxygen saturation (99%). An electrocardiogram revealed sinus tachycardia with an S1Q3T3 pattern and incomplete right bundle branch block. Point-of-care echocardiography demonstrated marked right heart dilatation, reduced tricuspid annular plane systolic excursion (TAPSE), and McConnell's sign. Troponin I was elevated. CT pulmonary angiography confirmed an extensive bilateral pulmonary thrombus extending into segmental and interlobar arteries. She was successfully managed with systemic thrombolysis (streptokinase 1.5 million IU) and low-molecular-weight heparin, later transitioning to oral apixaban, and was discharged after a 3-day hospital stay.

Conclusion:
Clinicians must maintain a high index of suspicion for PE in young women taking oral contraceptives who present with unexplained chest pain or tachycardia. Early integration of bedside echocardiography and ECG is crucial for timely diagnosis and management.

Keywords: pulmonary embolism; oral contraceptive pills; emergency medicine; young female; case report

How to cite this article: *Jay Shankar1 Dr. Saifa M. Latheef2 Dr. Shubham Gupta3* Dr. Pankaj Sood4. Pulmonary Embolism in a Young Woman: A Case Report Highlighting Oral Contraceptive Use as a Risk Factor. Int J Drug Deliv Technol. 2026;16(89s):974-980. DOI: 10.25258/ijddt.16.89s.102.*

Source of support: ***Nil.***

Conflict of interest: *None*

Introduction

Pulmonary embolism (PE) is a life-threatening manifestation of venous thromboembolism [1]. Diagnosing PE in young adults is challenging because symptoms like chest pain, dyspnoea, and palpitations frequently mimic benign conditions,

leading to critical delays in treatment. While combined oral contraceptives are a well-documented risk factor due to their oestrogenic effect on the coagulation cascade [2,3], PE with significant right ventricular involvement in this demographic remains uncommon. Here, we report a case of extensive bilateral pulmonary embolism with severe right ventricular strain in a young woman where recent OCP use was the sole identifiable provoking factor. This report follows the CARE reporting guidelines [12].

Case presentation

A 20-year-old woman arrived at the emergency department reporting an 8-hour history of acute chest pain, shortness of breath, palpitations, dizziness, and profuse sweating. She denied having fever, cough, haemoptysis, or lower-limb swelling. Her medical history was unremarkable, with no recent travel, surgery, or prolonged immobilisation. She did not smoke and had no family history of venous thromboembolism or known thrombophilia. Her only medication was a combined OCP (desogestrel 0.15 mg and ethinylestradiol 0.02 mg) started three months earlier. The timeline of events is summarised in Table 1.

Examination

On arrival, her airway was secure. Vital signs included a temperature of 37.0 °C, respiratory rate of 24/min, heart rate of 145/min, and blood pressure of 102/76 mmHg. Oxygen saturation was 99% on room air. Systemic examination was largely unremarkable; chest auscultation revealed bilateral equal air entry, and heart sounds were normal. She was fully conscious (Glasgow Coma Scale 15). Her extremities were warm with normal distal pulses and no pedal oedema.

Investigations

An initial ECG showed sinus tachycardia (136/min) alongside an S1Q3T3 pattern and incomplete right bundle branch block. Immediate point-of-care echocardiography (POCUS) revealed marked right atrial and ventricular dilatation, acute tricuspid regurgitation, a reduced TAPSE (1.1 cm), raised pulmonary artery pressure, and McConnell's sign. A formal 2D echocardiogram performed 48 hours later noted normal left ventricular function, mild right-sided dilatation, and no pericardial effusion.

Laboratory workup (detailed in Table 2) showed an elevated troponin I (3.14 ng/mL). Interestingly, her D-dimer was 0.22 µg/mL (reference < 0.50 µg/mL FEU). Her blood count showed a moderate normocytic anaemia (Hb 8.9 g/dL, MCV 97.7 fL) but was otherwise unremarkable (TLC 6.53 $\times 10^3/\mu L$, platelets 158,000/µL). Renal, hepatic, and viral profiles were unremarkable.

CT pulmonary angiography (CTPA) demonstrated substantial filling defects at the main pulmonary artery bifurcation. The thrombus partially occluded the right pulmonary artery and completely occluded the left, extending into all segmental and interlobar branches bilaterally—confirming an extensive pulmonary embolism (Figure 1).

Diagnosis

Acute bilateral pulmonary embolism with right ventricular strain. According to ESC criteria, this fell into the intermediate-high risk category (AHA/ACC Category C3: elevated clinical severity score with both RV dysfunction and a raised troponin), likely provoked by combined OCP use.

Treatment and outcome

Given the extensive thrombus burden and echocardiographic evidence of severe RV dysfunction, the patient received systemic thrombolysis with a 2-hour infusion of streptokinase (1.5 million IU) in the ICU. Subcutaneous enoxaparin (1 mg/kg twice daily) was administered concurrently. She tolerated the procedure well with no bleeding or allergic complications. After a 2-day ICU stay and 1 day on the ward, she was discharged on oral apixaban (10 mg twice daily for a week, followed by 5 mg twice daily). Routine outpatient follow-up was arranged for the following week.

Discussion

VTE is uncommon in young, healthy women. For this patient, a three-month history of OCP use was the primary clinical clue. Oestrogen significantly alters the coagulation cascade, and the thrombotic risk peaks during the first year of therapy [2,3]. While definitive proof of causation is difficult to establish in the emergency department without an exhaustive outpatient thrombophilia panel, the temporal link makes the OCP the most likely culprit.

Clinically, the diagnosis hinged on recognising the combination of unexplained tachycardia and acute chest symptoms. The S1Q3T3 pattern on ECG, while not highly sensitive, suggested acute right heart strain in this context [4,5]. This was corroborated by bedside POCUS showing classic signs of RV dysfunction (McConnell's sign and low TAPSE) [6,7]. The combination of RV strain and elevated troponin marked this as an intermediate-high risk PE, despite her stable blood pressure and oxygen saturation [8,9].

Relying solely on a normal blood pressure and oxygen saturation in such scenarios can provide false reassurance.

Notably, her D-dimer was within normal limits. False-negative D-dimers in extensive PE, though rare, can occur due to delayed presentation, impaired endogenous fibrinolysis, or specific assay limitations. This case reinforces the rule that diagnostic imaging should not be delayed by a negative D-dimer when pre-test clinical probability is high [10]. Additionally, the formal echocardiogram at 48 hours showed marked improvement compared to the initial POCUS, underscoring the rapid haemodynamic benefit of early thrombolysis. Her moderate normocytic anaemia (Hb 8.9 g/dL) was not attributed to acute bleeding, but it warranted cautious monitoring during anticoagulation and further outpatient evaluation.

Management in light of current guidelines

The decision to thrombolyse a normotensive patient requires careful clinical judgment. Under the 2026 AHA/ACC guideline, this patient fits category C3: an elevated clinical severity score (sPESI ≥1) with both RV dysfunction and a raised troponin [11]. Reperfusion therapy, including systemic thrombolysis, is considered reasonable for patients with persistent hypotension (Category E, class 2a), whereas its benefit in normotensive patients in Category C3 is unclear (class 2b) and it may be considered in incipient cardiopulmonary failure (Category D). Table 3 summarises the initial management by 2026 AHA/ACC clinical category.

In this case, the sheer volume of the bilateral thrombus and the degree of right heart failure prompted the use of an accelerated streptokinase regimen [8]. Thrombolysis in Category C3 is therefore an individualised decision, and the guideline recommends measuring lactate and involving a PE response team for Categories C–E [11]. Although streptokinase is an older agent with known bleeding risks, it remains a practical and effective option in resource-constrained settings. Low-molecular-weight heparin, the preferred parenteral anticoagulant in the 2026 guideline [11], was given together with thrombolysis without bleeding complications, and recovery was rapid and stable.

Learning points

- PE should be in the differential diagnosis of any young woman taking oral contraceptives who has acute chest pain, dyspnoea or unexplained tachycardia.
- A normal oxygen saturation and preserved blood pressure do not rule out significant PE, and a normal D-dimer should not be used to exclude PE when clinical probability is high.
- ECG changes of right heart strain and bedside echocardiography can raise early suspicion, while CT pulmonary angiography confirms the diagnosis.
- Under the 2026 AHA/ACC guideline, the benefit of thrombolysis in normotensive patients with RV dysfunction and a raised troponin (Category C3) is unclear; the decision should be individualised, with lactate measurement and PE response team input.
- Oestrogen-containing contraceptives should be discontinued after PE, and alternative non-oestrogen contraception discussed.

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Tables

Table 1. Timeline of the case.

Time	Event
Previous 3 months	Started combined oral contraceptive pill (Femilon)
Day 0, 8 hours before arrival	Onset of chest pain, shortness of breath, palpitations, dizziness and sweating
Day 0, emergency department	Heart rate 145/min, respiratory rate 24/min, BP 102/76 mmHg, SpO2 99%; ECG and point-of-care echocardiography
Day 0, after arrival	CT pulmonary angiography confirmed extensive bilateral pulmonary embolism
Day 0	Streptokinase infusion (1.5 million IU) with continuous monitoring in the ICU
ICU stay	2 days
Ward stay	1 day
Discharge	Stable condition

Table 2. Laboratory results on presentation.

Test	Result
Troponin I	3.14 ng/mL
D-dimer	0.22 µg/mL
Haemoglobin	8.9 g/dL
Total leucocyte count	6.53 ×10³/µL
Neutrophils / lymphocytes	78.2% / 17.1%
Eosinophils / monocytes / basophils	1.0% / 3.3% / 0.4%
Red blood cell count	2.77 ×10⁶/µL
Haematocrit	27.1%
MCV / MCH	97.7 fL / 32.1 pg
Platelets	158 ×10³/µL

MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin.

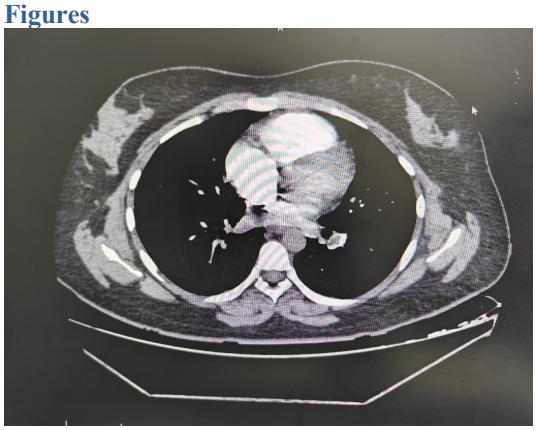
Table 3. Initial management by 2026 AHA/ACC clinical category [11].

Category	Description	Initial management
A	Asymptomatic, incidentally found PE	Individualised; evidence on the benefit of anticoagulation is still developing
B	Symptomatic, low clinical severity (PESI ≤85, sPESI 0, Bova ≤4)	Many patients can be treated at home with oral anticoagulation; a direct oral anticoagulant is preferred to a vitamin K antagonist
C	Symptomatic, elevated severity score (PESI >85, sPESI ≥1, Bova >4), with or without right ventricular dysfunction or raised biomarkers	Hospitalise; anticoagulation (low-molecular-weight heparin preferred if parenteral); PE response team review; systemic thrombolysis not recommended in preference to anticoagulation alone in C1–C2 (harm in C2); benefit unclear in C3 (class 2b)
D	Incipient cardiopulmonary failure: transient hypotension (D1) or normotensive shock (D2)	PE response team; close monitoring; advanced therapy (systemic or catheter-directed thrombolysis, thrombectomy) may be considered, benefit unclear (class 2b)
E	Persistent hypotension and/or cardiopulmonary failure (formerly massive or high-risk PE)	Urgent reperfusion: systemic thrombolysis, catheter-directed treatment or mechanical thrombectomy (class 2a), with anticoagulation and supportive care; surgical embolectomy

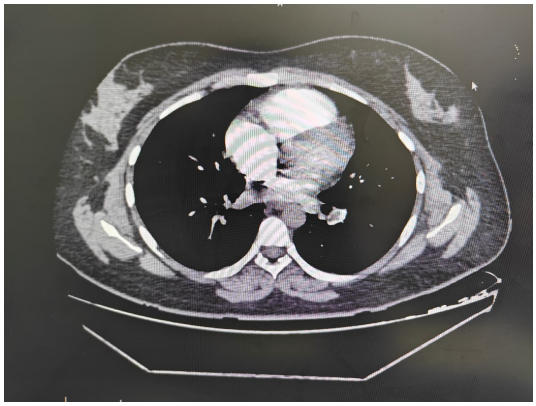
		and mechanical circulatory support in refractory cases

PESI, Pulmonary Embolism Severity Index; sPESI, simplified PESI.

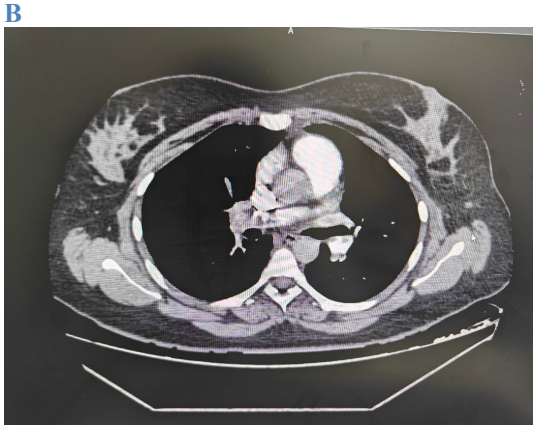
Figures



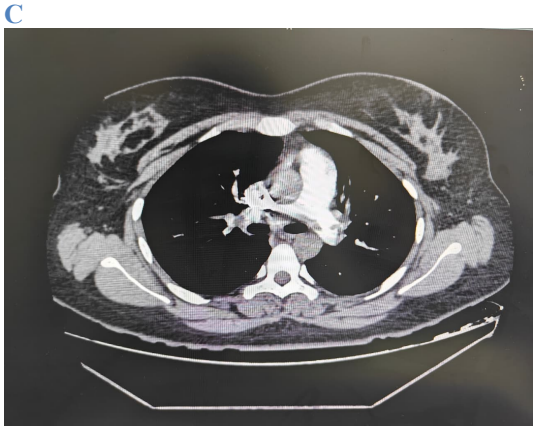
A



B



C



D

Figure 1. Axial CT pulmonary angiograms. (A) Upper mediastinal level. (B) Level of the pulmonary artery bifurcation. (C) Level of the main pulmonary trunk and the right and left pulmonary arteries. (D) Adjacent slice to C.