Case Report

OCP Induced Pulmonary Embolism in a Young Female: A Case Report

Shashwat Jindal¹, C. M. Adya¹, Himanshu Devender Kumar², Neha Sundhir¹

¹Junior Resident, Department of General Medicine, MMIMSR, Mullana, Haryana, India 133207 ²Professor, Department of General Medicine, MMIMSR, Mullana, Haryana, India 133207

Corresponding Author: Himanshu Devender Kumar

ABSTRACT

Healthy young females with active lifestyle without any family history are considered to be a low-risk subject for deep vein thrombosis (DVT) and pulmonary embolism(PE). We describe a case of18-yearold B Tech student who presented with breathing difficulties, chest discomfort and sudden onset cough caused by pulmonary embolism. Patient had no risk factor except she was on combined oral contraceptive (COC) pills. Thrombolytic therapy with reteplase was administered and patient was given Nicoumalone for 6 months and follow up was done. Patient was relieved of her symptoms with significant improvement of pulmonary angiogram.

Keywords: Combined oral contraceptive, Deep vein thrombosis, Pulmonary embolism

INTRODUCTION

The first case of venous thrombosis associated with contraceptive use was reported by Jordan in 1961. ^[1] Since then several researchers have shown an association of venous thrombosis after use of COC, some attributed risk of two to six times.^[2] Pulmonary embolism (PE) causes more than 100,000 deaths every year worldwide with mortality rate of more than 17% within first 12 weeks of diagnosis.^[3] The common risk factors for PE are older prolonged age, immobility, smoking, congenital or inherited clotting factors defects, and postoperative states other than malignancy, pregnancy, and chronic heart disease. Young active healthy females are considered to be a low-risk population for DVT and PE. We hereby present the unique case of a young woman, a B-Tech undergraduate, who developed bilateral PE likely due to COC pills use.

An 18-year-old female patient was admitted to hospital with complaints of dyspnea, palpitations, chest discomfort and dry cough two weeks prior to hospital admission. The patient's medical history was negative for significant disorders but the patient declared the use of oral contraceptives (OVRAL G: Ethinylestradiol mg, Levonorgestrel0.15mg; self-0.03 prescribed) for 4 months. On physical examination, tachycardia (HR= 130bpm) and tachypnea (RR= 40bpm) was present, chest auscultation was clear, S1 S2 was heard without any murmur and patient's SpO₂ on room air was 76%. ECG revealed sinus tachycardia without other abnormalities (Fig 1). Since the clinical presentation was suggestive of some heart or lung disease the patient underwent echocardiography, and blood samples were taken for laboratory tests.

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Parameter	Value	Reference
		range
Haemoglobin (g/dL)	9.2	12-16
TLC (cells/cmm)	14,000	4000-10,000
Polymorphs (%)	78	40-75
Lymphocytes (%)	19	20-40
AEC (cells/cmm)	420	40-440
PCV (mm/1 st hr)	34.5	36-46
Platelet count (cells/µL)	2.9 lac	1.5-4.5 lakhs
Urea (mg/dL)	17	15-40
Creatinine (mg/dL)	0.74	0.40-1.40
Sodium (meql/L)	139	135-145
Potassium (meql/L)	5.3	3.5-5
D-dimer (µg/L)	620	0-233
ECG	Sinus Tachycardia	
Chest Xrav	Normal lung fields	

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TLC: Total leukocyte count, AEC: Absolute eosinophillic count, PCV: Packed cell volume, ECG: Electrocardiogram

On echocardiography RA clot and indirect signs of acute pulmonary embolism were found including McConnell's sign, Dilated RA/RV, Moderate TR/PAH (55+ PAP) (Fig 2). The patient was sent for CT pulmonary angiogram (CTPA) which revealed B/L severe pulmonary P acute embolism extending into segmental, subsegmental branches with an area of pulmonary hemorrhage and infarction (Fig 3). Based on imaging, blood test results and clinical presentation, massive pulmonary embolism was diagnosed. Low molecular weight heparin – Enoxaparin at a weightadjusted dose of 1 mg/kg was administered

intravenous and the patient was shifted to CCU. Reteplase was used for thrombolysis with 18 mg i/v stat and the second dose was given 30 min later. The patient was put on non-invasive ventilation (NIV) and inotropic support in intensive care unit.

The patient's state improved significantly over a period of time. Bilateral lower limb Doppler was also done which ruled out any DVT. Post thrombolysis no recurrent dyspnoea, fainting or bleeding were observed. After 10 days, the patient was discharged on oral anticoagulant Acitrom (Nicoumalone) 2 mg once daily for 6 months with a target INR 2-3 and to follow up after 6 months with repeat CTPA. On follow up after 6 months, the patient was relieved of her signs and symptoms. Repeat Echocardiographic measures were s/o Normal RV systolic function, Normal size RA/RV, Trace MR, Trace TR, (PAP=34 mm of Hg), LVEF 50-55%, No AS and AR, Normal LVEDP, No Clot/Veg/ and CT Pulmonary angiography revealed chronic pulmonary thromboembolism lower lobar branch of Left pulmonary artery extending into segmental branch with mild PAH with significant resolution as compared to the previous scan (Fig 4).



Figure 1: ECG showing sinus tachycardia



Figure 2: Echocardiography showing RA clot and indirect signs of acute pulmonary embolism, Dilated RA/RV, Moderate TR/PAH (55+ PAP)



Fig 3: CT pulmonary angiogram (CTPA) showing bilateral severe acute pulmonary embolism extending into segmental, subsegmental branches with an area of pulmonary hemorrhage and infarction.



Figure 4: CT Pulmonary angiography showing chronic pulmonary thromboembolism lower lobar branch of Left pulmonary artery extending into the segmental branch with mild PAH with significant resolution as compared to the previous scan.

DISCUSSION

Pulmonary embolism (PE) remains one of the most common causes of sudden death among apparently young healthy women, especially during pregnancy and the postpartum period. ^[4] Combined oral contraceptives were long ago reported to induce venous thromboembolism (VTE), the most important determinant of the benefit/ risk profile of contraceptives. ^[5] There are reports concerning about one-half of cases of VTE in young women of reproductive age related to the use of oral contraceptives.^[6] Deep vein thrombosis (DVT) was reported in 60% and pulmonary embolism in 30% of contraceptive users aged from 18-49.^[7] In 59% of survivors, a permanent post-embolic residual deficit of lung perfusion was found on imaging.^[8] The highest risk of thrombosis is typical for the first year of contraceptive use. Estrogens raise the levels of factors II, VII, VIII and X as well as induce activated protein C resistance. The fibrinolytic potential is decreased in oral contraceptive users. Clinical presentation of pulmonary embolism may be nonspecific in COC users, including pain in the Paracostal low back and scapular region, as well as a cough.^[7]

CONCLUSION

Before COCs are prescribed, counseling on side effects, including VTE, is mandatory as well as careful evaluation of risk factors for VTE and screening for other abnormalities, including hypertension, obesity, and cigarette smoking and positive family history for spontaneous VTE is recommended.

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