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Case Report

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A CASE REPORT OF AN UNUSUAL CAUSE OF PULMONARY EMBOLISM

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ABSTRACT

Pulmonary embolism is a medical emergency with an incidence of approximately 70/100000.It is one of the most common preventable cause of death among hospitalized patients.[1] So finding out the underlying cause of pulmonary embolism is important.Here we report a case of pulmonary embolism due to native drug intake(Femino).

KEYWORDS: Pulmonary embolism, femino, RV dysfunction, native drug.

INTRODUCTION

Pulmonary thromboembolism has many risk factors such as cancer, obesity, cigarette smoking, systemic arterial hypertension, chronic obstructive pulmonary disease, chronic kidney disease, blood transfusion, long- haul air travel, air pollution, pregnancy, post menopausal hormonal replacement, surgery and trauma.[2] The most common symptom is unexplained breathlessness.[3] Certain drugs such as oral contraceptive pills cause pulmonary thrombo-embolism due to the following reasons – (i)increase in blood clotting factors, (ii)decreased anti-thrombin III, (iii)decreased plasminogen activator in endothelium, (iv)increased platelet aggregation.[4] The present case is due to a native drug given for polymenorrhoea.

CASE HISTORY

46 year old female from Villupuram presented to emergency department with complaints of breathlessness for 1 week. The dyspnea was sudden, gradually progressing, NYHA Grade 3 associated with diffuse chest pain on and off. She also gave history of bilateral pedal oedema with normal urine output. She has undergone cholecystectomy and tonsillectomy few years back. The patient is non diabetic and normotensive with a history of NSAID abuse for body ache. Patient had taken native drug Femino for irregular menstrual cycles(Ingrediants –Tribulus terrestris - 0.110g, Trachyspermum ammi - 0.110g, Hibiscus rosasinensis - 0.2170 g, Asterecantha longifolia - 0.055g and Erythrina variegate - 0.055 g) for the last 6 months.

On examination, patient was conscious, oriented, afebrile. She was pale with bilateral pitting pedal oedema. She had tachycardia(120 beats/min) with blood pressure of 160/110 mm Hg.CVS examination revealed ejection systolic murmur in aortic area. RS examination was normal. Since the patient had unexplained breathlessness and her Well's criteria score was 4.5, diagnosis of pulmonary embolism was considered.

Basic investigations showed HB -8.4, TC-17000, ESR - 17,HBA1C-8.4g%. Her renal functions, electrolyes, ESR and thyroid functions were all normal. ECG showed Sinus tachycardia, normal axis and S1Q3T3 changes. ECHO revealed right atrium and right ventricular dilatation with mild pulmonary artery hypertension (38mm Hg) and trivial MR and TR. There was no regional wall motion abnormalities. Left ventricular function was normal. Chest x-ray PA view showed hilar prominence and cardiomegaly. D-dimer was positive.

CT pulmonary angiogram showed partial thrombus causing <25% luminal narrowing in Left pulmonary artery, Partial thrombus causing >50% luminal narrowing in ascending and descending branches of both pulmonary artery and their segmental branches.

USG abdomen showed hepatomegaly with grade 1 fatty liver and post cholecystectomy status. Cardiac panel showed elevated BNP level.

To find out the cause of pulmonary embolism, venous doppler of both lower limbs was done and showed no evidence of deep vein thrombosis. Tests for prothrombotic states such as protein C, protein S, anti thrombin III levels were normal. Anti-nuclear antibody by IFA and anti phospholipid antibody(IgM and IgG) were negative. Hence we attributed the pulmonary embolism in this patient to prolonged native drug intake

(Tribulus terrestris) which was supported by a case report.[5] Our patient was managed in acute stage by thrombolysis and was started on anti-coagulants later. Patient was adviced to continue anti coagulants for 3 months on discharge.

DISCUSSION

Venous thromboembolism encompasses DVT and PE and causes cardiovascular death and disability.Patho-physiology of PE includes one of the 3 following mechanisms – (i)Inflammation and platelet activation, (ii) prothrombotic state and (iii) embolization.[6] Anti thrombin, protein C and protein S are naturally acting coagulation inhibitors. Deficiency of these inhibitors are associated with venous thromboembolism. Antiphospholipid antibody syndrome is the most common acquired cause of thrombophilia and is associated with venous or arterial thrombosis.[7] Non thrombotic PE etiologies include fat embolism after pelvic/long bone fracture, tumor embolism, bone marrow and air embolism. Intravenous drug users with substances such as hair,talc and cotton can develop PE.

Diagnosis of PE is challenging because its symptoms and signs are non-specific. The quantitative plasma D-dimer ELISA raises in presence of DVT or PE because of the breakdown of fibrin by plasmin. The sensitivity of D-dimer is >95% for PE.[8] However D-dimer is not specific. If D-dimer is negative, it rules out DVT/PE.

Cardiac biomarkers such as troponin, BNP rise in the presence of PE because of RV micro-infarction and failure.[9] ECG changes in PE include sinus tachycardia, S1Q3T3 sign,T wave inversion in leads V1 to V4.[10] The best known indirect sign of PE on transthoracic echocardiography is McConnel's sign[11] which is hypokinesis of RV free wall with normal or hyperkinetic motion of RV apex. Well established abnormalities in chest x-ray include focal oligaemia(Westermark's sign), peripheral wedge shaped density (Hampton's sign) and an enlarged pulmonary artery(Palla's sign).[12] CT of the chest with IV contrast is the principal imaging test for the diagnosis of PE.[13] For patients who are pregnant or with CKD, pulmonary embolism may be diagnosed with ventilation/perfusion scan. Effective anticoagulation is needed for successful treatment of PE.

Management for patients with normal RV and normotensive requires only secondary prevention by means of oral anti coagulants. For patients who are normotensive with RV hypokinesia,therapy is individualized. Embolectomy or thrombolysis is indicated for patients who are hypotensive.

The native drug Tribulus terrestris is used in Indian medicine for urinary tract infection, urolithiasis, dysmenorrhoea and oedema.[14] Other side effect of this drug include nephrotoxicity, gynaecomastia and reduced testosterone level.

CONCLUSION

Pulmonary embolism is one of the most common preventable cause of death in hospitalized patients. Finding out the etiology is very important to treat as well to prevent recurrence. Apart from the above mentioned risk factors, some native medications can also cause pulmonary thrombo-embolism rarely. So, this should be kept in mind while evaluating a patient with pulmonary thromboembolism and native drug intake has to be ruled before considering the etiology as idiopathic.

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