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Pulmonary Embolism - A Case Report

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Abstract:

PE remains a clinically challenging diagnosis, more often missed than found, with no decline in its incidental discovery at autopsy over the past 30 years. Pulmonary embolism should be considered in the differential diagnosis of every dyspnoea event that presents at an emergency department. We describe a case of 68 years old man with symptoms of dyspnoea who later diagnosed as pulmonary embolism. This case report emphasizes early diagnosis and treatment to avoid fatal outcome.

Key words: Pulmonary embolism, dyspnoea.

Introduction:

Pulmonary embolism (PE) ranges from incidental, clinically unimportant occurrences to causing sudden death. Virchow's triad of local trauma to the vessel wall, hypercoagulability, and stasis of blood leads to thrombus formation in the leg veins.[1] As thrombi form in the deep veins of the legs, pelvis, or arms, they may dislodge and embolize to the pulmonary arteries with potentially serious consequences. The most common sources of pulmonary emboli are the pelvic veins or deep veins of the thigh.[2] Pulmonary arterial obstruction by clot causes dilatation, dysfunction, and ischemia of the right ventricle.[1]

Pulmonary embolism and deep venous thrombosis is responsible for more than 250,000 hospitalizations and approximately 50,000 deaths per year in the United States. Because it is difficult to diagnose, the true incidence of pulmonary embolism is unknown, but it is estimated that approximately 650,000 cases occur annually.[1] Despite this high incidence, the diagnosis of pulmonary embolism continues to be difficult primarily because of the notorious varities of symptoms and signs in its presentation.[2]

We present the case of a patient with pulmonary embolism presented with dyspnoea and review the pathophysiology and diagnostic considerations.

Case report:

Mr. X 68 yrs old pleasant gentleman admitted in CCU of UHL for respiratory distress along with sweating for 2 hours. He had also history of exertional dyspnoea and dyspepsia for last 4 days. He is diabetic, hypertensive and nonalcoholic. On G/E his Pulse-104/min, BP-120/90mmHg, RR-20/min and cardiac examination showed that there was a pansystolic murmur over the tricuspid area which increased with accentuated pulmonary component of the second heart sound.

Initial investigations showed Hb-14gm/dl,TC-12.3X10^3/µl, Platelet-222 x10^3/µl, S,creatinine-1.45mg/dl,$H_s$troponinI-423ngm/ml, PT-13.7sec, INR-1.22, APTT-31.2sec, D Dimer-4860ng/ml(0-550ng/ml) . Electrocardiography showed sinus tachycardia with SIQ3T3. Chest X-Ray revealed cardiomegaly. 2-D transthoracic echocardiography showed a dilated right side of the heart with a 5.7 cm × 1.4 cm mass in the right pulmonary artery. Valvular morphology was normal with moderate tricuspid regurgitation and moderate pulmonary hypertension. CT pulmonary angiography found pulmonary embolism involving the right pulmonary artery and most of their segmental branches. He underwent a duplex scan of the deep venous system of both lower limbs, which was found to be deep venous thrombosis in left popliteal vein. The patient was treated with streptokinase which was given initially as a loading dose of 250,000 IU over 30 minutes, followed by a dose of 100,000 IU/hour over 72 hours. After initial evaluation, a blood sample was taken to examine the thrombophilia panel [protein C-61IU/dl(70-146IU/dl), protein S-75IU/dl (60-130IU/dl), antithrombin-III-76.7 µg/ml (75-125 µg/ml)].There his protein C level was low. Further echocardiography was performed and it revealed normal right heart function.

After the recovery of the patient from the acute stage, anticoagulant therapy was given, initially in the form of enoxaparin sodium at a dose of 80 mg/kg for seven days. In conjunction, rivaroxaban was given at a daily dose of 20 mg.

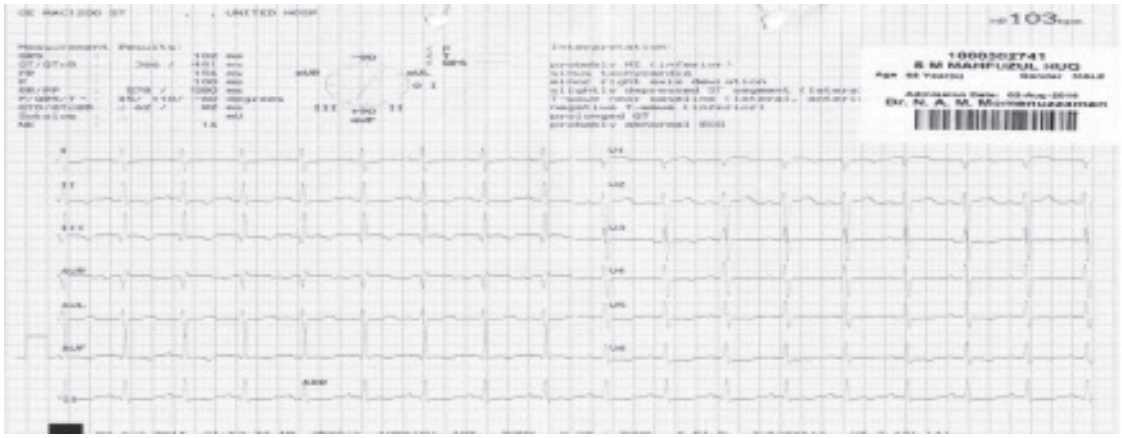

Fig.-1: *ECG showed SIQ3T3.*

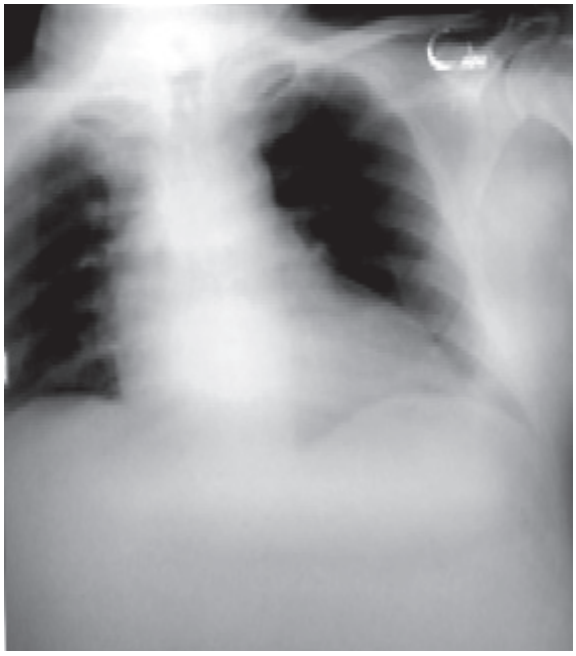

Fig.-2: *CXR P/A view showed*

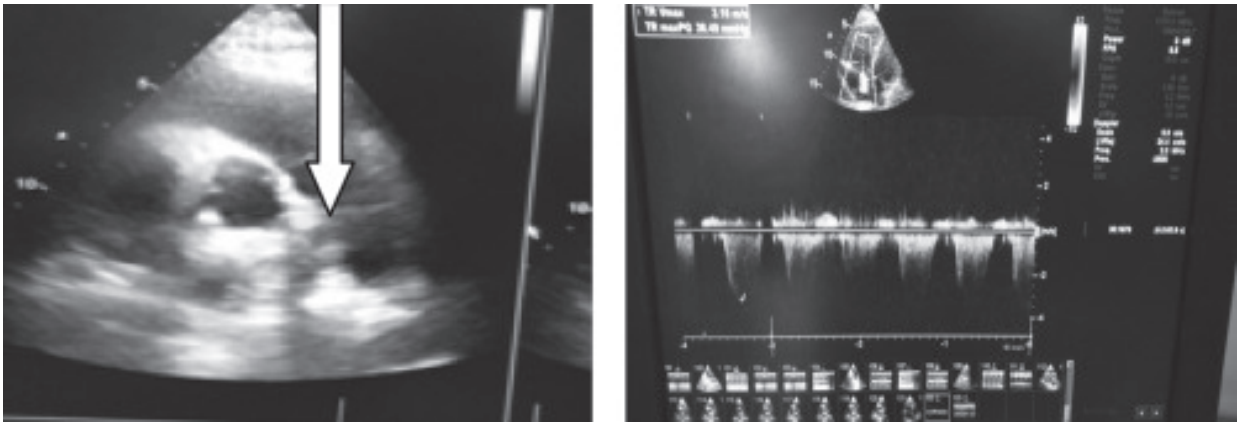

Fig.-3 & 4: *Echocardiography showed thrombus at right pulmonary artery and tricuspid regurgitation*

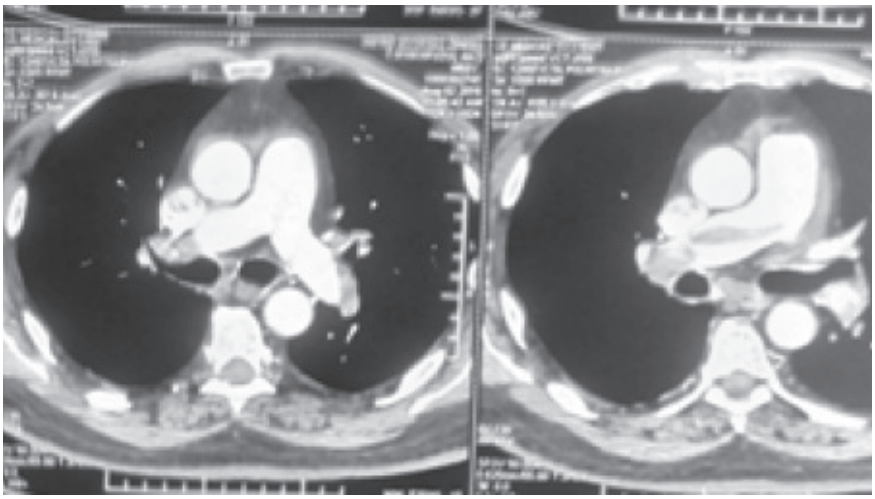

Fig.-5 : *CT pulmonary angiography showed thromnus at right pulmonary artery After thrombolysis*

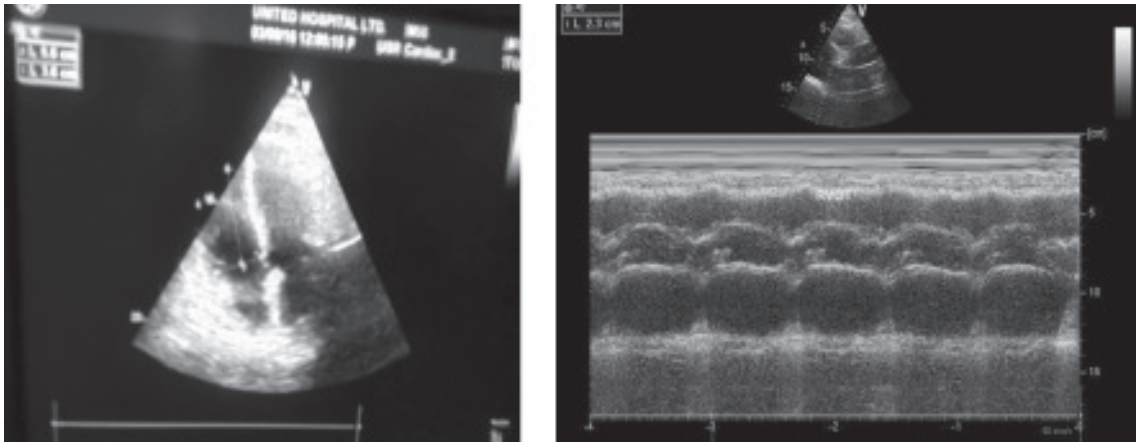

Fig.-6 : *Normal RA and RV dimension*

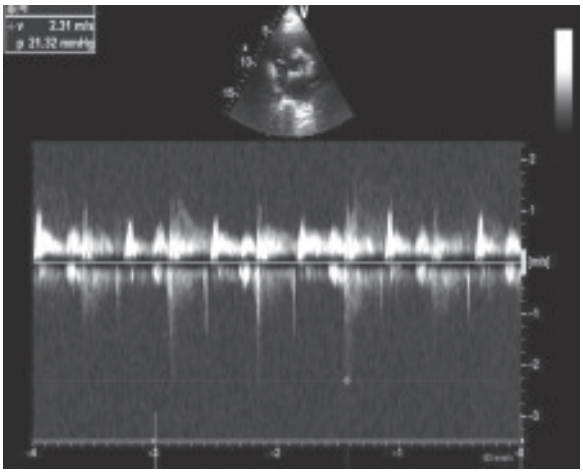

Fig.-7 : *Colour Doppler echocardiography showed trivial tricuspid regurgitation*

Discussion:

Pulmonary embolism is a serious medical emergency. Nevertheless, it remains difficult to diagnose. Pulmonary emboli differ considerably in size and number, and the underlying disorders, including malignancy, trauma, and protein C or S deficiency, are numerous.[1] Protein-C deficiency by plasma level alone is found in 1 in 200 to 1 in 500 persons in the general population .[3,4] However, many affected individuals remain asymptomatic throughout life. The cardinal clinical manifestation of heterozygous protein C deficiency is venous thromboembolism .[5,6] In this case report the patient presented with pulmonary embolism.

Signs and symptoms of pulmonary embolism occur suddenly. Dyspnea, tachypnea, chest pain, cough, and hemoptysis take place. In more severe cases, cyanosis, syncope and circulatory instability occur, and sometimes

peripheral edema may be present. Sudden death can occur in the most severe cases .[7] About 25% of PE cases present as sudden death while 15% of all cases of sudden death area attributable to PE.[8]The classic triad of pleuritic chest pain, dyspnea, and hemoptysis is rare, and clinically apparent DVT is present in only 11% of confirmed cases of pulmonary embolism in patients without underlying cardiopulmonary disease.[9]

However, the clinical picture of pulmonary embolism is variable and most patients suffering from acute pulmonary embolism present with one of three different clinical syndromes. These clinical syndromes are pulmonary infarction, acute unexplained dyspnea, and acute cor pulmonale. The pulmonary infarct syndrome usually occurs with a submassive embolism that completely occludes a distal branch of the pulmonary circulation. Patients with this condition have pleuritic chest pain, hemoptysis, rales, and abnormal findings on chest X-ray. The acute, unexplained dyspnea pattern may also be the result of submassive pulmonary embolism without pulmonary infarction. Results of a chest X-ray and electrocardiogram are usually normal, but pulse oxygen saturation is often depressed. The third pattern, acute cor pulmonale syndrome, is caused by the complete obstruction of 60 to 75% of pulmonary circulation. Patients with this pattern experience shock, syncope, or sudden death. [9,10] Syncope occurs in approximately 10% of patients with acute pulmonary embolism and is commonly ascribed to a massive, hemodynamically unstable acute pulmonary embolism. [11] Here our patient present with dyspnoea and investigation revealed S1Q3T3 in ECG, echocardiography revealed dilated RA,RV and thrombus in right pulmonary artery.

The $S_1Q_3T_3$ sign (prominent S wave in lead I, Q wave and inverted T wave in lead III) is a sign of *acute cor pulmonale* (acute pressure and volume overload of the right ventricle because of pulmonary hypertension) and reflects right ventricular strain.[12] This electrocardiogram (ECG) finding is present in 15% to 25% of patients ultimately diagnosed with pulmonary emboli (PE). This patient presented with this sign .A bedside transthoracic or transesophageal echocardiogram (TEE) can be used to demonstrate signs of right ventricular pressure overload, and right ventricular hypokinesis and/or dilatation. While the electrocardiogram and the echocardiogram have long been used to assess PE, several com-puted tomographic criteria recently have been validated in the assessment of the severity of PE. This allows a single test, namely the spiral CT, to establish the diagnosis and assess the severity of PE. In addition, elevated cardiac biomarkers such as cardiac troponin and brain naturetic peptide (BNP) have well established diagnostic and prognostic roles.[13]

Heparin constitutes the cornerstone of management of PE. Thrombolysis can be lifesaving in patients with massive pulmonary embolism, cardiogenic shock, or overt hemodynamic instability. Thrombolytic agents accelerate the lysis of the PE.[14]

In our case, the patient presented to the emergency department with complaints of dyspnea. He was hemodynamically stable at admission and diagnosed as a case of PE without any feature of DVT. There CTPA and duplex study identified thrombus in pulmonary artery and poplital vein respectively. Treated with Inj. Streptokinase dose of 2,50,000 unit/30 min and followed by 1,00,000 unit/h for 72 h. This case is interesting because these are rarely diagnosed and treated.

Conclusion:

Pulmonary emboli are potentially life threatening occurrences associated with significant morbidity and mortality. There are a variety of diagnostic tools that maximize our ability to detect PE and enable better prognostication. Right ventricular dysfunction and the release of cardiac biomarkers are associated with more adverse events. Patients treated with thrombolytic therapy show rapid improvement of right ventricular function and pulmonary perfusion which may lead to a lower rate of early recurrent PE and a decrease the late sequela of chronic pulmonary hypertension.

References

1. Wolfe TR, Allen TL. Syncope as an emergency department presentation of pulmonary embolism. J Emerg Med. 1998;16:27–31.
2. Manolis AS, Linzer M, Estes M III. Syncope: current diagnostic evaluation and management. Ann Intern Med. 1990;112: 850–863.
3. Tait RC, Walker ID, Reitsma PH, *et al.*: Prevalence of protein C deficiency in the healthy population. *Thromb Haemost1995, 73(1):87-93.*
4. Miletich J, Sherman L, Broze G Jr: Absence of thrombosis in subjects with heterozygous protein C deficiency. *N Engl J Med* 1987, 317(16):991-996.
5. Martinelli I, Mannucci PM, De Stefano V, *et al.*: Different risks of thrombosis in four coagulation defects associated with inherited thrombophilia: a study of 150 families. *Blood* 1998, 92(7):2353-2358.
6. Koster T, Rosendaal FR, Briet E, *et al.*: Protein C deficiency in a controlled series of unselected outpatients: an infrequent but clear risk factor for venous thrombosis (Leiden Thrombophilia Study). *Blood* 1995, 85(10):2756-2761.

7. Goldhaber S: Pulmonary thromboembolism. In Harrison's Principles of Internal Medicine. 16 edition. Edited by Kasper D, Braunwald E, Fauci A. New York, NY:McGraw-Hill; 2005; 1561–1565.

8. Beckman MG, Hooper WC, Critchley SE, *et al.*: Venous thromboembolism: a public health concern. Am J Prev Med. 2010; 38(4 Suppl): S495–501.

9 . Koutkia P, Wachtel TJ. Pulmonary embolism presenting as syncope: case report and review of the literature. Heart Lung. 1999;28:342–347

10. Varon J, Fromm RE. Syncope: the forgotten sign of pulmonary embolism. J Emerg Med.1998; 16:117–118. doi: 10.1016/S0736-4679(98)00061-4.

11. Bell WR, Simon TL, DeMets DS. The clinical features of submassive and massive pulmonary emboli.Am J Med. 1977;62:355–360.

12. Chan TC, Vilke GM, Pollack M, Brady WJ. Electrocardiographic manifestations: pulmonary embolism. J Emerg Med .2001 Oct;21(3):263-70.

13. Jarreau T, Hanna E, Rodriguez FH,et al.:Massive pulmonary embolism: a case report and review of the literature: J La State Med Soc.2008;160:189-194.

14. Jerjes S C, Ramírez R A, García ML, et al. Streptokinase and heparin versus heparin alone in massive pulmonary embolism: a randomized controlled trial. J Thromb Thrombolysis 1995; 2:227-229.